

# MANIPULATING PIG PRODUCTION V

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## CONTENTS

CONTRIBUTORS .....	xi
ACKNOWLEDGEMENTS .....	xxii
PREFACE .....	xxiii

### A.C. DUNKIN MEMORIAL LECTURE

#### REVIEW:

Future directions and research needs of the Australian pig industry .....	1
<i>R.G. Campbell</i>	

### APPLICATION OF RESEARCH

#### REVIEW:

Organisation and application of research and development in commercial pig herds: The Danish approach .....	7
<i>B.K. Pedersen, V. Ruby and E. Joergensen</i>	

### BEHAVIOUR AND HOUSING

#### ABSTRACTS:

Pig performance in low-cost, straw-bedded, alternative housing systems - Preliminary results .....	19
<i>H.G. Payne</i>	
The effects of bedding and human contact before parturition on the onset of parturition in sows .....	20
<i>G.M. Cronin, G.J. Simpson and B.N. Schirmer</i>	
Effect of enclosure of the feeding space on feeding behaviour of growing pigs .....	21
<i>G.D. Hutson</i>	
Turn-around stalls and the welfare of pigs .....	22
<i>J.L. Barnett and I.A. Taylor</i>	
Improving piglet survival through intensive supervision at farrowing .....	23
<i>P.K. Holyoake, T. Trigg, V.L. King, W.E. Marsh and G.D. Dial</i>	
A comparison of indoor and outdoor pig production systems in New Zealand .....	24
<i>J.D.G. Anderson, P.C.H. Morel and B.J. Stevenson</i>	

### NUTRIENT DIGESTIBILITY

#### ABSTRACTS:

A new method for determining available lysine in feedstuffs .....	25
<i>S.M. Rutherford and P.J. Moughan</i>	
The use of near infrared reflectance spectroscopy to predict the true ileal digestibility of amino acids in oilseed meals for pigs .....	26
<i>P.E.V. Williams, J.C. Bodin, R. Maillard and D.A. Jackson</i>	
Algal proteins labelled with <sup>15</sup> N can be used to quantify endogenous protein loss from the small intestine in pigs .....	27
<i>N.J. Gannon and P.J. Reeds</i>	
True ileal digestible sulphur amino acid requirements for young pigs .....	28
<i>P.H. Simmins, J.C. Bodin, A.K. Kies and P.E.V. Williams</i>	

Effect of lupin kernels on the apparent ileal digestibility of amino acids by growing pigs .....	29
<i>R.J. van Barneveld, J. Baker, S.R. Szarvas and M. Choct</i>	
Effect of lupin kernels on the ileal and faecal digestibility of energy by pigs .....	30
<i>R.J. van Barneveld, J. Baker, S.R. Szarvas and M. Choct</i>	
Digestibility of non-starch polysaccharides by pigs fed graded levels of lupin kernels .....	31
<i>R.J. van Barneveld, J. Baker, S.R. Szarvas and M. Choct</i>	
Digestibility of amino acids and energy in naked oats ( <i>Avena sativa</i> cv. Bandicoot) fed to growing pigs .....	32
<i>R.J. van Barneveld, J. Baker, S.R. Szarvas and A.R. Barr</i>	
Factors affecting the determination of digestible energy in <i>Lupinus angustifolius</i> cv. gungurru for growing pigs .....	33
<i>G.C. Wigan, the Late E.S. Batterham and D.J. Farrell</i>	
Long-chain hydrocarbons as a marker for digestibility studies in monogastric species .....	34
<i>M. Choct and R.J. van Barneveld</i>	
The rat as a model for the pig for determining true ileal reactive lysine digestibility .....	35
<i>S.M. Rutherford, S.M. Hodgkinson and P.J. Moughan</i>	
Carbon dioxide is not an alternative to halothane anaesthesia in nitrogen digestibility studies .....	36
<i>S. Prawirodigo, N.J. Gannon, D.J. Kerton, B.J. Leury, R.J. van Barneveld and F.R. Dunshea</i>	
Effect of condensed tannin in cottonseed hulls on true ileal amino acid digestibility in casein .....	37
<i>F. Yu, P.J. Moughan and T.N. Barry</i>	
Effect of energy source and feeding/collection time on ileal contents obtained from weaner pigs .....	38
<i>D.I. Officer, L.M. Andersen and L.R. Giles</i>	
Determination of apparent ileal protein and amino acid digestibility in feed for pigs using the "Mobile Nylon Bag Technique" .....	39
<i>R. Mosenthin, U. Bornholdt, W.C. Sauer, F. Ahrens, H. Jorgensen, and B.O. Eggum</i>	
An <i>in vitro</i> method for predicting digestible energy applied to milling by-products .....	40
<i>Jiai Chen, S. Boisen, G. Pearson, P.C.H. Morel and P.J. Moughan</i>	

## REGULATION OF GROWTH

### SYMPOSIUM: Novel methods to enhance growth

Introduction .....	41
<i>D.K. Revell and J.R. Pluske</i>	
Potential of exogenous metabolic modifiers for the pig industry .....	42
<i>F.R. Dunshea and P.E. Walton</i>	
Manipulation of endogenous hormones to increase growth of pigs .....	52
<i>I. McCauley, A. Billinghamurst, P.O. Morgan and S.L. Westbrook</i>	
Commercialisation of novel growth enhancers in pigs - Economic and political considerations .....	62
<i>M.J.M. Bent</i>	
Conclusions .....	75
<i>D.K. Revell and J.R. Pluske</i>	

**ABSTRACTS:**

- Relative aversiveness of a new injection procedure for pigs .....81  
*P.H. Hemsworth, J.L. Barnett and R.G. Campbell*
- Absorption of insulin-like growth factor-I in neonatal pigs is independent  
of gut closure .....82  
*R.J. Xu and T. Wang*
- Effects of intra-venous administration of insulin-like growth factor II on  
energy metabolism .....83  
*P.C. Owens, W. Morley, K.J. Quinn, G.L. Francis, J.A. Owens, F.R. Dunshea and  
R.G. Campbell*
- $\beta$ -Adrenergic receptors on porcine immune cells .....84  
*J.R. Keys, P.C. Wynn and M.R. Jones*
- Production and endocrine responses in pigs to acute pre-natal testosterone  
treatments ..... 85  
*C. McKenzie, S. Manickam and B. Hosking*
- Endocrine regulation of somatotrophin secretion by IGF-I and IGF-I analogues in pigs ..86  
*V. Dunaiski, F.R. Dunshea, I.J. Clarke, C. Goddard, P.C. Owens and P.E. Walton*

**REPRODUCTION****ABSTRACTS:**

- Effect of concentration of spermatozoa on *in vitro* fertilization of pig oocytes  
matured *in vitro* .....87  
*S.J. Robinson, G. Evans and W.M.C. Maxwell*
- Morphology and *in vitro* fertilizing capacity of boar spermatozoa following sex  
selection by flow cytometry .....88  
*J.K. O'Brien, S.L. Catt, W.M.C. Maxwell and G. Evans*
- Introduction of gilts to boars elevates plasma cortisol but does not  
affect reproduction .....89  
*A.I. Turner, P.H. Hemsworth, P.E. Hughes and A.J. Tilbrook*
- The effects of contact frequency and season on the efficacy of the boar effect .....90  
*G. Philip and P.E. Hughes*
- Effect of pre-pubertal body fat on the development of the reproductive tract in gilts ...91  
*J.B. Gaughan, R.D.A. Cameron and G. McL. Dryden*
- The efficacy of the boar effect when conducted in a detection-mating area .....92  
*R. Siswadi and P.E. Hughes*

**LACTATION AND PRE-WEANING GROWTH****SYMPOSIUM: Constraints to pre-weaning growth**

- Introduction .....93  
*R.H. King*
- Metabolic regulation of sow lactation .....94  
*P.E. Hartmann, M.J. Thompson, L.M. Kennaugh and C.S. Atwood*
- The influence of substrate supply on milk production in the sow .....101  
*J.E. Pettigrew*
- Sow milk as a major nutrient source before weaning .....107  
*I.H. Williams*
- Piglets' role in determining milk production in the sow .....114  
*D.E. Auldist and R.H. King*
- General summary - Constraints to pre-weaning growth .....119  
*J.E. Pettigrew*

**ABSTRACTS:**

The voluntary food intake of lactating sows in five commercial herds .....	127
<i>G. Handley, R.H. King and A.K. King</i>	
Body fatness reduces voluntary feed intake and alters plasma metabolites during lactation.....	128
<i>D.K. Revell, I.H. Williams, J.L. Ranford, B.P. Mullan and R.J. Smits</i>	
Super-alimentation of gilts during lactation.....	129
<i>J.R. Pluske, I.H. Williams, E.C. Clowes, L.J. Zak, A.C. Cegielski and F.X. Aherne</i>	
Total creatine in sows' colostrum and milk.....	130
<i>L.M. Kennaugh and P.E. Hartmann</i>	
Effects of dietary lysine during the first lactation on subsequent litter size of sows.....	131
<i>S.M. Tritton, R.H. King, R.G. Campbell, P.E. Hughes and S.S. Kershaw</i>	
The digestibility of amino acids in human milk.....	132
<i>A.J. Darragh and P.J. Moughan</i>	
The effect of food intake during lactation on nitrogen metabolism of first-litter sows .....	133
<i>R.H. King, J. Toussaint, P.J. Eason and L. Morrish</i>	
Potential milk production in gilts .....	134
<i>The Late R.H. Head and I.H. Williams</i>	
Can nutrition in pregnancy and lactation affect the development of the mammary gland? .....	135
<i>R.J. Smits, D.K. Revell, I.H. Williams, B.P. Mullan, J.L. Ranford and D.S. Chappell</i>	
A high-protein diet maximizes milk output and minimizes weight loss in lactation .....	136
<i>D.K. Revell, I.H. Williams, J.L. Ranford, B.P. Mullan and R.J. Smits</i>	
Effect of increased suckling frequency on mammary development and milk yield of sows.....	137
<i>D.E. Auldust, D. Carlson, L. Morrish, C. Wakeford and R.H. King</i>	
The effect of dietary lysine on the composition of milk in gilts .....	138
<i>H.Y. AL-Matubsi, S.M. Tritton, R.G. Campbell and R.J. Fairclough</i>	

**GASTROINTESTINAL HEALTH AND PHYSIOLOGY****REVIEW:**

Intestinal spirochaetal infections of pigs: An overview with an Australian perspective.....	139
<i>D.J. Hampson and D.J. Trott</i>	

**ABSTRACTS:**

Fermentation in the large gut and swine dysentery.....	170
<i>P.M. Siba, D.W. Pethick, J.R. Pluske, B.P. Mullan and D.J. Hampson</i>	
Application of a polymerase chain reaction assay to diagnose proliferative enteritis in pig herds.....	171
<i>P.K. Holyoake, G.F. Jones, P.R. Davies, D.L. Foss, M. Panaccio, D. Hasse and M.P. Murtaugh</i>	
Bacterial colonization of the piglet gut.....	172
<i>M.J. Thompson, C.M. Wakeford and P.E. Hartmann</i>	

The potato fibre preparation POVEX enhances pancreatic secretion in the pig.....	173
<i>S.G. Pierzynowski, I. Mattsson, M-J. Thaela, B.R. Weström and B.W. Karlsson</i>	
Weight at weaning, causes and consequences .....	174
<i>P.D. Cranwell, I. Tarvid, L. Ma, D.T. Harrison and R.G. Campbell</i>	
Gut development from 4 to 23 weeks of age .....	175
<i>P.D. Cranwell, I. Tarvid, L. Ma, D.T. Harrison and R.G. Campbell</i>	
Gastric proteases in light and heavy pigs at 4, 6 and 23 weeks of age.....	176
<i>L. Ma, P.D. Cranwell, R. Vavala, I. Tarvid, D.T. Harrison and R.G. Campbell</i>	
Pancreatic proteases in light and heavy pigs at 4, 6 and 23 weeks of age .....	177
<i>I. Tarvid, P.D. Cranwell, L. Ma, D.T. Harrison and R.G. Campbell</i>	
Small intestinal peptidases in light and heavy pigs at 4 and 6 weeks of age.....	178
<i>I. Tarvid, P.D. Cranwell, L. Ma, D.T. Harrison and R.G. Campbell</i>	
Reduced plasma concentrations of glutamine and its metabolites in weaned pigs.....	179
<i>A.I. Ayonrinde, I.H. Williams, R. McCauley and B.P. Mullan</i>	
Glutamine stimulates intestinal hyperplasia in weaned piglets.....	180
<i>A.I. Ayonrinde, I.H. Williams, R. McCauley and B.P. Mullan</i>	
Digestibility of bovine immunoglobulin in the piglet.....	181
<i>P.C.H. Morel, L.M. Schollum, T.R. Buwalda and G. Pearson</i>	
Porcine pancreatic exocrine function during the first five days after weaning .....	182
<i>S.G. Pierzynowski, P. Kiela, D. Rantzer, M-J. Thaela, B.W. Karlsson and J. Svendsen</i>	
Colostrum feeding stimulates pancreatic growth in newborn pigs .....	183
<i>J.N. Mubiru, T. Wang and R.J. Xu</i>	
Stability of gastrin in the gastro-intestinal lumen of the pig .....	184
<i>R.J. Xu, Y.L. Mao and M.Y.W. Tso</i>	
Physiological response of lactating sows to feeding rapeseed meal "00" and microbial phytase.....	185
<i>Z. Mroz, W. Krasucki and E. Grela</i>	
Diagnosis of swine dysentery and intestinal spirochaetosis by the use of polymerase chain reaction tests on faecal samples.....	186
<i>R.F. Atyeo and D.J. Hampson</i>	

## APPLIED NUTRITION AND ENGINEERING

### ABSTRACTS:

The effect of body-weight on the marginal efficiency of nitrogen utilization in pigs .....	187
<i>P. Bikker, V. Karabinas, H. van Laar, M.W.A. Verstegen and R.G. Campbell</i>	
AUSPIG increases profits by improved feed formulation.....	188
<i>R.J. Smits, I.H. Williams and N. Prestegar</i>	
The effect of AUSPIG determined dietary lysine levels on the growth performance of pigs between 63 and 112 days of age.....	189
<i>C.J. Brewster, D.J. Cadogan, R.G. Campbell, D.T. Harrison and S.S. Kershaw</i>	
$\beta$ -Glucanase supplementation of barley-based pig feeds reduces digesta viscosity and short chain fatty acid concentration.....	190
<i>J. Inbarr, B. Borg Jensen, K.E. Bach Knudsen, M. Skou Jensen and K. Jakobsen</i>	

Effect of phytate, phytase and lactic acid on faecal digestibility of ash and some minerals in pigs .....	191
<i>A.W. Jongbloed, P.A. Kemme, Z. Mroz, M. Mäkinen and A.K. Kies</i>	
Variation in the protein quality of blood meals .....	192
<i>G. Pearson, N. Meads, P.C.H. Morel and P.J. Moughan.</i>	
Effects of dietary available phosphorus and phytase (Natuphos) on the performance of pigs from 19 to 40 days post-weaning .....	193
<i>R.G. Campbell, D.T. Harrison, K.J. Butler and P.H. Selle</i>	
Interrelationships between protein source and Biofeed Plus on the performance of weaned pigs from 23 to 42 days of age .....	194
<i>R.G. Campbell, A. Whitaker, T. Hastrup and P.B. Rasmussen</i>	
Effect of phytate, phytase and lactic acid on the ileal amino acid digestibility in pigs .....	195
<i>P.A. Kemme, A.W. Jongbloed, Z. Mroz, M. Mäkinen and A.K. Kies</i>	
Zinc oxide supplementation for weaner pigs .....	196
<i>B.P. Mullan, J.G. Allen, J. Hooper, J.L. Ranford and S.Z. Skirrow</i>	
Biotin and pre-weaning mortality in pigs .....	197
<i>J.S. Kopinski and K. McGuigan</i>	
Performance of weaner pigs fed diets with two levels of animal protein and six levels of citric acid .....	198
<i>J.B. Gaughan and B.C. Granzin</i>	
High ambient temperature decreases voluntary feed intake but does not increase backfat thickness in entire male finishing pigs .....	199
<i>D.P. Irwin, B.P. Mullan, D.K. Revell and R.J. Smits</i>	
A protocol for evaluating pig feeders .....	200
<i>S.R.O. Williams and G.A. Moore</i>	
Disturbance-free pig weighing .....	201
<i>S.R.O. Williams, G.A. Moore and E. Currie</i>	
Characterization of piggery anaerobic lagoons in Southern Queensland .....	202
<i>K.D. Casey, E.A. Gardner and E.J. McGahan</i>	

## OCCUPATIONAL HEALTH AND SAFETY

### REVIEW:

The effects of environmental conditions inside pig housing on worker and pig health .....	203
<i>K.J. Donham</i>	

### ABSTRACTS:

The Australian pig industry takes the lead in occupational health and safety development .....	222
<i>A.G. Jackson</i>	
Air quality in weaner accommodation .....	223
<i>C. Cargill, N. Masterman, S.Z. Skirrow and T. Banhazi</i>	
Effects of pelleting feed on aerosols in pig sheds .....	224
<i>C. Cargill, S.Z. Skirrow, N. Masterman and T. Banhazi</i>	



## HEALTH

### ABSTRACTS:

- Physiological changes in growing pigs associated with pleuropneumonia challenge .....225  
*H.J. Bray, L.R. Giles, P.C. Wynn, J.L. Black and J.M. Gooden*
- Genetic and antigenic studies on *Haemophilus parasuis* .....226  
*P.J. Blackall, D.J. Trott, V.J. Rapp-Gabrielson and D.J. Hampson*
- Immunomagnetic capture polymerase chain reaction for detection of *Mycoplasma hyopneumoniae* .....227  
*S.P. Djordjevic, M. Zalunardo, M.J. Walker, G.J. Eamens and J. Chin*
- Subunit antigen profiles of geographically diverse porcine mycoplasmas recovered from pneumonic lungs .....228  
*A. Scarman, L. Romalis, J. Chin, G.J. Eamens, V. Taylor, S. Delaney and S.P. Djordjevic*
- Strategies for developing a subunit mycoplasmal vaccine for enzootic pneumonia .....229  
*G.J. Eamens, S.P. Djordjevic, J. Chin, P. Fagan, M.J. Walker and A. Scarman*
- A post-mortem study of sow deaths on a large Victorian pig farm .....230  
*I.D. Connaughton, R.A. Paterson and W.M. Forsyth*
- Leptospirosis - more than meets the eye .....231  
*R.J. Chappel and B. Adler*

## PIG HEALTH MONITORING SCHEMES

### REVIEW:

- Evaluation of the pig health monitoring scheme as an industry service .....232  
*A.M. Pointon*

## MEAT QUALITY

### ABSTRACTS:

- A comparison of P2 backfat measurements on the live pig, at slaughter and at dissection .....249  
*L.R. Bradley and R. Evans*
- Determination of fat and moisture content in sausages by near-infrared spectroscopy .....250  
*Y.H. Yeung and R.J. Xu*
- Pigmeat eating habits of children .....251  
*D.G. Laing, N. Oram, J. Owen, G. Moore, G. Rose and I. Hutchinson*
- pH decline and meat quality in red and white muscles of PSE and normal pork carcasses .....252  
*J.H. O'Halloran, R.D. Warner, B.J. Leury and D.N. D'Souza*
- Quality assurance in the Australian pig industry .....253  
*S.Z. Skirrow, P. Ryan, A.M. Paterson and P. Higgins*
- Assessment of the magnitude and significance of "boar taint" in Australia .....254  
*L. Salvatore, D.P. Hennessy, L. Sali, D. Waldron and R. Walsh*
- Effects of gender and age at slaughter on the quality of pig meat .....255  
*C.Y.J. Lee, Y.Y.T. Lo and R.J. Xu*

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Evaluation of pork quality defects.....	256
<i>The Late G.A. Eldridge, P. Maynard, R.D. Warner, A. Pengelly and H.M. Knowles</i>	
Evaluation of meat quality in commercial pigs in New Zealand .....	257
<i>P.C.H. Morel, G. Pearson and D.G. Hartley</i>	
The effects of adverse handling of pigs on farm and at the abattoir on meat quality .....	258
<i>D.N. D'Souza, the Late G.A. Eldridge, R.D. Warner, B.J. Leury, F.R. Dunshea and H.M. Knowles</i>	
Genetic relationships between carcass composition and meat quality in Australian pigs.....	259
<i>S. Hermes, B.G. Luxford and H-U. Graser</i>	
Effects of dietary vitamin E on post-mortem muscle quality traits of pigs.....	260
<i>R.D. Warner, R.G. Kauffman and D. Jerome</i>	
AUTHOR INDEX .....	261

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## PREFACE

These are the Proceedings of the Fifth Biennial Conference of the Australasian Pig Science Association (APSA). The Association held its first meeting in 1987 and has since been recognised as the major forum for scientific dialogue for the Australian pig industry. A major attraction of the biennial meeting is the diverse range of topics discussed and this characteristic is reflected in these proceedings as either reviews, symposia or submitted abstracts.

These proceedings include a record number of submitted abstracts accepted for publication (101). The strong contribution from Australasia reflects the good support that the pig industry receives from Government and industry. In particular the Australian Pig Research and Development Corporation is to be congratulated for their continued support of this conference. It is pleasing to report an increase in the number of abstracts submitted by overseas delegates as their contribution signals a growing awareness of this conference as an international forum for pig science, as well as helping to increase the exchange of information.

The pig industry like many other agricultural industries is undergoing tremendous change as it competes against other commodities on an international market. The importance of product quality, a reduced cost of production and sustainability are recognised as being central to the future of the pig industry. These and other issues are addressed in these proceedings.

Dr D.P. Hennessy and Mr P.D. Cranwell are to be congratulated on the excellent job as Editors of these proceedings, and the high standards that they have demanded ensure that these proceedings will remain an important reference for future pig research.

I would like to thank the Organising Committee for their efforts during the past two years. The Committee consisted of Dr D.K. Revell (Secretary), Mr R.J. Smits (Treasurer), Dr A.M. Paterson (Vice President), Dr M.R. Taverner (Past President), Assoc Prof P.E. Hartmann, Assoc Prof D.J. Hampson, Drs S.Z. Skirrow and F.R. Dunshea. Drs A.J. Peacock, R.S. Cutler, and Ms L. Dann provided local support for the committee and this was complemented by the staff of ACTS Pty Ltd as conference organisers. Ms J.L. Ranford and Mr J. Noonan also provided invaluable assistance through the maintenance of the membership database and the production of the APSA Newsletter.

Early in 1994 APSA lost one of its most active members with the untimely death of Dr Ted Batterham. To recognise the major contribution that Ted made to the Association, particularly as Editor of the Proceedings of the 1991 and 1993 Conferences, the committee of APSA have established the Batterham Memorial. The award has been made possible through the generous financial support of the following national and international companies: BASF, Barastoc, Farmstock, Heartland Lysine (US), Purina Mills (US) and Rhône-Poulenc Animal Nutrition.

B.P. MULLAN  
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# A REVIEW - FUTURE DIRECTIONS AND RESEARCH NEEDS OF THE AUSTRALIAN PIG INDUSTRY

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## Introduction

The Australian pig industry has undergone considerable change during the last 20-25 years in both structure and efficiency. The structural changes have been continuous and rather dramatic over the last 10-20 years. They are likely to continue at a similar or even faster rate over the next 5 to 10 years. Changes in the efficiency of the industry have been in part responsible for the structural changes but have been less spectacular and absolutely flat during the last five years. The latter has made the industry vulnerable to imports from countries more efficient at producing pig meat which in turn could force more structural changes on the Australian industry. These changes have implications for all stakeholders and in this paper I have attempted to "predict" likely further changes in the industry and the impact that these will have on the future research and development needs and funding for the industry.

## Industry changes

### 1960-1994

Changes over time in the numbers of producers and sows, and in the production of pig meat in Australia are shown in Figures 1, 2 and 3 respectively.

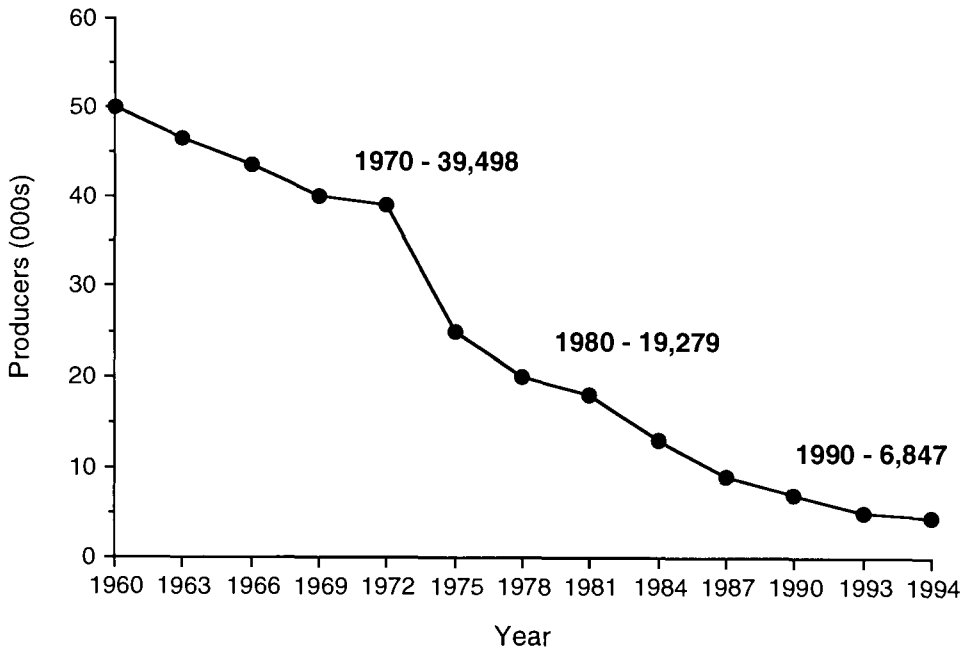


Figure 1. Changes in the number of Australian pig producers between 1960 and 1994.



The results suggest that the industry is mature. It is also clear from Figures 1 and 2 that whilst sow numbers have remained constant for the last five years producer numbers have declined by 33% to 4,600 in the same period.

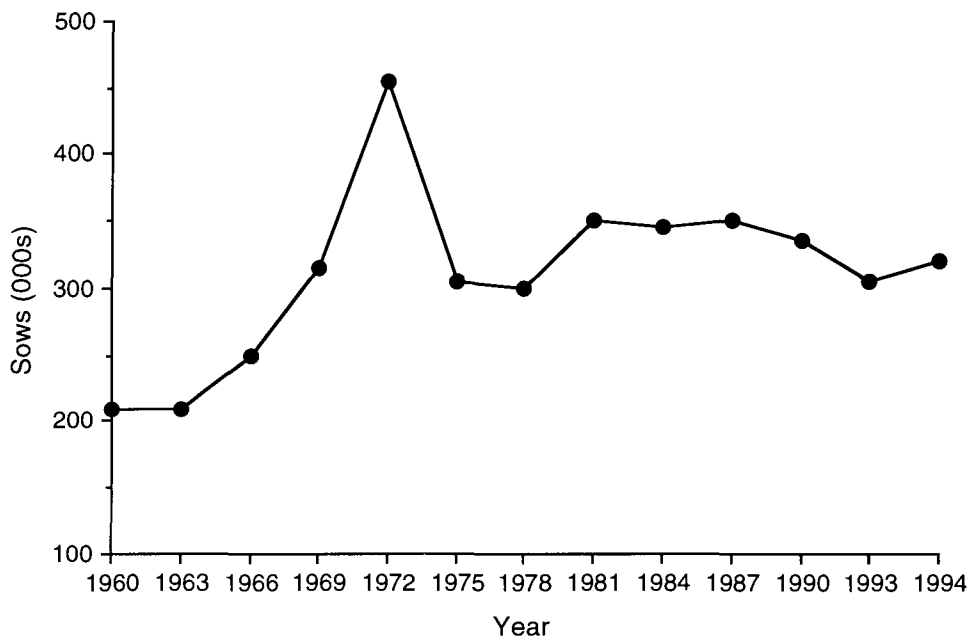


Figure 2. Changes in the size of the Australian sow herd between 1960 and 1994.

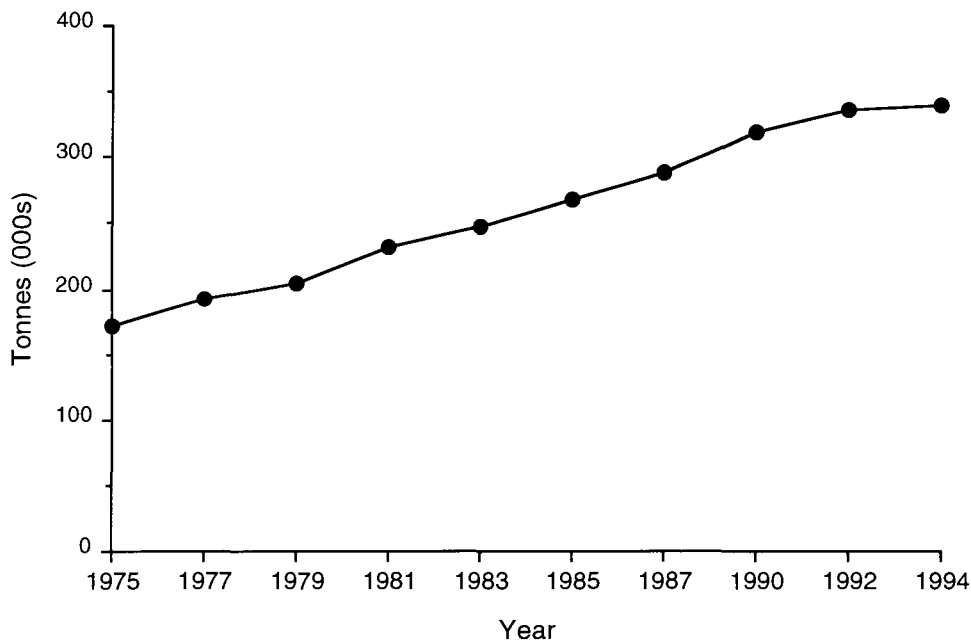


Figure 3. Changes in pig meat production in Australia between 1960 and 1994.

The rapid change in ownership of the herd over this period has been caused by the general lack of profitability of the Australian industry over the last five years. The saw tooth nature of the pig price graph (Figure 4) over the last seven years further demonstrates how the industry has matured over the same period. Price appears to be negatively related to pig supply in the same year and positively to feed prices in the previous year. The supply (Figure 3) and price (Figure 4) results also suggest the real demand for pig meat in Australia has plateaued and possibly even declined during the last five years. The ability of manufacturers to import pig meat from Canada since July 1992 has put further pressure on pig prices in Australia and consequently on the viability of what might be termed the marginal or less efficient producers in the industry. Unless the demand for pig meat can be increased and/or the cost effectiveness of production improved it would seem inevitable that the terms of trade for the industry and producer numbers will continue to decline.

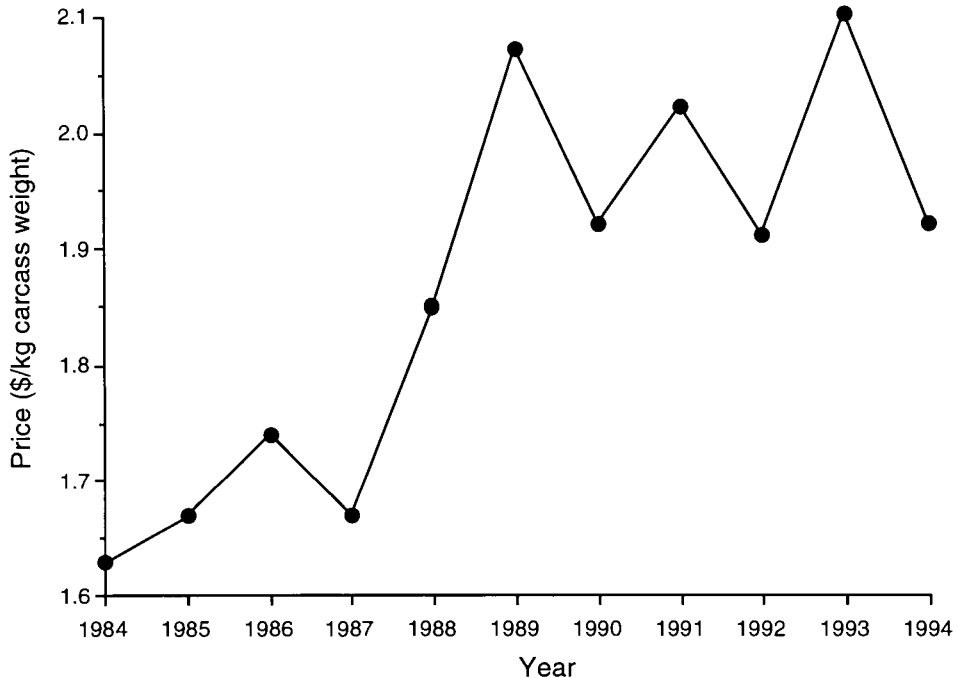


Figure 4. Changes in the farm gate price for pigs in Australia between 1984 and 1994.

#### Future changes and direction

The restructuring of the Australian pig industry which has occurred over the last 20-25 years will continue and is likely to accelerate over the next decade. There are a number of factors which will be responsible for this change. The first is the low and largely negative margins experienced by most producers during 1994 and 1995. The second is the fact that most of the larger and more efficient producers in the industry are likely to expand. The latter expansion has the potential to increase sow numbers by 20 to 25% over the next five years. The third factor will be the establishment of additional production units by companies such as Grain Co, Danpork and one or more major Japanese meat businesses. These new developments are likely to increase sow numbers over the next 5 to 8 years by as much as 50,000. It is possible that within 10 years 50 to 60% of the sows in Australia, and as much as 70% of the production, will be controlled by as few as six "producers". Unless there is a marked increase in the demand for pig meat in Australia, or for Australian pig meat/or pig meat products overseas during the same period, pig prices will fall to levels not previously experienced, and production units currently considered efficient and profitable will become non-viable.

Two of the causes of these changes are that demand for pig meat is static or falling and the industry as a whole is not globally competitive. Consequently, small increases in production by the more efficient producers will place pressure on those less efficient at producing pig meat. This pressure will be further increased by imports from Canada and the USA, and possibly even Europe in the longer term.

The relative competitiveness of the Australian, USA and Canadian industries is shown in Table 1 and indicates that Australia is considerably less competitive than the USA or Canada. Much of the pressure for change would be removed if Australian producers could produce pig meat for \$1.30 - \$1.40/kg carcass weight. The latter is not impossible and is being achieved currently by a number of Australian businesses. However, it is of concern that based on information published in the publication "Pig Stats 94", edited by Ransley and Cleary (1995), there has been little change in key performance indicators or cost of production for the industry in the last five years. The relatively high cost of production indicated by these data is one of the reasons Australia has become a target for imports. It is also a catalyst for expansion by the more efficient businesses and the entry of new players. The inevitable result of all these changes is that the industry will never be the same or as profitable or predictable as it was in the past.

**Table 1. Comparative costs of production for the Australian, USA and Canadian pig industries (Aus \$/kg carcass weight - 1994).**

Australia <sup>1</sup>	USA <sup>2</sup>	Canada <sup>2</sup>
1.91	1.58	1.61

<sup>1</sup>Ransley and Cleary (1995). <sup>2</sup>Tank (1994).

#### Implications for research and for research providers

They keys to survival in the Australian pig industry are clear, we simply have to reduce our cost of production to \$1.50/kg carcass weight or below and/or increase the real demand for pig meat in Australia and internationally. It could be argued that the need to be globally competitive demands greater expenditure and focus on research and development. However, based on data presented by Ransley and Cleary (1995) it could be argued that expenditure over the last 10 years on basic and commercial research and development has not been particularly effective in improving the efficiency or competitiveness of the industry. Based on the profitability of the Australian industry and demand for pig meat similar arguments could be levelled against the effectiveness of our promotion programmes.

However, these criticisms would be unfair because there is little doubt that both research and promotion programmes have been effective in improving productivity and demand. Indeed, the former has to some extent been responsible for the increase in production which has occurred over the last decade and in turn the fall in the average real price for pigs and change in ownership of the industry. Nevertheless, the industry has changed and the current models for research and promotion may no longer be appropriate. Furthermore, they are likely to become increasingly irrelevant and unsustainable if ownership of the industry changes as rapidly as predicted over the next 5-10 years.

**Table 2. Changes in key performance indicators and cost of production for the Australian pig industry between 1990 and 1994 (Ransley and Cleary, 1995).**

	1990/91	1991/92	1992/93	1993/94
Pigs sold/sow/year	18.7	18.8	18.5	18.6
Herd feed conversion (carcass weight)	4.30	4.23	4.27	4.26
Growth rate (from birth) (g)	537	545	552	565
Cost of production (\$/kg carcass weight)	1.81	1.88	1.89	1.91

As the number of producers decreases and herd sizes increase individual enterprises will be more able to afford, and are likely to establish their own research teams and programmes. Consequently, the need for public/industry funded generic type research, particularly in the production arena, will decline. These changes will be driven by a number of factors. The first is that the constraints on future improvements in productivity and profit differ among individual enterprises and are best alleviated or removed by research and development programmes focused on the specific problem. The second is that the results of research conducted under commercial situations are immediately applicable to the conditions under which they have been generated. The third is that technical and competitive advantages offered by such research can be protected from national and international competitors. The latter contrasts with public or industry funded research and development where the results are immediately available to our international competitors and are often implemented overseas before they are in Australia.

Changes to government or public funding of industry based research might also alter how future funds should be spent to obtain maximal producer benefits. These changes to research and development will occur. However, the rate of change will depend on how rapidly the industry restructures and individual enterprises establish their own research and development units and networks, or are able to conduct meaningful research within their production facilities.

For the reasons outlined previously production-type research will and should be conducted within commercial units rather than government or university facilities. The latter agencies should focus more on basic research because the long term future of the industry will be dependent on gaining a better understanding of the factors which control the biology of the pig and how they might be manipulated than on production type research *per se*.

Consequently, I see a shift in the focus of research and development and in how this is provided. Production type research will move from the public to the private domain whilst public funding will concentrate more on basic research and off farm factors which constrain the competitiveness of the industry as a whole. Nevertheless, even the latter will need to be conducted in conjunction with, and not independently of, industry simply because it makes much better use of human resources and limited facilities. There will also be the need during the transition for a more integrated approach to research and development between scientists and industry. The latter is currently spoken about a lot but occurs to only a small extent and is one of the reasons for the lag in the adoption of research findings.

Another change that is likely to occur is a marked decline in the need for extension services or public expenditure on communication programmes. These are not currently used by, and are of little value to, the larger production units in Australia. The relevance of such programmes will decline as the ownership of the industry contracts. Funds currently spent on these functions can and should be directed towards basic and off farm research and/or the funding of production type research in conjunction with commercial units.

Training of graduate and postgraduate students for the industry may also have to change and the Industry Placement Award concept developed recently by the Pig Industry Research and Development Corporation should be extended in preference to the more conventional postgraduate training. The former allows for education and training to be integrated with the business of producing pigs and pig meat products, the latter does not.

## Conclusions

Based on the changes likely to occur within the Australian industry over the next 10 years and beyond we are unlikely to need additional funding for research and development but a marked change in how the funds are spent. Future research and development programmes and for that matter promotion programmes should be developed within an overall industry framework and plan. They should be focused on alleviating or removing constraints to improving our global competitiveness and/or increasing demand and returns nationally. Indeed, there are likely to be periods in the future when all funds available to the industry might be better spent on increasing

national and international demand for Australian pig meat products rather than funding generic promotion or research and development *per se*.

The future success of the Australian industry and its individual participants will be very much dependent on being better than our competitors. This will in turn require an understanding of the changes that are likely to occur to the national and international pig industries, the development of integrated industry plans and programmes designed to exploit future opportunities and remove current constraints and threats, and above all being a lot more flexible than we have in the past. There will be roles for everybody in the new industry but they will be different and will require a more strategic and integrated approach than they have in the past.

### References

- RANSLEY, R. and CLEARY, G. (1995). "Pig Stats 94". (Pig Research and Development Corporation and Australian Pork Corporation: Canberra).
- TANK, A. (1994). Global opportunities for the US Pork Industry. In "Proceedings of Pork Summit '94", pp. 23-27. (A.L. Laboratories, Inc.: Fort Lee, NJ).

## **A REVIEW - ORGANISATION AND APPLICATION OF RESEARCH AND DEVELOPMENT IN COMMERCIAL PIG HERDS: THE DANISH APPROACH**

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### **Summary**

This article reports on the organisation, development and application of a unique, applied pig research scheme - the Danish Applied Pig Research scheme (DAPR) - which was initiated to complement traditional research methods. The DAPR was established in 1975 under the auspices of the Federation of Danish Pig Producers and Slaughterhouses with the purpose of conducting comparative studies of current and new developments in housing, nutrition and management, which have potential benefits for commercial pig production. National research institutes, particularly the National Institute of Animal Science, have supervised DAPR activities. In contrast to other applied pig research schemes DAPR employs a traditional research approach comprising a control group and one or several experimental groups within the same commercial herd. Most experiments employ a randomized complete block design using herd as a blocking factor. Original projects are focused on housing and feeding methods. Expansion of DAPR activities has led to other projects on such topics as the working environment, farm animal welfare and solving ventilation problems. Currently 50-60 projects, involving about 100 farms, are in operation. In general, DAPR activities have a large impact on Danish pig production.

### **Introduction**

Transfer and validation of research results in commercial pig production is an important issue in evaluating research efficiency. In order to investigate the causal relationship between production factors and animal response, detailed physiological and behavioural studies are required. Such studies are often carried out without considering herd conditions. In pig herds production is greatly affected by management. For instance, under commercial conditions feed rations may not be changed at the scheduled time, feeding may be delayed because of work load in other departments, or stocking density may increase due to random fluctuations in the number of pigs produced. As a result of differences between experimental conditions in a research organization and production conditions in a commercial enterprise, findings from basic research may be distorted or may not even be valid under production conditions. The causal relationship may still exist but the magnitude of the effects experienced in production herds are likely to be different from those observed in controlled experiments. This reasoning might explain why farmers tend to apply new production methods slowly.

Moreover, pig producers are exposed to new technologies in an ever increasing flow. New technologies might have little or no relevance to research. As a consequence, producers may have to rely exclusively on company information regarding product performance, which increases the risk of investing in merchandise of poor quality.

In Denmark three approaches have been used to solve such problems. These include the use of management information systems to compare methods of production; the use of observation herds or demonstration herds to evaluate feeding systems and new technology; and the use of commercial herds for making comparisons of all aspects of pig production. The last mentioned scheme (DAPR) was initiated in 1975. Since then it has played an important role in the validation, demonstration and dissemination of research results to Danish pig producers. The aim of this paper is to describe the current system and to present some results.

## Historical review

The first Danish initiatives regarding an applied research scheme trace back to the 1950's. Supported financially by the Marshall Aid plan a productivity foundation was formed in March 1953 to finance the establishment of 22 demonstration herds which were similar to ordinary pig herds. Simple trials were carried out to compare different feeding and housing systems. To demonstrate the results of these studies farm visits were allowed, up to twice a week.

In 1971 the demonstration herds were replaced by the so-called observation and control herds. Six observation herds were operated along similar lines to the demonstration herds, however, no visits were allowed. Trials in observation herds usually involved two experimental groups, and an assistant carried out the test treatments and recordings.

In the control herds (36 in total) no actual testing was undertaken, as the main aim was to demonstrate the value of recording systems in production management. These herds were located throughout the country, and each was supervised by a local pig advisor. Once a fortnight a technician visited the herd and assisted in data collection. The control herds were of significant importance for the development of the Danish efficiency control system.

In 1973 Denmark became a member of the European Economic Community (EEC), and this led to an expansion in Danish pig production. Although the number of pig farms was reduced from 100,000 in the early 1970's to 20,000 in 1995, production has more than doubled in this time, and in 1994 20 million pigs were slaughtered. The herd structure has also changed. Today, farms have become specialized in pigs or dairy production or mink for example, while in the early 1970's none were specialized. However, since corporate farming is not allowed, the family farm is still the mainstay of Danish agriculture. The size of herds has also increased with the majority of pigs being produced on farms with 200-1,000 sows.

Membership of the EEC has also brought about a significant increase in investment in new facilities and modernization of existing pig equipment. Also, pig production systems have become more diversified. As a result of these developments many pig producers invested in facilities and equipment of poor quality. In 1975 the National Committee for Pig Breeding, Health and Production (NC), under the auspices of the Federation of Danish Pig Producers and Slaughterhouses, decided to establish an organisation for the rapid testing and evaluation of systems 'on farm'. It was called "Den rullende Afproevning" or the Danish Applied Pig Research Scheme (DAPR).

The aims of DAPR were to conduct comparative studies of both current and alternative production systems which might prove relevant to future practice; to ensure a fast flow of information about new technologies; and to reduce the risk of pig producers making poor investments.

## Current organisation

Within the framework of the DAPR scheme 50-60 concurrent projects are carried out in more than 100 production herds which are located throughout the country (Figure 1). Projects have a duration of 6 months to 3 years. Herds participating in a project change, but frequently one herd participates in several projects. Selection of herds is undertaken in association with local pig advisors. Projects cover subjects in the fields of housing, feeding, management, cross-breed evaluation and the working environment.

**Table 1. Danish Applied Pig Research Scheme (DAPR) budget 1 October 1993 to 30 September 1994 expressed in three currencies (millions).**

Budget	DKK*	ECU†	A\$‡	Percent
Levy	20.0	2.6	4.35	80
Public funds	5.0	0.7	1.09	20

\*Danish crowns. †European currency unit. ‡Australian dollar.

The DAPR, which is controlled by the NC, employs a permanent staff of 42 people who conduct the studies. Most projects (Table 1) are financed through producer levies (9 DKK per pig), which also pays for other activities such as health services and breeding programmes. A minor financial contribution for the projects comes from government grants which support various research programmes. A large number of trials are carried out as cooperative projects involving national and international research institutes. An advisory committee of national research institutes has been appointed to provide support and advice regarding experimental design, recording and publication.

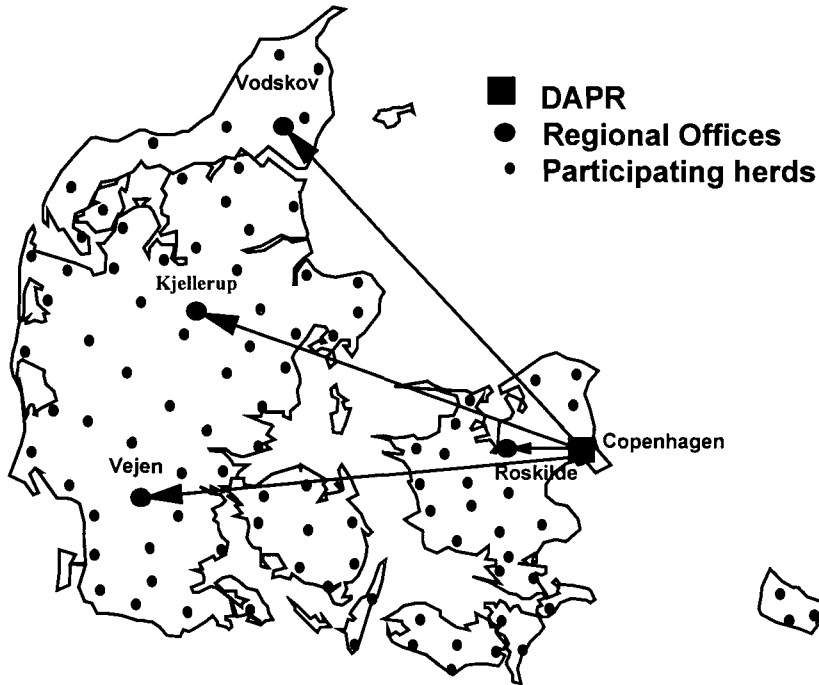


Figure 1. Location of central and regional offices of DAPR and participating herds.

Usually, a project involves two to four herds. Mostly DAPR employs farmers who intend to apply a new management or feeding system. This ensures that the producer is motivated to involve himself, his herd and staff in the research project. Economically, DAPR guarantees the farmer full financial coverage for all expenses associated with a study.

Each project employs a project supervisor and two or three technicians who carry out the recordings in the herds. The project supervisor is responsible for the detailed planning and implementation of the project from approval to publication. A recording supervisor is responsible for daily coordination of the activities of all the technicians, for the preparation of check lists, and for maintaining consistency in recordings.

## Methods

### *Project type*

Repeated trials that compare various treatments within a herd are the most common type of project. Normally, the difference between two treatments is examined, e.g. different feed components or different housing systems (Figure 2). It is difficult to carry out more than two treatments within one herd. The animals are distributed randomly between the treatments and receive the same treatment throughout the test period.



Much information is also collected through questionnaires. Feedback from questionnaires is subsequently related to production performance or disease occurrences. Additionally, close contact with herds facilitates the implementation of minor pilot projects with the purpose of obtaining first-hand impressions of new technologies prior to the implementation of a full-scale project.

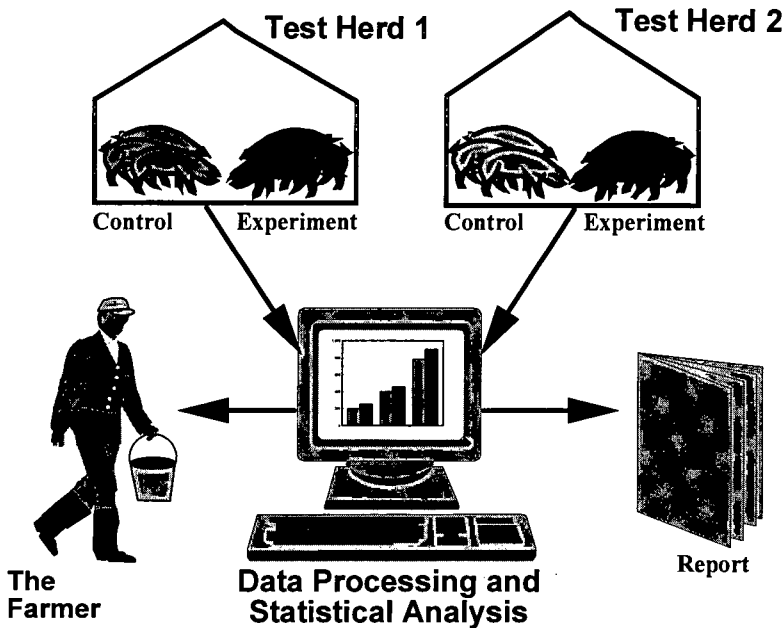


Figure 2. *Experimental design employed in most DAPR trials.*

### *Recording*

Daily recordings of production performance and certain management procedures are carried out by the herd owner, whereas DAPR technicians visit the farms every week or fortnight to record specific data such as behaviour and lesions. The technician also collects data recorded by the farm staff, and cross-analyses and discusses recordings with the staff. Furthermore, researchers from cooperating institutes collect data (e.g., Joergensen, 1992; Joergensen and Ruby, 1981; Mortensen *et al.*, 1992).

Most projects regarding housing and equipment employ specific procedures (Table 2) in addition to the general ones described above. The specific methods may be divided into those used for equipment, mechanical systems and housing principles.

Equipment evaluation involves comparison of four to six different company brands (5 to 12 units of each brand) within each farm over a period of 6 to 12 months. Evaluation criteria comprise lesion scoring (present or not present) for specific parts of the pig's integument, e.g., teats, hooves and legs for flooring; head and ears for feeders and drinkers. Behavioural measures include lying down, standing up and locomotion for flooring; aggression and accessibility associated with feed dispensers and drinking devices.

Products are also evaluated according to hygiene, ease of management, installation and short term durability. Measurements of behaviour, functional performance and management are based on a scale allowing one of four possible scores to be assigned for each criterion. As an additional activity, manufacturers are invited to meetings at farms used for equipment evaluation with the aim of discussing preliminary results.

Mechanical systems are usually not compared on a single farm. Ease of management, reliability and durability of systems are the main foci of observation in such projects.

Conventional measures, such as air temperature, relative humidity, air speed, air flow pattern and gaseous content are used when evaluating functional performance of ventilation systems. In addition, certain aspects of pig behaviour including lying, dunging and activity are recorded. Measurements used to evaluate feeding systems include feeding accuracy at different points of the feed pipeline. For housing systems the main foci of observation are productivity, management and behaviour.

**Table 2. Pig housing and equipment evaluation - measures employed in DAPR trials.**

Project type	Items	Evaluation criteria	
		Biological	Technical
Equipment	Flooring Feeders Waterers Crates	Lesions Behaviour	Functional performance Management
Mechanical systems	Ventilation Feeding Waste handling	Behaviour	Functional performance Management
Housing principles	Housing design Pen design Feeding principle Drinking principle Stocking rate	Productivity Behaviour	

As a new activity, DAPR is becoming directly involved in design, development and application of new housing systems and equipment. Such activities include supplying manufacturers with biological data, providing courses in equipment design for manufacturers, and evaluating prototypes prior to marketing.

An increasing number of recordings are automated. Thus, recordings from electronic feeders combined with video recordings of behaviour, and recordings from liquid feeding systems are transferred directly to portable personal computers (PCs). From the slaughterhouse detailed recordings of the lean meat content of each carcass section, and the skatole content (boar taint) in a sub-cutaneous fat sample are transferred from computerized classification centres.

#### *Data management*

The DAPR is highly dependent on a reliable data management system. Today the system has been modified to include a PC network system with the possibility of recording and accessing data directly from the herds (Figure 3). A strict discipline with recording data in daily operations is essential to ensure good data quality. Programmes for entering, managing and controlling trials have been developed to ensure reliable and efficient data acquisition.

It has been observed that data of the highest possible quality is obtained by adjusting the recording form to suit the working methods applied in the herds. Existing management information systems (MIS) are applied, such as Efficiency Control (IBM, 1980; Joergensen *et al.*, 1992) and the Health and Production Surveillance System (Mousing *et al.*, 1990). It is a requirement that within each project the same recording form and software must be used. Acquisition of slaughter data and data from mechanical feeding systems is performed by automatic data transfer (Mortensen *et al.*, 1992).

The processing of data is performed by means of the Statistical Analysis System (SAS, 1989a; SAS, 1989b; SAS, 1992). Several modules have been developed in SAS for

the processing of various herd analyses. Upon initiating a test it is often necessary to develop new control and management programmes for the technicians. In most cases an adjustment to existing programmes is all that is required.

### Statistical analyses

Generally, a randomized complete block design is employed in DAPR trials using herd as a blocking factor. Depending on the study, sow, pen or a complete compartment are considered as the experimental unit. Primarily, general linear models are employed in statistical analyses. Frequently, significantly different effects of treatments are observed among herds. Such interactions indirectly confirm that general conclusions should not be drawn from data from only one test herd, e.g., an experimental farm at a research station.

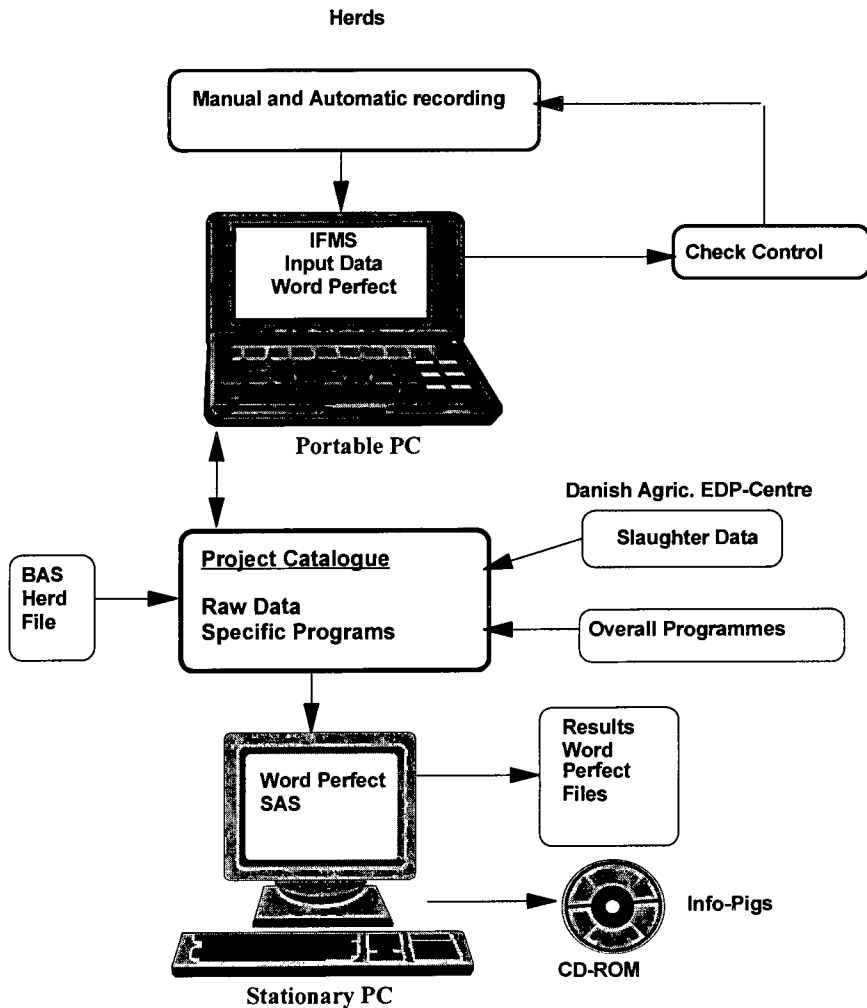


Figure 3. DAPR network and data administration.

### Dissemination of information

Results from DAPR research are published in reports called "Meddelelse fra Den rullende Afproevning" (DAPR reports), which target the pig producers and the extension service. The layout of the reports are similar to scientific articles.

Users often demand access to preliminary results. Therefore, other types of information dissemination have been developed. These include regional seminars, articles in pig trade magazines as well as brief notes. Results from DAPR research has also been incorporated in text books for agricultural colleges and in pamphlets for the extension service.

The rapid progress in information technology has led to a new initiative, which includes the development of a computer text database comprising a comprehensive store of information about pigs from Danish sources. This CD-ROM-based database, which is called "INFO-SVIN", is expected to constitute the primary information source for pig producers and the extension service in the future. INFO-SVIN will be an easily accessible medium including pictures, drawings and links between relevant topics. Information will be divided into three categories: Research reports on feeding and housing systems from Danish research institutes from 1975 onwards; processed information, i.e., materials that have been developed for the advisory service, (eg., literature on feed composition, building design, breeding strategy, pig diseases); and brief information which includes short term information such as the announcement of seminars, new initiatives by the National Committee for Pig Breeding, Health and Production and economic forecasts.

## Results

To illustrate the importance of the work of DAPR the main categories of a case study are described. The description comprises direct as well as indirect results from the project.

### *Equipment and facilities*

The DAPR quickly established itself as a significant factor within the Danish pig industry. This was partly caused by DAPR's speed in identifying trends in housing and equipment design and partly because it became a power in terms of affecting marketing of equipment. The first trials focused on housing principles for growing-finishing pigs and feeding equipment. Later, equipment testing was added as a complement to the evaluation of housing systems. A few cases are described below.

Confining sows in stalls was under heavy criticism during the 1970's (Bäckström, 1973) and led to proposals for banning stalls as well as research into ways of tethering sows. The DAPR in cooperation with the National Institute of Agricultural Engineering conducted a study of four different types of tethers including girth tethers and three categories of neck tethers (Landsudvalget for Svin, 1979; Moeller and Pedersen, 1981). The experiment was divided in two parts: Firstly an evaluation of injuries on 4 farms, and secondly an analysis of the attributes of equipment, eg., durability and maintenance, at the National Institute of Agricultural Engineering. The results of these investigations indicated that curved type neck tethers performed significantly better for all features studied than the other tethers. Results were presented at national conferences, in pig trade magazines and in research reports. The outcome was that only manufacturers and traders of the better performing tethers were able to sell them, while market forces quickly led to the withdrawal of the poor ones.

In 1985/86 neck tethers were again in focus because of reports from slaughterhouses indicating a high incidence of lesions in sows. Since pig producers at that time were reluctant to give up the use of tethers DAPR initiated a scheme for the improvement of tether design which involved an evaluation of all marketed brands (8) of tethers, meetings with manufacturing companies, as well as recording the anatomical measurements of several hundred sow necks (Landsudvalget for Svin, 1987; Landsudvalget for Svin, 1989). The outcome of these measures included standardising tether colours with respect to size of tether (black = large; blue = medium; red = small), which led to a reduction in the number of varieties marketed. Although tethers will be banned in the future (Council Directive, 1991) measures such as those initiated by DAPR, have had a positive effect on animal welfare. The result was that, later reports from slaughterhouses indicated a reduction in sows with neck lesions.

One of the main arguments for maintaining sows in confinement has been that it offers the potential for controlling individual feed intake. However, in the early 1980's electronic sow feeding systems (ESF-systems) were introduced as an alternative to confinement of gestating sows (Edwards *et al.*, 1984). Therefore, ESF was considered a major break-through for sow housing. By means of transponder-technique ESF allows sows to be individually fed while residing in groups. Early developments took place in the UK and The Netherlands and the first systems were marketed in Denmark in 1986.

The DAPR initiated trials with ESF as soon as the first systems were marketed. Information about ESF was circulated as well as warnings suggesting that farmers not invest in these systems until performance data were available. These activities apparently discouraged farmers from installing ESF. Moreover, preliminary data as well as the final report (Landsudvalget for Svin, 1990) regarding performance of animals and equipment were quite negative, suggesting that further improvement in equipment and housing design was needed before ESF might be generally recommended.

These early initiatives led to the situation that ESF was never distributed in quantities comparable to those in other countries such as the UK, The Netherlands and Sweden. One drawback was that further development in Denmark ceased because improved systems were not introduced. However, DAPR has initiated new trials of later ESF versions as well as the design of pen lay-outs (Landsudvalget for Svin, 1992).

The ESF-studies led to close collaboration with applied ethologists at the National Institute of Animal Science. While DAPR coordinated farm recruitment and data acquisition of production performance, the ethologists were responsible for recording behavioural and physiological traits as well as lesions. Apart from results reflecting performance and well-being of confined or group-housed sows, the outcome of this cooperative project was the development of methods for the rapid analysis of housing design using behaviour as a "tool" (Jensen and Pedersen, 1989). Moreover, these methods were developed for staff who had no previous experience in recording aspects of pig behaviour and these were used in later trials.

In addition to collaboration with applied ethologists the ESF studies led to cooperation with the National Agricultural Centre, Stoneleigh, UK, and resulted in the exchange of results at ESF workshops. Later studies of group-housing systems for sows have led to cooperation with researchers at Rosmalen, The Netherlands, which was an attempt to coordinate and use research funds more efficiently. While the Dutch investigated effects of group housing in detail at experimental stations, the DAPR carried out similar studies on commercial farms.

Although cooperative projects require a considerable amount of coordination, the outcome of most projects has been beneficial for both the Danish pig industry and for its partners because of the utilization of the unique competencies within individual institutes.

Equipment testing has become one of DAPR's major tasks. Several types of flooring, crates, feeders and waterers have been evaluated over the last decade. Equipment evaluation has led to close communication with equipment companies. Thus, in 1988 DAPR proposed that all equipment for pigs should be labelled with a standard declaration comprising a company description of technical data for products as well as a DAPR description of its functional performance. Although this project has been restricted by inertia at some stages, it now seems to be developing more steadily.

Feed constitutes the largest variable cost in pig production. Therefore, trials to examine different feeding systems were among the first to be instigated by DAPR. Initial studies comprised an evaluation of the accuracy of feed delivery for dry feeding and liquid feeding systems. Later investigations have encompassed testing of feeders and waterers as well as comparisons of feeding methods, i.e., dry, wet or liquid. While initial research focused on feeding systems, later trials examined feeding methods and equipment.

The DAPR has also devoted considerable efforts to evaluating flooring for all categories of pigs. This has led to the development of a comprehensive set of recommendations for the width of slots and slats, and the best types of materials to use for each category of pigs. A subset of these recommendations is presented in Table 3.

### Ventilation

Ventilation has become an increasingly important issue over the last two decades, as a result of the intensification of pig production. Thus, stocking density has increased while air volume per pig has decreased. In addition, wet surface area has increased because of the implementation of fully slatted floors. These developments have increased demands for more sophisticated ventilation systems.

In general, DAPR comparisons have indicated that production and health are not significantly affected by the type of ventilation system used, ie., negative, neutral or positive pressure systems. However, casual observations in pig herds indicate that poor health and productivity can be attributed to poor ventilation, ie., systems are not adjusted correctly or are poorly maintained.

Consequently, in 1986 DAPR initiated a trouble shooting scheme which offered producers a technical examination of their pig accommodation. Results from such investigations confirmed that most errors are as a result of incorrect calculations of air flow rate as well as lack of maintenance of ventilation equipment. These errors often contribute to poor productivity. Moreover, subsequent DAPR studies indicate that the standard values used in designing ventilation systems need to be adjusted to match the design of the building. Thus, air-moisture content is higher with fully slatted floors than with partly slatted floors because of the differences in wet surface area, which is not predicted from formulas. Heat balances for buildings indicate that practical and theoretical values differ more than would be expected, suggesting that standard values should be corrected.

**Table 3. Danish recommendations regarding flooring for different categories of pigs.**

Category of pigs	Width (mm)	
	Slat	Slot
Boars <sup>1</sup>	60-80	16-18
Gestation <sup>1</sup>		
Sows, confined	70-100	18-22
Sows, group	60-80	80-20
Farrowing <sup>2</sup>		
Plastic	10-16	10-12
Metal	10-30	10-12
Concrete	30-45	10-14
Weaners (5-30 kg body-weight)		
Plastic	10-16	10-12
Metal	10-16	10-12
Concrete <sup>3</sup>	30-45	10-14
Growers (16-50 kg)		
Metal	10-20	12-15
Concrete <sup>3</sup>	45-70	15-18
Finishers <sup>1,3</sup> (25-100 kg)	70-90	18-20

<sup>1</sup>Concrete. <sup>2</sup>Confined sows. <sup>3</sup>2 mm should be added to slot width when using straw.

In the mid 1980's DAPR initiated investigations into the working environment in pig barns. This initiative was triggered by a study at the University of Århus, which indicated that respiratory disorders were increasing among pig producers. Based on this investigation and on a concurrent international study (Donham *et al.*, 1984), DAPR in co-operation with the University of Århus conducted a survey (by questionnaire) of 4,800 pig producers as well as health examinations of 425 pig producers.

Although these examinations indicated the extent of the problem, they did not state the actual cause. Therefore, a new project comprising a combination of technical and medical examinations has been initiated so as to study the development and causes of lung disorders as well as the possibility of reducing the dust content of the environment.

#### *Feed components and nutrients*

Evaluation of diets, feed components and nutrients were initiated in 1979 with the aim of establishing nutrient standards and to evaluate the quality of diets. The first studies included evaluation of commercial feed mixtures. Feeds were selected randomly and were compared in pairs. The purpose was to monitor the feedstuff companies and to ensure that the nutrient composition of the diets would provide pig producers with the highest possible marginal returns.

In the early 1980's rations of varying composition entered the market, i.e., feed ingredients might vary within certain time intervals. Consequently, comparisons of commercial mixtures became less important as the composition of the mixtures varied from month to month. Thus, basic recommendations regarding the nutrient content of the diets for each category of pig were brought into focus so that the composition of raw materials was taken into account in ration formulation.

In recent years research has also focused on the fibre content of feed mixtures and on how feed ingredients affect male skatole production. In collaboration with the National Institute of Animal Science new projects have been initiated with the aim of studying effects of feed on gastric health as well as evaluating feeds with reduced contents of nitrogen and phosphorous.

#### *Management, health and breed evaluation*

The DAPR database may be used for purposes other than that for which it was originally intended such as studying management procedures, estimating productivity differences among herds and comparing cross-breeds. The management information systems used in Danish pig production are closely associated with the recording systems that have been developed by DAPR. A detailed description has been made by Joergensen *et al.* (1992).

Recently, new forms of management information systems have been included, eg., International Standard Organisation (ISO) standards, and "ethical accounting". An ethical account is a summary of the effects of farming on the environment and is made once a year for each farm. Also, data are utilized to a considerable degree in connection with projects under the Danish Informatics Network in Agricultural Sciences. Research concerning management has constituted an important part of the work of DAPR. Thus, efforts have been made to study the influence of various management strategies on the reproductive physiology of the breeding herd. Studies include induction of puberty in gilts, pig production based on 1st parity sows, number of services per oestrous cycle and the use of artificial insemination (Pedersen *et al.*, 1985).

Postgraduate studies of aspects of pig health have resulted in four PhD theses in the fields of mastitis, metritis and agalactia (MMA); scours; worms; and lung disorders (Jorsal, 1983; Svensmark, 1984; Roepstorff, 1986; Baekbo, 1989, respectively).

Cross-breed evaluation has also been an important part of the activities of DAPR and has required that new research methods be developed. These include the development of recording systems, eg., the Danish Efficiency Control system which can be used to analyse the effects of different breeding strategies within a herd, and also compare meat quality of various cross-breeds with reference to markets (Joergensen *et al.*, 1982). These studies are of significance to the Danish pig industry in terms of demonstrating the value of cross-breed production as compared to single breed production.

#### **Discussion and conclusion**

Trials in commercial herds have proved to be an efficient way of demonstrating and describing the effects of various treatments. The activities of DAPR in evaluating

equipment and facilities have had a considerable influence on Danish pig production and the equipment industry. Thus, the use of commercial farms as the experimental unit has been valuable in terms of adding credibility and relevance to results as well as providing cheap, but nonetheless updated facilities for research. Moreover, cooperation with national and international research institutes as well as with the Danish extension service has played an important role in terms of allowing quick transfer of research findings to production.

However, an applied research scheme such as that undertaken by DAPR encounters problems, eg., experimental variables are difficult to control in commercial herds and occasionally trials have to be stopped because of outbreaks of disease or errors in management. In addition most studies are carried out in the better performing herds simply because stringent discipline in the collection of data is required. Moreover, performance is usually improved in DAPR herds because production is more closely monitored than under normal circumstances. As a consequence, herds with average or less than average performance might not be able to reproduce findings from test herds. Any errors in the conclusions reached from DAPR research results might have far reaching implications due to the significant demonstration effect, which has been experienced with this scheme. The DAPR is part of an agricultural organization. Therefore, most studies target short-term, every-day problems on commercial farms. This sometimes results in long-term and more expensive studies, eg., studies on animal welfare, being hampered or neglected.

In contrast with other methods of obtaining information, such as between farm comparisons which are available from management information systems, DAPR's approach does have several advantages. First of all, data quality is considerably higher because of the establishment of comparable groups within a herd. Furthermore, the close contact among herd owners and agricultural technicians ensures that the producer's daily observations are recorded and made available to other producers.

The evaluation of equipment has had major implications for product development, eg., products of poor quality have been quickly removed from the market; product design and quality have been improved for items that had not previously been subject to evaluation; and products from different suppliers have tended to become more uniform in design and appearance. The last mentioned might be considered a negative effect since company creativity has been hampered because of DAPR activities. As a consequence, the Animal Centred Design Group (ACDG)<sup>1</sup> was established in 1994 under the initiative of the NC with the purpose of serving as a link between research and production by intensifying the transfer of biological data to manufacturers and producers as well as encouraging creativity. The ACDG develops design guides for equipment such as waterers, feeders, and flooring as well as organizing design seminars for equipment manufacturers.

Results from studies of different housing principles have set the trend for current pig production practice. Since the DAPR is organized to evaluate rather than develop new systems, it might be argued that it has had a conservative effect on pig housing. As a result of increasing legislation regarding animal welfare and the external environment there is a growing need for systematic development of new facilities for pigs. As a consequence, DAPR will devote more efforts in the future to developing "tomorrow's" systems in cooperation with research institutes and companies.

The success of an applied research scheme such as DAPR is highly dependent on close collaboration among industry, the extension service and research institutes, and has ensured rapid dissemination and implementation of results. The strong cooperative structure of Danish agriculture as well as a relatively uniform herd size and a short distance between farms has also been beneficial.

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<sup>1</sup>The Animal Centred Design Group is a working group which has members from the following countries: Australia, Canada, England, USA, The Netherlands, Sweden, and Denmark.



## References

- BÄCKSTRÖM, L. (1973). Environment and animal health in piglet production. A field study of incidences and correlations. *Acta Veterinaria Scandinavica*. Supplement 41:200-240.
- BAEKBO, P. (1989). Air quality and pig health in Danish pigs: Effects of air quality on pathogenesis with respect to *Pasteurella multocida*. PhD Thesis. The Royal Veterinary and Agricultural University, Copenhagen.
- COUNCIL DIRECTIVE. (1991). "Minimum standards for the protection of pigs. (91/630/EEC) L340/33-L340/38." (Commission of the European Communities: Brussels).
- DONHAM, K.J., ZAVALA D. C and MERCHANT, J.A. (1984). Respiratory symptoms and lung function among workers in swine confinement buildings: A cross-sectional epidemiological study. *Archives of Environmental Health*. 39:96-101.
- EDWARDS, S. A., ARMSBY, A.W. and LARGE, J. W. (1984). Behaviour of group-housed sows using an electronic individual feeding system. *Proceedings of the International Congress on Applied Ethology in Farm Animals*, Kiel, pp. 232-235.
- IBM. (1980). "Animal production management at Landbrugets EDB-Center". (IBM Eurocoordination S.A: Paris).
- JENSEN, K.H. and PEDERSEN, B. K. (1988). Routine ethological registrations in field trials - experience from housing systems with transponder feeding of loose pregnant sows. *Applied Animal Behaviour Science*. 21:373-374.
- JORSAL, S.E. (1983). Morbidity in sows. Epidemiologic studies in sow herds: The MMA-syndrome. PhD Thesis. The Royal Veterinary and Agricultural University, Copenhagen.
- JOERGENSEN, E. (1992). The utility value of information in pig production. In "Satellite Symposium on Pig Management Information Systems" (43rd Annual Meeting of the EAAP), pp. 135-143, eds S.M. Rillo, A. Aumaitre, L. den Hartog, G. Backus, P. Glodek, F. Saiz and A. Salehar. (EAAP: Madrid)
- JOERGENSEN, E. and RUBY, V. (1981). Simulation of different production strategies in sow units, 5 pp. *Proceedings of the 32nd Annual Meeting of the EAAP*, Zagreb.
- JOERGENSEN, E., RUBY, V., STAUN, H., PEDERSEN, O.G. and UDESEN, F. (1982). Evaluation of Danish cross breeds: Reproduction, growth, feed conversion and meat quality. 428th Report of The National Institute of Animal Science, Tjele.
- JOERGENSEN, E., RUBY, V. and HERLOEV, L. (1992). Review of Danish experiences with decision support systems in pig production, 8pp. *Proceedings of the 4th International Congress for Computer Technology in Agriculture*, Paris-Versailles.
- LANDSUDVALGET FOR SVIN. (1979). Evaluation of sow tethers. 8th Report of The Danish Applied Pig Research Scheme, Copenhagen.
- LANDSUDVALGET FOR SVIN. (1987). Annual Report. The Federation of Danish Pig Producers and Slaughterhouses, Copenhagen.
- LANDSUDVALGET FOR SVIN. (1989). Test of sow tethers. 172nd Report of The Danish Applied Pig Research Scheme, Copenhagen.
- LANDSUDVALGET FOR SVIN. (1990). Housing of gestating sows: Electronic sow feeding and stalls. 181st Report of The Danish Applied Pig Research Scheme, Copenhagen.
- LANDSUDVALGET FOR SVIN. (1992). Annual Report. The Federation of Danish Pig Producers and Slaughterhouses, Copenhagen.
- MORTENSEN, B., RUBY, V., JOERGENSEN, E. and JOHANSEN, B.B. (1992). Automatic data transfer from feeding equipment used in field test herds. In "Satellite Symposium on Pig Management Information Systems" (43rd Annual Meeting of the EAAP), pp. 55-60, eds S.M. Rillo, A. Aumaitre, L. den Hartog, G. Backus, P. Glodek, F. Saiz and A. Salehar. (EAAP: Madrid).
- MOUSING, J., ELLEGAARD, B., CHRISTENSEN, J. and PETERSEN, B. K. (1990). Management information system in Danish pig production: Pig health and productivity - a new concept. *Agrarinformatik*. 20:140-154.
- MOELLER F. and PEDERSEN, S. (1981). Sow tether evaluation. 5th Report of The National Institute of Agricultural Engineering, Horsens.
- PEDERSEN, O. G., UDESEN, F. and RUBY, V. (1985). Reproduction in 29 DAPR herds 1980-1985. Report of the Danish Applied Pig Research Scheme, Copenhagen.
- ROEPSTORFF, A. (1986). Worms in pigs: Epidemiologic studies of helminths in Danish pig herds. PhD Thesis. The Royal Veterinary and Agricultural University, Copenhagen.
- SAS. (1989a). "SAS/STAT® User's Guide Version 6", 4th edn, Volume 1. (SAS Institute: Cary).
- SAS. (1989b). "SAS/STAT® User's Guide, Version 6", 4th edn, Volume 2. (SAS Institute: Cary).
- SAS. (1992). "SAS/STAT® Software: Changes and enhancements". Release 6.07, SAS Technical Report P-229. (SAS Institute: Cary).
- SVENSMARK, B. (1984). Diarrhoea in pigs from Danish sow herds: Epidemiology with respect to rota virus. PhD Thesis. The Royal Veterinary and Agricultural University, Copenhagen.

## PIG PERFORMANCE IN LOW-COST, STRAW-BEDDED, ALTERNATIVE HOUSING SYSTEMS - PRELIMINARY RESULTS

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Low cost, welfare friendly housing systems for grower/finisher pigs, with fixed costs less than a third of those for conventional facilities, have been developed overseas using tunnel housing comprised of tubular steel framing with a plastic cover and timber side barriers (Connor *et al.*, 1994). This paper reports the first of a series of trials being conducted at the Medina Research Centre to evaluate similar systems now available in Australia for less than A\$65/m<sup>2</sup>.

Two shelters approximately 22 m × 9 m, were erected 20 m apart with their long axis orientated east-west on a green field site with a free draining sandy soil type. Batches of 150 eight-week-old weaners from a high health status, outdoor production unit were supplied on the 1st and 8th March 1995. On arrival pigs were separated by gender into groups of 75 and allotted to the shelters which were partitioned to enable groups from each batch to be reared separately at a stocking density of 1.32 m<sup>2</sup>/pig. Diets containing 0.89, 0.85, 0.74 and 0.78 g total lysine/MJ DE were fed to all pigs from 20-40 kg, 40-65 kg and to castrates and females from 66-90 kg respectively. Feed was supplied from four wet/dry feeding spaces on each side of a bulk hopper positioned in the partition at the east end of the shelters. Four bite-type drinkers were mounted on the side walls opposite the feeders. A pad of railway sleepers was laid under the feeders and drinkers with the remaining soil surfaces covered with deep straw bedding.

Table 1. Performance of pigs reared in groups of 75 on deep-straw litter in low-cost shelters at the Medina Research Centre from March to May 1995 (mean ± SD).

	Shelter 1	Shelter 2
Number beginning trial	150	150
Gender	Females	Castrates
Initial weight (kg)	21.0 ± 2.9	22.3 ± 3.6
Final weight (kg)	86.4 ± 6.7	90.7 ± 7.1
Live-weight gain on trial (g/d)	850 ± 62	901 ± 70
Whole-of-life growth rate (g/d)	634 ± 47	668 ± 52
Hot carcass weight (kg)	59.1 ± 4.9	61.9 ± 5.3
Dressing %	68.4 ± 1.7	68.2 ± 1.5
P2 (mm)	13.4 ± 3.0	16.6 ± 3.5
Feed/gain	2.42	2.57

During hot weather, temperatures inside the shelters closely followed the outside air temperature, indicating that the shelters were adequately ventilated under the conditions of this trial. However, spray cooling was provided when the temperature exceeded 24°C. Straw usage to maintain a dry lying area averaged 1.09 kg/pig/d.

Excretory behaviour was concentrated in a triangle extending from across the 3 m wide gates at the west end of the shelter, to a point about 15 m along the central partition. This behaviour may have been reinforced by the placement of the cooling system above the central partition, covering most of this area with a coarse droplet spray.

The overall mean whole-of-life growth rate was 651 g/d which compares favourably with an industry benchmark for entire males and females of 647 g/d (Ransley and Cleary, 1995). The observed performance may be partly attributed to a "new shed" effect. It will be necessary to determine whether this level of performance can be sustained over time before the economic viability of the shelters can be assessed.

### References

- RANSLEY, R. and CLEARY, G. (1995). In "Pig Stats 94", p.29. (Pig Research and Development Corporation and Australian Pork Corporation: Canberra).
- CONNOR, M.L., ONISCHUK, L., ZHANG, Z., PARKER, R.J. and ELLIOT, J.I. (1994). Forum on Innovations For Swine Housing, paper no. 94:232. (Canadian Society of Agricultural Engineers: Saskatchewan).

## THE EFFECTS OF BEDDING AND HUMAN CONTACT BEFORE PARTURITION ON THE ONSET OF PARTURITION IN SOWS

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Cronin *et al.* (1993) found that the regular application of sawdust to farrowing crates for young, pre-parturient sows, resulted in more pre-farrowing activity, a shorter parturition time and more live born pigs/litter. However, whether these effects were the result of the sawdust *per se* or human contact associated with provision of sawdust was unclear. This experiment examined the effects of sawdust as bedding for sows before farrowing, while controlling for the level of human contact when sawdust was applied.

The experiment was conducted at a commercial farm as a 2 x 2 factorial design with three replicates. The two main effects were 1) Bedding - sawdust (SDust) or no sawdust (NoSDust) provided; and 2) Application Rate - every 30 min (30Min) or twice daily at feeding times (FTime). The SDust/NoSDust treatments were applied from the aisle in front of the crates, while all observations on sow behaviour (every 5 min) and the occurrence of piglet births were recorded by observers walking quietly behind the sows. While the amount of human activity along the aisle in front of the treatment crates was regulated according to the treatments, there was no restriction on access by the piggery stockpeople during their work hours (0700 to 1600 h). Parity 1 to 5 sows that were due to farrow over a period of 3 d were included in the experiment. Treatments were allocated at random within rows (ie., four treatments with 5 to 6 sows per treatment in adjacent crates) and there were four rows across the shed. One of the inner two rows of crates, selected at random, was not used in each replicate. Observations were conducted over consecutive days in each replicate, from 0700 to 0400 h (ie., for 21h in each 24h), with a total of six observers working in pairs.

One hundred and twenty-eight of the 218 treatment sows were observed to farrow. There was a significant effect of Bedding on the proportion of sows that farrowed. Proportionally more SDust than NoSDust treatment sows farrowed (78 of 109 and 50 of 109, respectively; ( $\chi^2_1=14.8$ ,  $P<0.01$ ) but there was no effect of Application Rate (62 of 106 and 66 of 112, respectively for the 30Min and FTime treatments;  $P>0.05$ ). While there were no differences resulting from the main effects on litter size born (total or alive), there were trends for sows to have a shorter mean duration of parturition (adjusted for litter size) as a result of Bedding (171 vs 206 min/sow, respectively for SDust and NoSDust) and Application Rate (164 vs 205 min/sow, respectively for 30Min and FTime).

The finding that proportionally fewer sows in the NoSDust treatments farrowed during the three-day observation periods was unexpected. The result may have been as a result of the increased level of human activity behind the sows (associated with recording behaviour observations at 5-min intervals), suggesting that at least some sows may have been disturbed by the observers, and delayed the onset of parturition until the experiment was over. The proportionally greater number of sows farrowing in the SDust treatments may therefore have been a consequence of the availability of sawdust bedding facilitating the process of parturition by diverting the sows' attention away from the human distractions. Clearly, our understanding of the (physical and human) factors that influence farrowing behaviour of sows is limited and requires further investigation in modern intensive farrowing systems.

### References

- CRONIN, G.M., SCHIRMER, B.N., MCCALLUM, T.H., SMITH, J.A. and BUTLER, K.L. (1993). *Applied Animal Behaviour Science*. 36:301-315.

## EFFECT OF ENCLOSURE OF THE FEEDING SPACE ON FEEDING BEHAVIOUR OF GROWING PIGS

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Feed wastage has been identified as an important problem in the Australian pig industry. Estimates of wastage range from 4 to 30% (Payne, 1991), with an industry average probably in the region of 10 to 15%. Much wastage is the result of poor feeder design.

Single space wet and dry feeders are attracting increasing attention as pigs perform as well or better with these feeders compared with conventional multispace feeders (Taylor *et al.*, 1994). Possible advantages of single space design are greater protection for the head and shoulders whilst feeding, and availability of water, reducing the need to move away during a meal. A possible disadvantage of single space design is that the pig must feed in visual isolation from other pigs.

This experiment was designed to investigate whether enclosure of a single feeding space influenced feed intake and behaviour of growing pigs. Individual pigs (20-25 kg) were housed in 1.2 x 2.0 m pens on a concrete floor with access to two single space tunnel feeders. The feeding space opening was 285 mm wide x 425 mm high x 250 mm deep. One feeder was closed with a solid rear wall; the other was open, so that the feeding pig could see through it to an adjacent pen.

Three feeding conditions were compared: 1. Pigs could see through the open feeder to an empty pen (control condition). 2. Pigs could see through the open feeder to an adjacent pen of two pigs. 3. Pigs could see through the open feeder to an adjacent pen of one pig, which could feed from a similar open feeder placed back-to-back with the experimental feeder. Eight pigs were observed on each condition for eight days. Daily intake was recorded as the percentage eaten of the amount offered in each feeder and behaviour was sampled for two random 24 h periods using time lapse video.

**Table 1. Effect of feeder view on mean daily intake and number of feeding events in open and closed single space feeders.**

Feeding condition	Intake (%)			Feeding events		
	Open	Closed	Significance	Open	Closed	Significance
No view of pigs	30.2	26.7	NS	70.4	66.4	NS
View of two pigs	41.5	21.0	***	63.5	98.8	*
View of adjacent feeding pig	36.9	20.5	***	53.6	102.8	*

NS=Not significant; \*= $P < 0.05$ ; \*\*\*= $P < 0.001$ .

Pigs had higher intakes in open feeders when they could see other pigs through the open feeder (Table 1). There were no differences in intake between open and closed feeders when no other pigs were visible. Pigs also performed more feeding events (defined as head within the feeding space) in closed feeders when other pigs were visible through the open feeder.

Views of other pigs clearly affect feeding behaviour. Intake in open feeders is stimulated, presumably by social facilitation (Hsia and Wood-Gush, 1983). Wastage may also be influenced by enclosure of the feeding space. An increase in feeding events, and hence feeder withdrawals, will provide a greater opportunity for feed loss to occur.

### References

- PAYNE, H. (1991). Study tour report: single space wet and dry feeders. Report to the Pig Research and Development Corporation.  
 TAYLOR, G., KRUGER, I. and FERRIER, M. (1994). "Plan it - Build it". (NSW Agriculture: Tamworth).  
 HSIA, L.C. and WOOD-GUSH, D.G.M. (1983). *Animal Production*. 37:149-152.

## TURN-AROUND STALLS AND THE WELFARE OF PIGS

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A criticism of conventional stall housing for pregnant sows is that sows are unable to turn around. This experiment evaluated welfare (assessed predominantly on any evidence of a chronic stress response) of pregnant gilts in two designs of stall that allowed them to turn around (McFarlane *et al.*, 1988; Johnson *et al.*, 1990) and compared them with tether stalls of a design known to produce a chronic stress response (Barnett *et al.*, 1991) and group housing. One of the stall designs approximated the commercial MoorComfort® gestation stalls. While over 12,000 MoorComfort® stalls are in use in the USA, no comparative production data are available. Anecdotal reports from producers using these stalls suggest reduced feet and leg problems, improved acceptance by gilts of individual confinement, improved conception rates and a reduced stillbirth rate. Future controlled studies are required to validate these claims.

The four treatments were imposed four weeks post-mating: 1) Group - five groups of four pigs with a space allowance of 1.4 m<sup>2</sup>/pig; 2) Tether - tether stalls with vertical bars separating neighbouring pigs; 3) Diagonal - three conventional stalls (each 2 x 0.66 m) were divided diagonally to make two fixed stalls with sufficient space for gilts to turn around; 4) Hinged - a modified conventional stall, similar to the MoorComfort® stall, in which the stall sides were hinged at 60 cm from the front creating a swing partition between the rear 2/3rds of adjacent stalls which allowed a gilt to "borrow" space sufficient to turn around. Treatments 2-4 had eight pigs per treatment and all treatments were in a naturally lit building with open flush channels in the rear of each stall/pen. Blood samples were collected via catheters on days 30 and 60, at 1 h intervals from 0800-1700 h to obtain a "daytime average" of free cortisol concentrations. Adrenal responsiveness was assessed the next day on the basis of the total cortisol concentration measured 60 min after an IM injection of 75 IU adrenocorticotrophic hormone (ACTH; Synacthen, Ciba Geigy).

Table 1. Effects of housing treatment on daytime free cortisol (F) concentrations and the total cortisol response to ACTH (nmol/L; log(x+1) transformed data for the hormone data; n=5 for treatment 1 and n=8 for treatments 2-4).

Parameter	Tether	Group	Diagonal	Hinged	SEM
Daytime free F	2.36 <sup>by</sup>	1.76 <sup>axy</sup>	1.65 <sup>x</sup>	1.56 <sup>x</sup>	0.236
F response to ACTH	6.33 <sup>y</sup>	5.68 <sup>x</sup>	5.71 <sup>x</sup>	5.92 <sup>x</sup>	0.211

ab, xy denote significant differences at P<0.05 and P<0.01 respectively.

The daytime profile of free cortisol and the cortisol response to ACTH were significantly higher in the Tether than other treatments (P<0.05). The lack of significant differences among the Group, Diagonal and Hinged treatments suggests that turn-around stalls may be a satisfactory option for the welfare of sows.

### References

- BARNETT, J.L., HEMSWORTH, P.H., CRONIN, G.M., NEWMAN, E.A. and McCALLUM, T.H. (1991). *Applied Animal Behaviour Science*, **32**:23-33.  
 McFARLANE, J.M., BOE, K.E. and CURTIS, S.E. (1988). *Journal of Animal Science*, **66**:326-333.  
 JOHNSON, R.W., CURTIS, S.E., BALSBAUGH, R.K. and TAYLOR, I.A. (1990). *Journal of Animal Science*, **68** (Supplement 1):263.

## IMPROVING PIGLET SURVIVAL THROUGH INTENSIVE SUPERVISION AT FARROWING

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Most deaths in pig herds occur within 4 d of birth, as a result of stillbirths, starvation, diarrhoea or crushing by the sow (English and Morrison, 1984; Spicer *et al.*, 1986). Jackson (1975), emphasised that obstetrical attendance minimised stillbirths by reducing sow dystocia. Cerne and Jochle (1981), suggested that prostaglandins, used to synchronise farrowings, may also reduce stillbirths and pre-weaning deaths, without supervision. This study, conducted on a 1200-sow herd in Minnesota, assessed the effects of induction and intensive farrowing supervision, on pre-weaning deaths.

During a 12-week period, 251 sows and gilts were randomly assigned to 1 of 4 treatments in a 2 x 2 factorial design; (1) supervised/induced, (2) supervised/non-induced, (3) unsupervised/induced, and (4) unsupervised/non-induced. Supervised sows/litters were observed constantly from at least 3 h before to 3 d after farrowing. Induced sows were injected with 250 µg of cloprostenol into the vulva mucosa.

There was an increase ( $P=0.012$ ) in the number of piglets weaned from supervised groups (10.17 piglets/litter) compared to unsupervised groups (9.45 piglets/litter) (Table 1). This increase was as a result of fewer stillbirths ( $P=0.001$ ) and pre-weaning deaths ( $P=0.026$ ), as less piglets were overlain ( $P=0.009$ ) and more low birth weight, low viability pigs survived ( $P=0.003$ ). Induction did not affect the pre-weaning death rate in unsupervised groups. Assuming the marginal cost of rearing a piglet to 95 kg live-weight is 75% of the total variable cost of \$1.43/kg (Ransley and Cleary, 1995), the profit from extra pigs is \$0.52/kg live-weight (\$49.40/pig), assuming a sale price of \$1.59/kg live-weight (Ransley and Cleary, 1995). The results of this study suggest that intensive supervision at farrowing provides an opportunity to improve production efficiency on pig farms, through increased piglet survival.

**Table 1. Least squares means ( $\pm$  SEM) of piglet production and loss parameters for induced/supervised sows.**

Supervised	-	-	+	+
Induced	-	+	-	+
No. sows/gilts	64	63	62	62
Total born/litter	11.51 (0.35) <sup>a</sup>	11.17 (0.35) <sup>a</sup>	11.18 (0.35) <sup>a</sup>	11.08 (0.35) <sup>a</sup>
Live born/litter	10.58 (0.34) <sup>a</sup>	10.31 (0.34) <sup>a</sup>	10.66 (0.34) <sup>a</sup>	10.77 (0.34) <sup>a</sup>
Stillbirths/litter	0.76 (0.11) <sup>a</sup>	0.6 (0.11) <sup>a</sup>	0.35 (0.11) <sup>b</sup>	0.17 (0.11) <sup>b</sup>
PW deaths/litter	1.39 (0.19) <sup>a</sup>	1.2 (0.19) <sup>a</sup>	0.75 (0.19) <sup>b</sup>	0.98 (0.19) <sup>b</sup>
Total weaned/litter	9.32(0.28) <sup>a</sup>	9.57(0.29) <sup>a</sup>	10.41(0.29) <sup>b</sup>	9.93 (0.29) <sup>b</sup>

<sup>ab</sup>Means in a row having different superscripts are significantly different  $P<0.05$ .

### References

- CERNE, F. and JOCHLE, W. (1981). *Theriogenology*. 16:459-467.  
 ENGLISH, P.R. and MORRISON, V. (1984). *Pig News and Information*. 5:369-375.  
 JACKSON, P.G.G. (1975). *Veterinary Record*. 97:411-412.  
 RANSLEY, R. and CLEARY, G. (1995). "Pig Stats 94". (Pig Research and Development Corporation and Australian Pork Corporation: Canberra).  
 SPICER, E.M., DRIESEN, S.J., FAHY, V.A., HORTON, B.J., SIMS, L.D., JONES, R.T., CUTLER, R.S. and PRIME, R.W. (1986). *Australian Veterinary Journal*. 63:71-75.

## A COMPARISON OF INDOOR AND OUTDOOR PIG PRODUCTION SYSTEMS IN NEW ZEALAND

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Producers farm pigs to make money. Before they invest, farmers need to assess the costs of entry, potential profits and risks during production and costs of exit. There has been a major investment made in outdoor units in recent years. Outdoor units have lower set-up and exit costs than intensive indoor units. This survey was designed to compare investment variables of different pig production systems.

Data were obtained from 5 indoor, 5 outdoor and 13 combined indoor/outdoor pig farms. Gross margin (GM), return on capital and internal rate of return were used to compare the financial performances of the different systems.

Indoor systems produce higher GMs per sow than either combined systems or outdoor systems (\$4,007 versus \$2,931 and \$2,016 respectively; Table 1). Higher gross income (ie., more pigs sold per sow and year) was the main contributor to the difference in GMs. Information from outdoor units showed that the reduced number of pigs per sow was the result of fewer litters per sow, fewer pigs born per litter and a higher pre-weaning mortality, but not the result of a younger herd.

**Table 1. Average financial and production indicators for indoor, combined and outdoor pig production systems.**

	Indoor	Combined	Outdoor
Income/sow	\$4,007	\$2,931	\$2,016
Expenses/sow	\$2,809	\$2,427	\$1,465
Gross margin/sow	\$1,119	\$504	\$551
Capital/sow	\$6,594	\$5,465	\$2,334
Return on capital	20.4%	11.6%	18.1%
Internal rate of return	15.0%	6.4%	15.3%
Feed <sup>1</sup>	67%	59%	63%
Labour <sup>1</sup>	14.9%	21.4%	24.4%
Litters/sow/year	2.19	2.01	1.89
Pigs sold/sow/year	21.1	18.7	16.5

<sup>1</sup>Expressed as a percentage of total expenses.

Expenses for labour, animal health, replacement stock, levies, electricity, repairs and maintenance and effluent were found to increase as the system type moved from outdoors to indoors. The average capital value of the three systems was significantly higher for indoor systems and combined systems compared to outdoor systems (Table 1). Subsequently, the return on capital was found to slightly favour indoor system (at 20.4%) over outdoor systems at 18.1%. However, when sow capital costs were made common, the internal rate of return was found to favour outdoor systems (15.3%) over indoor systems (15.0%) over combined systems (6.4%).

It was concluded that although the gross margin for indoor systems was greater, the outdoor systems may provide a better potential long-term investment when other factors such as risk, animal welfare, environmental sustainability, costs of exit and depreciation are included in the analysis.

## A NEW METHOD FOR DETERMINING AVAILABLE LYSINE IN FEEDSTUFFS

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In feedstuffs that have undergone processing, lysine can react with other compounds to become nutritionally unavailable. Some of these reacted lysine derivatives are acid-labile and revert back to lysine during the acid hydrolysis step of amino acid analysis, leading to inaccuracy in digestible lysine estimates (Hurrell and Carpenter, 1981). Furthermore, chemical assays that determine reactive lysine do not measure available lysine since unaltered lysine is not completely absorbed from the small intestine of the pig (Moughan, 1991). A method for determining digestible reactive lysine (available lysine) has been developed<sup>†</sup> (Moughan and Rutherford, 1996). The method involves collection of ileal digesta and the determination of reactive lysine in the diet and digesta based on the guanidination reaction.

Digestible reactive lysine (new assay) and digestible total lysine (conventional amino acid analysis) were determined in 150 g body-weight Sprague Dawley male rats given different feedstuffs as the sole source of protein in semi-synthetic diets. Ileal digesta samples were collected at slaughter. Endogenous amino acid flows at the terminal ileum were determined following the feeding of enzymatically hydrolysed casein to rats and ultrafiltration of the ileal digesta (Moughan *et al.*, 1990). Chromic oxide was included in the diets as an indigestible marker.

**Table 1.** Mean true ileal digestible total and reactive lysine (g/kg feedstuff) in processed feedstuffs as determined in the growing rat (n=8).

Protein source	Digestible total lysine	Digestible reactive lysine	Overall SE	Significance <sup>1</sup>
Blood meal (BM)	85.9	85.1	0.52	NS
Soya bean meal (SBM)	30.5	31.2	0.17	**
Heated skim milk powder (HSMP) <sup>2</sup>	19.8	16.6	0.25	***
Cottonseed meal (CSM)	12.9	10.3	0.42	***

<sup>1</sup>NS, non significant,  $P > 0.05$ ; \*\* $P < 0.01$ ; \*\*\* $P < 0.001$ . <sup>2</sup>Autoclaved at 121°C for 3 min.

Digestible total lysine and digestible reactive lysine in SBM and rotary dried BM were similar (Table 1). These results suggest that these feedstuffs either, do not contain appreciable amounts of altered lysine or, that the altered lysine derivatives, if present, are generally acid stable. For more highly processed feeds like CSM and HSMP, the digestible reactive lysine content was significantly lower than the digestible total lysine content, reflecting the presence of acid-labile Maillard-type lysine derivatives. For such feedstuffs, conventional ileal digestible total lysine overestimates lysine availability and, in this study, the overestimation of available lysine was 19% for HSMP and 25% for CSM. The new true ileal digestible reactive lysine assay offers promise for predicting lysine availability in processed feedstuffs.

### References

- HURRELL, R.F. and CARPENTER, K.J. (1981). *Progress in Food and Nutrition Science*. 5:159-176.  
 MOUGHAN, P.J. and RUTHERFURD, S.M. (1996). *Journal of Agricultural and Food Chemistry*. (In press).  
 MOUGHAN, P.J. (1991). In "Recent Advances in Animal Nutrition", p. 45, eds W. Haresign and D.J.A. Cole. (Butterworth-Heinemann: Oxford).  
 MOUGHAN, P.J., DARRAGH, A.J., SMITH, W.C. and BUTTS, C.A. (1990). *Journal of the Science of Food and Agriculture*. 52:13-21.

<sup>†</sup>NZ patent application number 272486.



## THE USE OF NEAR INFRARED REFLECTANCE SPECTROSCOPY TO PREDICT THE TRUE ILEAL DIGESTIBILITY OF AMINO ACIDS IN OILSEED MEALS FOR PIGS

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Precise definition of the amino acid requirements for growing pigs is important because of the animals high amino acid requirements, and the need to reduce the protein content of diets so as to minimise nitrogen excretion. Use of true ileal digestibility (ILD) values of amino acids in feed materials when formulating rations increases the precision of estimating digestible amino acids in feeds.

Several sets of results for the ILD of amino acids, based on *in vivo* measurements in pigs and cockerels, have been published (Rhône Poulenc Animal Nutrition, 1993; Institut Technique des Céréales et des Fourrages, 1995). The results are presented as mean ILD values for a range of raw materials. *In vivo* measurement of the ILD of amino acids in feed materials is time consuming. However, while the use of mean tabular values is an improvement on faecal digestibility values, it is not as precise as measurements made on individual raw materials.

Using near infrared reflectance spectroscopy (NIRS) the ILD of individual amino acids of 17 samples of oilseed meals were measured in male pigs and a further 27 different oilseed meals were measured in cockerels (Rhône Poulenc Animal Nutrition, 1993). Using a NIRS 6500 (Perstorp Analytical) together with the NIRS 2.00, version 3.00 (ISI) software, prediction calibrations were developed, together with the calibration performance parameters; correlation coefficient ( $r^2$ ), standard error of calibration (SEC) and standard error of cross validation (SECV; % of fresh weight).

**Table 1. Prediction of total concentration of individual amino acids, ILD of lysine and methionine in oilseed meals for pigs and poultry using NIRS.**

	No. of samples	Mean (%) as fed	$r^2$	SEC	SECV
Lysine - (total)	55	2.14	0.95	0.15	0.17
Methionine - (total)	52	0.63	0.84	0.04	0.07
Lysine - (pigs)	17	1.65	0.98	0.09	0.15
Methionine - (pigs)	22	0.55	0.95	0.02	0.05
Lysine - (poultry)	27	1.56	0.98	0.10	0.13
Methionine - (poultry)	30	0.52	0.84	0.04	0.09

It is concluded that NIRS is capable of predicting total and ILD of amino acids in a range of oilseed meals. The main limitation of the technique appears to be the requirement to perform *in vivo* measurements to construct calibration sets which encompass the complete range of raw materials likely to be encountered. Calibrations obtained for different classes of raw materials are more accurate than the global calibration of oilseed meals presented above. The use of NIRS permits rapid determination of ILD of individual amino acids in raw materials, enhances the precision of ration formulation and assists in the choice of different sources of raw materials.

### References

- RHÔNE POULENC ANIMAL NUTRITION. (1993). "Rhodimet Nutrition Guide". (Rhône Poulenc Animal Nutrition: Paris).
- INSTITUT TECHNIQUE DES CÉRÉALES ET DES FOURRAGES (1995). "Digestibilité iléale des acides aminés des matières premières". (Institut Technique des Céréales et des Fourrages: Paris).

## ALGAL PROTEINS LABELLED WITH $^{15}\text{N}$ CAN BE USED TO QUANTIFY ENDOGENOUS PROTEIN LOSS FROM THE SMALL INTESTINE IN PIGS

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The secretion of endogenous proteins, predominantly secreted enzymes, mucins and sloughed mucosal cells, into the intestinal lumen is variable and depends on the physical, chemical and immunological characteristics of the diet. Labelling techniques are required to study endogenous protein flow from the intestine as it cannot be distinguished from undigested dietary protein in digesta. The synthesis and secretion of endogenous proteins may have a significant energy cost to the host and, as the amino acids contained in the endogenous proteins are not generally absorbed intact from the hindgut, the flow of endogenous proteins out of the ileum may be a major factor determining amino acid requirements. Recently, de Lange *et al.* (1992) and Schulze (1994) reported the effects of dietary components on endogenous protein secretion using the  $^{15}\text{N}$  dilution technique. These authors and others have used single amino acids to label the endogenous proteins and have recognised that improved precision could be obtained by using a complete mixture of amino acids. The purpose of the present study was to evaluate algal proteins uniformly labelled with  $^{15}\text{N}$  for use in labelling endogenous proteins and then to assess the effect of two sources of protein on endogenous protein secretion in young pigs.

Lyophilized, protein-rich blue green-algae, *Spirulina platensis*, were obtained from a commercial source where they had been grown on media containing  $^{15}\text{NO}_3^-$ . The algae were homogenized in deionised water and agitated for 24 h to solubilize the proteins. The proteins were then enzymically hydrolyzed using a non-specific protease. An amino acid infusate was prepared from the hydrolysate by ion-exchange chromatography and filter sterilisation. Six castrated, male 14-day-old Large White  $\times$  Landrace pigs (mean initial body-weight = 3.4 kg) were weaned onto a commercial solid milk replacer diet. After 5 d the pigs were surgically prepared with jugular catheters and a T-shaped cannula in the terminal ileum. From 5 d after surgery each pig was continuously infused intra-venously with the  $^{15}\text{N}$  amino acid mixture. At the time the infusion commenced, 3 pigs were offered the commercial milk replacer substituted with casein (CAS) at 8% and the other 3 pigs the same milk replacer with soya bean protein isolate (SOY) substituted at 8%. On the fifth day of feeding, a 6-hour, total ileal digesta collection was performed. The dietary treatments were then reversed and fed for a further 5 d and another ileal digesta collection was performed. Blood samples were taken at the same time as the digesta collections for determination of plasma free amino acid  $^{15}\text{N}$  enrichment as an estimate of the enrichment of the precursor pool for endogenous protein synthesis as discussed by Schulze (1994). Plasma and digesta  $^{15}\text{N}$  enrichment was determined, after Dumas combustion, using a Europa gas-isotope-ratio mass spectrometer.

No significant effect of order of feeding was found and therefore the results for each protein source were pooled. The mean ( $\pm$  SEM) ratio of enrichment of digesta: enrichment of plasma free amino acids for the pigs fed the CAS diet was  $0.200 \pm 0.025$  and for the SOY diet was  $0.190 \pm 0.044$ . These results indicate that on milk-replacer based diets approximately 20% of the protein found at the terminal ileum was of endogenous origin and this was not greatly altered by changing the protein source. The continuous infusion of a complete mixture of  $^{15}\text{N}$  amino acids is a practical method for use in estimating the contribution of endogenous proteins to total protein in ileal digesta.

### References

- DE LANGE, C.F.M., SAUER, W.C., SOUFFRANT, W.B. and LIEN, K.A. (1992). *Journal of Animal Science*. 70:1848-1856.
- SCHULZE, H. (1994). Endogenous ileal nitrogen losses in pigs: Dietary factors. PhD Thesis. Wageningen Agricultural University.

## TRUE ILEAL DIGESTIBLE SULPHUR AMINO ACID REQUIREMENTS FOR YOUNG PIGS

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Formulation of diets based on total sulphur amino acid (TSAA) requirements, rather than on the individual amino acids, methionine and cystine, has been considered adequate for achieving efficient lean tissue growth in pigs. The objective of the present work was to assess the effects of the ratios of true ileal digestible (Green *et al.*, 1987) methionine (IDM) plus cystine (IDC) to lysine (IDL) on TSAA requirements for the efficient growth of young pigs.

A two by four factorial experiment with 96 female and 96 castrated male Large White × Landrace pigs, 13 to 30 kg live-weight, was conducted using four ratios of ileal digestible total sulphur amino acids (IDSAA; 0.44, 0.50, 0.55, 0.61) to IDL. The ratios were obtained using two levels of IDC, 2.00 (LC) and 2.90 (HC) g/kg feed, which were supplied at four levels of IDM: 2.85, 3.45, 4.05, 4.65 g/kg feed for diets LC1, LC2, LC3, LC4, and 1.95, 2.55, 3.15, 3.75 g/kg feed for diets HC1, HC2, HC3, HC4 respectively on an as fed basis. The diets, which were based on cereal, manioc, peas and soya, were formulated to contain 11g IDL/kg, 14.13 MJ DE/kg and 166 (LC) or 192 (HC) g protein/kg feed as fed. Pigs were randomly allocated in pairs of the same gender. Food was provided *ad libitum* as 2.5 mm diameter pellets. Nitrogen balance was estimated using eight castrated male pigs to evaluate the four low and four high cystine diets in two, four by four, latin square experiments respectively. Data were analysed for a randomised, complete-block-design experiment by analysis of variance; pair-wise comparisons were done by the Newmans-Keuls test.

Table 1. Effect of methionine plus cystine to lysine ratios on food intake and growth performance of female and castrated-male grower pigs (mean ± SED).

Diet	LC1	LC2	LC3	LC4	HC1	HC2	HC3	HC4		
IDSAA:IDL	0.44	0.50	0.55	0.61	0.44	0.50	0.55	0.61		
TSAA (% of the diet)	0.57	0.63	0.69	0.75	0.60	0.66	0.72	0.78		
IDM:IDL	0.26	0.31	0.37	0.42	0.17	0.23	0.29	0.34	SED	P value
Food intake (kg/day)	1.13	1.16	1.06	1.04	1.18	1.08	1.16	1.10	0.032	0.03
DLWG <sup>1</sup> (g/day)	618	665	611	582	571	548	646	648	16.4	<0.001
FCR <sup>2</sup>	1.82	1.75	1.74	1.78	2.06	1.97	1.81	1.69	0.021	<0.001

<sup>1</sup>DLWG; daily live-weight gain. <sup>2</sup>FCR; food conversion ratio.

The poorest performance was obtained with diets HC1 and HC2 and corresponded to the highest levels of urinary nitrogen excretion. Urinary nitrogen was 0.921, 0.752, 0.620 and 0.608 g/day/kg<sup>0.75</sup> (SED = 0.0494; P<0.05) respectively for diets HC1, HC2, HC3 and HC4. Overall, DLWG and FCR were optimal at an IDSAA:IDL ratio of 0.61 for the HC diets, and growth rate was greatest at an IDSAA:IDL ratio of 0.50 for the LC diets. There appears to be a closer relationship between animal performance and the IDM:IDL ratio than cystine levels. The DLWG was greatest at an IDM:IDL ratio of 0.31, and FCR was lowest at a ratio of 0.34. No effect of cystine was observed and, therefore, it is unlikely to be limiting in commercial feed formulations up to 30 kg live-weight. However, formulation of diets for sulphur amino acids without taking the ileal digestible methionine:lysine ratio into consideration could lead to impaired performance.

### References

GREEN, S., BERTRAND, S.L., DURON, M.J.C. and MAILLARD, R. (1987). *Journal of the Science of Food and Agriculture*. 41:29-43.

## EFFECT OF LUPIN KERNELS ON THE APPARENT ILEAL DIGESTIBILITY OF AMINO ACIDS BY GROWING PIGS

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At present, crude, and neutral-detergent fibre are the two main descriptors used to define the fibre composition of feed ingredients used in pig diets. Dietary fibre, however, is far more complex. As well as cellulose and hemicelluloses, other fractions, eg., water-soluble polysaccharides, resistant starches and the oligosaccharides can have fibre-like properties. Lupins have high levels of dietary fibre compared to other legumes (Evans *et al.*, 1993) that are often not accounted for when formulating pig diets. In particular, soluble non-starch polysaccharides (NSP) from lupins may increase the viscosity of digesta hindering the action of digestive enzymes and may affect microbial activity and hence influence the digestion of other nutrients. This experiment was to determine the effect of lupin kernels, a source of soluble NSP, on the digestion of dietary amino acids.

Four sorghum-based diets were formulated to contain 0, 12, 24 or 36% lupin kernels (*Lupinus angustifolius* cv. gungurru). Dietary crude protein was equalised at 187 g/kg (air-dry basis). Roller-milled lupin kernels were added at the expense of casein and starch, and acid-insoluble ash (added Celite®) was used as a marker. Ileal digestibility of dietary amino acids were determined using 16 Large White male pigs (40-45 kg body-weight) fitted with simple T-piece ileal cannulas using the technique described by van Barneveld *et al.* (1994). Diet allocations were based on a randomised block design (four pigs/diet). Diets were fed for 7 d prior to 8 h digesta collections over two consecutive days. Faeces sub-samples were collected during the digesta collection period.

**Table 1. The effect of lupin kernels included in sorghum-based diets at 0, 12, 24 or 36% on the apparent ileal amino acid digestibility coefficients in growing pigs.**

Kernel inclusion (%)	0	12	24	36	Diet <sup>a</sup>	Linear <sup>b</sup>	SEM
Threonine	0.87	0.87	0.88	0.84	NS	NS	0.017
Valine	0.90	0.91	0.90	0.87	NS	NS	0.012
Isoleucine	0.88	0.89	0.89	0.86	NS	NS	0.012
Leucine	0.93	0.93	0.92	0.89	NS	*	0.010
Phenylalanine	0.93	0.93	0.92	0.89	*	**	0.009
Lysine	0.93	0.94	0.93	0.88	*	**	0.010
Histidine	0.92	0.93	0.92	0.88	*	*	0.010

<sup>a</sup>Comparison of amino acid digestibilities among diets. <sup>b</sup>Test for linear changes in amino acid digestibility with kernel inclusion.

The addition of lupin kernels to sorghum-based diets resulted in a significant linear decrease ( $P < 0.05$ ) in the apparent ileal digestibility of leucine, phenylalanine, lysine and histidine (Table 1). There was no significant effect on the digestibility of threonine, valine or isoleucine, possibly as a result of the small number of animals used in this experiment rather than a difference in the mode of digestion of these amino acids.

As diets were equalised for protein and energy, and lupins were added at the expense of casein and starch, higher levels of NSP from lupin kernels may account for this decrease in digestibility. The losses in pig production which could result from the decrease in dietary amino acid digestibilities need to be quantified as the levels of lupin kernels used in this experiment are representative of those used in commercial pig diets.

### References

- EVANS, A.J., CHEUNG, P.C.K. and CHEETHAM, N.W.H. (1993). *Journal of the Science of Food and Agriculture*. **61**: 189-194.  
 van BARNEVELD, R.J., BATTERHAM, THE LATE E.S. and NORTON, B.W. (1994). *British Journal of Nutrition*. **72**:221-241.

## EFFECT OF LUPIN KERNELS ON THE ILEAL AND FAECAL DIGESTIBILITY OF ENERGY BY PIGS

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Because of the high levels of non-starch polysaccharides (NSP) in lupins, digestible energy (DE) may not be the most appropriate measure of the energy available from lupins for pigs. This is as a result of the large proportion of the carbohydrates in lupins which are digested in the hind-gut. By examining the effects of including graded levels of lupin kernels in the diet on the ileal and faecal digestibility of dry matter (DM) and energy, the effects of NSP in lupins on the energy available to the pig can be quantified.

Four sorghum-based diets were formulated to contain 0, 12, 24 or 36% lupin kernels (*Lupinus angustifolius* cv. gungurru). Dietary crude protein was equalised at 187 g/kg (air-dry basis). Roller-milled lupin kernels were added at the expense of casein and starch, and acid-insoluble ash (added Celite®) was used as a marker. Ileal digestibilities of dry matter and energy were determined using 16 Large White male pigs (40-45 kg body-weight) fitted with simple T-piece ileal cannulas using the technique described by van Barneveld *et al.* (1994). Diet allocations were based on a randomised block design (four pigs/diet). Diets were fed for 7 d prior to 8 h digesta collections over two consecutive days. Faeces sub-samples were collected during the digesta collection period.

**Table 1.** Effect of graded levels of lupin kernels on ileal and faecal apparent digestibilities of the diet for dry matter (DM) and gross energy (GE) (digestibility coefficients), and DE (MJ/kg, air-dry basis) in growing pigs.

Kernel inclusion (%)	0	12	24	36	Diet <sup>a</sup>	Linear <sup>b</sup>	SEM
<u>Ileal digestibility</u>							
DM	0.85	0.82	0.77	0.67	**	***	0.027
GE	0.90	0.87	0.83	0.76	**	***	0.021
DE	16.85	16.47	15.84	14.47	*	**	0.399
<u>Faecal digestibility</u>							
DM	0.90	0.91	0.90	0.90	NS	NS	0.008
GE	0.94	0.94	0.93	0.93	NS	NS	0.006
DE	17.72	17.67	17.64	17.67	NS	NS	0.119

<sup>a</sup>Comparison of amino acid digestibilities among diets. <sup>b</sup>Test for linear changes in amino acid digestibility with kernel inclusion.

The inclusion of lupin kernels at graded levels in diets resulted in a significant ( $P < 0.01$ ) linear decrease in the ileal digestibility of the diets for dry matter, GE and DE (Table 1). However, there were no significant differences in their faecal digestibilities.

It appears that a significant proportion of the NSP in lupins were digested by the hind-gut microflora. This would significantly decrease the efficiency of use of energy derived from lupins by the pig. These results support the findings of Taverner *et al.* (1983) who reported that the net energy content of lupin seed meal was low.

When formulating diets in which lupin kernels are included, a correction factor should be used to compensate for the lower amount of dietary energy available to the pig.

### References

- TAVERNER, M.R., CURIC, D.M. and RAYNER, C.J. (1983). *Journal of the Science of Food and Agriculture*. 34:122-128.  
 van BARNEVELD, R.J., BATTERHAM, THE LATE E.S. and NORTON, B.W. (1994). *British Journal of Nutrition*. 72:221-241.

## DIGESTIBILITY OF NON-STARCH POLYSACCHARIDES BY PIGS FED GRADED LEVELS OF LUPIN KERNELS

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Little work has been done to quantify the anti-nutritional effects of non-starch polysaccharides (NSP) from grain legumes in growing pigs. Research by van Barneveld *et al.* (1993) showed that a commercially available, soluble NSP (guar gum) can significantly depress the ileal digestibility of amino acids in pigs. The anti-nutritive effects of NSP are likely to be influenced by the structure of the polysaccharides, the NSP composition of the other ingredients in the diet, the influence of the NSP on digestive enzymes and digesta viscosity, and the ability of the animal to digest the ingested NSP. The aim of this experiment was to examine the effect of including lupin kernels in the diet on the ileal digestibilities of the constituent sugars of NSP and of starch.

Four sorghum-based diets were formulated to contain 0, 12, 24 or 36% lupin kernels (*Lupinus angustifolius* cv. gungurru). Dietary crude protein was equalised at 187 g/kg (air-dry basis). Roller-milled lupin kernels were added at the expense of casein and starch, and acid-insoluble ash (added Celite®) was used as a marker. Ileal digestibility of dry matter and energy were determined using 16 Large White male pigs (40-45 kg body-weight) fitted with simple T-piece ileal cannulas (van Barneveld *et al.*, 1994). Diet allocations were based on a randomised block design (four pigs/diet). Diets were fed for 7 d prior to 8 h digesta collections over two consecutive days. The total NSP content, NSP-constituent sugars and starch were determined in the diets and digesta using gas chromatography. The values were used in digestibility calculations (Table 1).

**Table 1.** Effect of lupin kernels on the apparent ileal digestibility coefficients for total non-starch polysaccharides (NSP), NSP constituent sugars and starch.

Kernel (%)	0	12	24	36	Diet <sup>a</sup>	Linear <sup>b</sup>	Quadratic <sup>c</sup>	SEM
Arabinose	0.31	0.43	0.41	0.30	NS	NS	NS	0.065
Xylose	0.09	0.29	0.26	0.11	NS	NS	NS	0.082
Galactose	0.00	0.48	0.35	0.09	**	NS	**	0.091
Total NSP	0.11	0.37	0.26	0.05	NS	NS	*	0.090
Starch	0.99	0.98	0.98	0.96	NS	*	NS	0.007

<sup>a</sup>Comparison of amino acid digestibilities among diets. <sup>b</sup><sup>c</sup>Test for linear or quadratic changes in amino acid digestibility with kernel inclusion.

The addition of graded levels of lupin kernels had no significant effect ( $P > 0.05$ ) on the digestibility of arabinose or xylose, but resulted in a significant ( $P < 0.05$ ) quadratic change in the apparent ileal digestibility of galactose and total NSP. Inclusion of lupin kernels resulted in a significant linear decrease ( $P < 0.05$ ) in dietary starch digestibility.

It appears that low levels of dietary NSP coincided with poor NSP digestion in the small intestine (SI). Small increases in dietary NSP resulted in an increase in the ability of the animal to digest some of the constituent sugars, and hence total NSP, possibly as a result of an increase in the activity of the microbial flora in the SI. However, as the dietary level of lupin kernels was increased, the extent of NSP digestion in the SI decreased. This may have coincided with elevated gut viscosity resulting in an inhibition of digestion and absorption of nutrients including the NSP. The results suggest that there is a digestive response to the presence of lupin kernel NSP, but the capacity of the pig to digest this NSP in the SI is rapidly exceeded as the level of lupin kernels increases.

### References

- van BARNEVELD, R.J., ANNISON, G., BAKER, J. and SZARVAS, S.R. (1993). *Proceedings of the Nutrition Society of Australia*. 18:59.  
 van BARNEVELD, R.J., BATTERHAM, THE LATE E.S. and NORTON, B.W. (1994). *British Journal of Nutrition*. 72:221-241.

## DIGESTIBILITY OF AMINO ACIDS AND ENERGY IN NAKED OATS (*AVENA SATIVA* CV. BANDICOOT) FED TO GROWING PIGS

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Dehulled oats (groats) are an excellent energy source for pigs but the added cost of dehulling restricts their use to situations where the price of whole oats is low. Naked oats have been bred so that the lemma and palea of the seed thresh free at harvest and hence require no further processing (apart from coarse crushing) prior to inclusion in pig diets. Hence, naked oats have considerable potential as a dietary ingredient. The aim of the present experiment was to determine the ileal digestibilities of amino acids and digestible energy (DE) in two samples of naked oats (*Avena sativa* cv. Bandicoot) and to compare them with the corresponding digestibilities in wheat and groats.

The test cereals were the sole sources of protein in the experimental diets, and Celite® was added as an acid-insoluble ash marker. Ileal amino acid digestibility and DE were determined using four Large White male pigs (40-45 kg body-weight) fitted with simple T-piece ileal cannulas using the technique described by van Barneveld *et al.* (1994). Diet allocations were based on a 4 × 4 latin square design. Diets were fed for 7 d prior to 8 h digesta collections over two consecutive days. Faeces sub-samples were collected during the digesta collection period.

Table 1. Ileal digestibility coefficients for some essential amino acids and DE (MJ/kg; air-dry basis) in wheat, groats and naked oats fed to growing pigs.

	Cereal				Significance	
	Wheat	Groats	Naked oat 1	Naked oat 2	SEM	Diet
Threonine	0.77	0.76	0.77	0.78	0.018	NS
Valine	0.83	0.83	0.85	0.85	0.013	NS
Isoleucine	0.80	0.82	0.82	0.84	0.014	NS
Leucine	0.86	0.85	0.85	0.85	0.012	NS
Phenylalanine	0.86	0.86	0.87	0.87	0.012	NS
Lysine	0.87 <sup>a</sup>	0.92 <sup>bc</sup>	0.89 <sup>ac</sup>	0.82 <sup>d</sup>	0.008	**
Histidine	0.84	0.83	0.84	0.84	0.015	NS
DE	14.61 <sup>a</sup>	16.80 <sup>b</sup>	17.17 <sup>c</sup>	16.74 <sup>b</sup>	0.105	***

<sup>abc</sup>Values in the same row with different superscripts differ significantly ( $P < 0.05$ ).

The ileal digestibility of lysine in groats was significantly higher ( $P < 0.01$ ) than in either wheat or naked oats (Table 1). The DE values for groats and naked oats were higher ( $P < 0.001$ ) than that for wheat and agree with those reported by Barr and Teague, (1994).

Bandicoot naked oats and groats have higher total levels of most amino acids than wheat, and are superior as sources of amino acids, despite having similar ileal amino acid digestibility coefficients. As naked oats require no further processing they represent a more convenient, and possibly more cost-effective, dietary ingredient than dehulled oats because naked oats have a similar "groat yield" to conventional oats. A major limitation of groats is the rapid decline in quality when they are stored over extended periods. Further research is required to determine the storage life of naked oats relative to groats.

### References

- BARR, A.R. and TEAGUE, T.J. (1994). "Bandicoot Naked Oats - An End User Guide". (Grains Research and Development Corporation: Canberra).
- van BARNEVELD, R.J., BATTERHAM, THE LATE E.S. and NORTON, B.W. (1994). *British Journal of Nutrition*. 72:221-241.

## FACTORS AFFECTING THE DETERMINATION OF DIGESTIBLE ENERGY IN *LUPINUS ANGUSTIFOLIUS* CV. GUNGURRU FOR GROWING PIGS

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There is considerable variation in the estimates of digestible energy (DE) for *Lupinus angustifolius* (13.1 to 15.3 MJ/kg air-dry, SCA (1987)). This may reflect differences among samples and/or techniques used to assess digestibility. The latter could be influenced by dietary energy source or by the degree to which the lupins were ground. The DE of lupins is normally assessed using a diet of lupins in combination with a cereal of known DE, determining the DE of that diet and then calculating the DE of the lupins by difference. An alternative to this is to use lupins in a sugar-based diet and assume 100% digestibility of the sugar component when calculating the DE of the lupins.

Estimates for DE have been made for coarsely crushed lupins in a sugar-based diet (Wigan *et al.*, 1993) and for finely crushed lupins in both wheat- and sugar-based diets. The lupins were crushed by hammer-milling through a 5 mm (course) or 3 mm (fine) screen. Each diet contained 50% lupins (air-dry) and ferric oxide (as an indigestible marker). Male pigs of approximately 30 kg live-weight were housed in metabolism cages and there were six pigs per treatment. Digestible energy was determined using a 7-day total faecal collection (Table 1). A wheat diet was included in each experiment as a positive control, allowing assessment of the effect of fineness of grinding.

**Table 1. Digestible energy (DE) and gross energy digestibility coefficients (GED) of fine and coarsely crushed lupins determined in wheat- or sugar-based diets (mean  $\pm$  SE).**

	Finely crushed		Coarsely crushed
	Lupin (wheat)	Lupin (sugar)	Lupin (sugar)
DE (MJ/kg air-dry)	14.4 $\pm$ 0.29	13.2 $\pm$ 0.29	12.3 $\pm$ 0.32
GED	0.79 $\pm$ 0.019	0.73 $\pm$ 0.019	0.68 $\pm$ 0.018

Energy source influenced both GED and DE. Wheat-based diets led to higher ( $P < 0.05$ ) values for both measurements than sugar-based diets (Table 1). The DE of coarsely crushed lupins (12.3 MJ/kg) was not significantly different ( $P > 0.05$ ) to the finely ground sample (13.2 MJ/kg), however this may have important commercial implications.

The higher GED of lupins in the wheat-based diet may be a result of increased hindgut fermentation because of the greater quantity of complex carbohydrates passing to the large intestine of these pigs. The trend towards a higher DE value with fine grinding is thought to be as the result of increased susceptibility of the lupin particles to digestive secretions and hindgut fermentation.

These results indicate that when determining DE values in lupin-seed meal for use by industry, finely ground meal should be used in cereal-based diets. In addition, lupins may have a greater feeding value for pigs when finely ground.

### References

- SCA (1987). "Feeding Standards for Australian Livestock". (CSIRO: Australia)  
 WIGAN, G.C., BATTERHAM, E.S. and FARRELL, D.J. (1993). In "Recent Advances in Animal Nutrition in Australia, 1993", p. 2B, ed. D.J. Farrell. (University of New England: Armidale).



## LONG-CHAIN HYDROCARBONS AS A MARKER FOR DIGESTIBILITY STUDIES IN MONOGASTRIC SPECIES

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The digestibility of nutrients can be accurately estimated by including a marker in a test diet. The most frequently used substances as markers are chromium oxide and acid-insoluble ash (AIA). Although they are well established markers, their determination is laborious and time-consuming. It is also worth noting that chromium oxide in digesta and faeces is regarded as chemical waste, and the treating of acid-insoluble ash with boiling HCl is considered hazardous. Other shortcomings of these markers are the relatively high inclusion levels in the diet and the requirements for large samples for analysis.

Hydrocarbons are naturally present in plants and they are not utilised or metabolised in monogastric species (Dove and Mayes, 1991). Long, even chain hydrocarbons such as hexatriacontane ( $C_{36}H_{74}$ ) and tetratriacontane ( $C_{34}H_{70}$ ) are readily available commercially and can be used as a digestibility marker in pig and poultry diets. As these compounds are soluble in warm oil, they can be easily mixed into the diet and an inclusion of 100-200 mg/kg is adequate for a precise measurement of the marker. An experiment was conducted to ascertain the validity of the hydrocarbons as a digestibility marker. Hexatriacontane was added to pig diets at 200 mg/kg, which also contained 20 g/kg AIA (Celite). There were four different diets containing lupins (*L. angustifolius*) at 0, 12, 24, or 36%, respectively. Each diet was fed to four pigs.

A total of 16 ileal digesta samples and 16 faecal samples were collected and analysed for AIA and the hydrocarbon ( $C_{36}H_{74}$ ) (Figure 1). The hydrocarbon was determined after extraction with hexane followed by quantification using a GC. A near perfect correlation between the established marker, acid-insoluble ash, and the new hydrocarbon marker was found in ileal samples ( $n=16$ ;  $r^2=0.972$ ), faecal samples ( $n=16$ ;  $r^2=0.951$ ) or in both ileal and faecal samples pooled together ( $n=32$ ;  $r^2=0.978$ ).

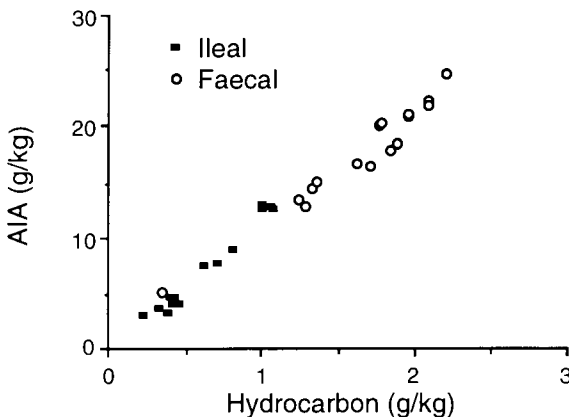


Figure 1. Correlation between acid-insoluble ash (AIA) and the hydrocarbon ( $C_{36}H_{74}$ ) marker in either ileal or faecal samples from pigs.

The long chain hydrocarbons offer an attractive alternative to AIA and chromium oxide as a digestibility marker. Hydrocarbons are easy to measure and require only small samples of digesta for accurate determination with a gas chromatograph.

### References

DOVE, H. and MAYES, R.W. (1991). *Australian Journal of Agricultural Research*. **42**:913-952.

## THE RAT AS A MODEL FOR THE PIG FOR DETERMINING TRUE ILEAL REACTIVE LYSDNE DIGESTIBILITY.

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A new method for determining the true ileal digestibility of reactive lysine in heated protein sources based on an *in vivo* pig assay has recently been developed by Rutherford and Moughan (1995). However, pig ileal digestibility assays are labour intensive and costly. The use of the laboratory rat as a model animal may allow a less costly and more rapid assay to be developed.

True ileal reactive lysine digestibility and the conventional true ileal digestibilities of other acid-stable amino acids were determined in a heated lactose/casein mixture using the rat and pig. For true ileal reactive lysine digestibility, reactive lysine was determined in the diet and digesta (slaughter method) of 25 kg body-weight Landrace  $\times$  Large White entire male pigs and 150 g body-weight Sprague Dawley male rats using guanidination. For the remaining true ileal amino acid digestibilities, amino acids were determined using conventional amino acid analysis. The enzymatically hydrolysed casein/ultrafiltration method was used to determine endogenous amino acid flows at the terminal ileum (Moughan *et al.*, 1990). True ileal amino acid digestibilities were calculated by correcting apparent coefficients for endogenous loss. Chromic oxide was included as an indigestible marker.

True ileal reactive lysine digestibility was not significantly different when determined in the rat and the pig (Table 1). Further, the true ileal digestibilities of most of the remaining amino acids were also not significantly different between the two species. There was a significant 4% difference in alanine digestibility between species.

**Table 1. Mean true ileal amino acid digestibility (%) in a heated lactose/casein mixture, for the rat (n=8) and pig (n=8).**

	Pig	Rat	Overall SE	Significance <sup>1</sup>
Aspartic acid	96.6	95.0	0.89	NS
Threonine	95.6	94.0	1.10	NS
Serine	89.9	92.5	1.99	NS
Glutamic acid	95.6	95.7	0.85	NS
Glycine	78.5	74.4	5.32	NS
Valine	96.4	96.9	0.80	NS
Alanine	95.1	99.2	1.07	*
Isoleucine	94.7	95.5	1.08	NS
Leucine	98.8	99.6	0.38	NS
Tyrosine	99.3	99.3	0.36	NS
Phenylalanine	98.9	99.4	0.32	NS
Histidine	93.8	94.6	0.70	NS
Arginine	95.0	94.3	2.10	NS
Reactive lysine	98.2	98.0	0.47	NS

<sup>1</sup>NS, non significant,  $P > 0.05$ ; \*  $P < 0.05$ .

The laboratory rat may be a suitable model for the pig for determining true ileal reactive lysine digestibility, and true ileal amino acid digestibility in heated protein sources. The rat may offer a cheaper and more routine approach to dietary protein quality assessment for the pig.

### References

- MOUGHAN, P.J., DARRAGH, A.J., SMITH, W.C., and BUTTS, C.A. (1990). *Journal of the Science of Food and Agriculture*. 52: 13-21.
- RUTHERFURD, S.M. and MOUGHAN, P.J. (1995). In: "Manipulating Pig Production V", p. 25, eds D.P. Hennessy and P.D. Cranwell. (Australasian Pig Science Association: Werribee).

## CARBON DIOXIDE IS NOT AN ALTERNATIVE TO HALOTHANE ANAESTHESIA IN NITROGEN DIGESTIBILITY STUDIES

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A technique involving collection of ileal digesta under halothane anaesthesia has been described as an alternative to the simple T-piece cannulation technique for use in digestibility experiments (Moughan and Smith, 1987). A disadvantage of the anaesthetic approach is that the carcass becomes unsuitable for human consumption. A possible alternative is to use CO<sub>2</sub> stunning and commercial slaughter procedures combined with careful removal of the ileal digesta during evisceration. The aim of this experiment was to measure the apparent ileal digestibility of nitrogen (AIDN) with digesta collected using either the CO<sub>2</sub> stunning technique or the halothane anaesthetic technique for grower pigs fed either cottonseed meal (CSM) or soya bean meal (SBM).

Twenty-four male pigs (37.3 ± 2.7 kg) were individually housed and randomised to either CSM or SBM diets. Diets contained 40% of the meals in a wheat starch:sugar (1:1) base containing vitamins and minerals, and Cr<sub>2</sub>O<sub>3</sub> as an indigestible marker. Rations were offered (1800 g/pig/d) in 3 meals/d on d 4 to 11 and 8 meals/d on day 12 to 13. On day 14, the pigs were fed hourly for 8 h, after the 8th meal half of the pigs were anaesthetised with halothane and digesta were sampled from a 150 cm portion of the terminal ileum before a lethal injection of barbiturate. The remaining pigs were stunned with CO<sub>2</sub> and processed, with the ileal digesta being carefully collected during evisceration. Faeces were collected directly from the rectum. Results are given in Table 1.

**Table 1. Effect of collection technique and site of digesta collection on apparent nitrogen (N) and organic matter (OM) digestibilities in soya bean meal (SBM) and cottonseed meal (CSM) diets.**

Digestibility (%)		CO <sub>2</sub>		Halothane		SED <sup>1</sup>	Significance <sup>2</sup>
		Ileal	Faecal	Ileal	Faecal		
N	SBM	54.9	81.1	70.5	80.4	5.37	D***, M*, S**
	CSM	51.0	56.4	64.5	56.1		
OM	SBM	72.0	92.7	77.2	91.5	4.07	D***, S***
	CSM	69.7	74.3	71.8	73.8		

<sup>1</sup>Standard error of the difference for diet × method × site. <sup>2</sup>Levels of significance, \*P<0.05; \*\*P<0.01; \*\*\*P<0.001; where D = diet, M = method, S = site.

Ileal digestibility of N was lower when digesta were sampled using the CO<sub>2</sub> technique than when obtained under halothane anaesthesia. This is most likely as a result of the sloughing of intestinal cells after CO<sub>2</sub> stunning and processing. Thus, the use of the CO<sub>2</sub> technique is not recommended for AIDN studies. From results obtained using halothane anaesthesia it is evident that both ileal and faecal N and OM digestibility were lower in CSM than SBM diets. For pigs fed SBM there were further increases in digestibility of both N and OM in the hindgut whereas for the CSM there were no further changes in digestibility in the hindgut. Therefore, much of the N and OM in CSM is apparently indigestible in both the fore and hindgut and unavailable for use. These data are consistent with the lower growth rates of pigs fed the CSM diet (508 vs 708 g/d; P<0.001).

### References

MOUGHAN, P.J. and SMITH, W.C. (1987). *Animal Production*. 4:319-321.

## EFFECT OF CONDENSED TANNIN IN COTTONSEED HULLS ON TRUE ILEAL AMINO ACID DIGESTIBILITY IN CASEIN

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Condensed tannin (CT) is present in commercially produced cottonseed meal (CSM) in significant concentrations. Condensed tannin is comprised of polyphenolic compounds which are capable of precipitating proteins in aqueous solutions and have been shown to have anti-nutritional effects in monogastric animals (Huisman *et al.*, 1990). The objective of this study was to determine the effect of cottonseed CT on the true ileal amino acid (AA) digestibility of casein.

Twenty-four Sprague-Dawley rats ( $176 \pm 4.5$  g body-weight) were randomly allocated to four semi-synthetic diets, such that there were three males and three females per diet. The diets contained casein as the major protein source and  $\text{Cr}_2\text{O}_3$  as an indigestible marker. Two of the casein diets did not contain hulls whilst the remaining two diets each contained 70 g cottonseed hulls/kg. The cottonseed hulls contained 51 g CT/kg dry matter. For each pair of diets, polyethylene glycol (PEG) was either included (2 mg/mg total CT) or excluded. Polyethylene glycol was used to bind dietary CT and to displace protein from the CT-protein complexes (Jones and Mangan, 1977). The effect of CT was quantified by comparing control rats (-PEG) with PEG supplemented rats (+PEG) at each level of dietary hulls. Endogenous AA flows were obtained in a separate but related study (Yu *et al.*, 1995) for rats given diets containing graded levels of cottonseed hulls.

**Table 1. Mean true ileal amino acid digestibility (%) for rats fed a casein-based diet.**

Cottonseed hulls Polyethylene glycol (PEG)	0%		7%		Overall SE	Significance <sup>1</sup>		
	-	+	-	+		A	B	C
Arginine	98	98	95	97	0.45	NS	***	**
Histidine	98	98	93	97	0.49	NS	***	***
Isoleucine	93	93	88	91	0.81	NS	***	**
Leucine	98	98	95	97	0.33	NS	***	**
Lysine	98	99	95	98	0.35	NS	***	***
Phenylalanine	98	99	96	98	0.30	NS	***	**
Threonine	91	93	89	92	0.84	NS	*	*
Valine	95	96	91	93	0.72	NS	**	*

<sup>1</sup>A: PEG+ vs PEG-, 0% hull; B: 0% vs 7% hull without PEG; C: PEG+ vs PEG-, 7% hull; NS, non significant,  $P > 0.05$ ; \* $P < 0.05$ ; \*\* $P < 0.01$ , \*\*\* $P < 0.001$ .

There was no significant difference in true ileal AA digestibility between the hull-free diets in the presence and absence of PEG (Table 1), indicating that there was no effect of PEG *per se* on dietary AA digestibility in the absence of CT. Inclusion of 70 g/kg cottonseed hulls in the diet significantly depressed the true ileal digestibility of all AA by 3-6 percentage units. When PEG was added to the diet containing hulls, the true ileal digestibility for all AA was restored significantly, to levels similar to those found in the absence of hulls.

The addition of cottonseed hulls to the casein based diet reduced the true ileal digestibility of amino acids because of an effect of the CT present in the hulls.

### References

- HUISMAN, J., van der POEL, A.F.B., VERSTEGEN, M.W.A. and van WEERDEN, E.J. (1990). *World Review of Animal Production*. 25:77-82.  
 JONES, W.T. and MANGAN, J.L. (1977). *Journal of the Science of Food and Agriculture*. 28:126-136.  
 YU, F., MOUGHAN, P.J. and BARRY, T.N. (1995). *Journal of the Science of Food and Agriculture*. 68: (In press).

## EFFECT OF ENERGY SOURCE AND FEEDING/COLLECTION TIME ON ILEAL CONTENTS OBTAINED FROM WEANER PIGS

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Previous studies with weaner pigs offered sugar-based diets have shown that ileal collections made using the dissection technique are often too small to allow accurate determination of nutrient digestibility. The small ileal collections were obtained despite the fact that the piglets were offered dry mash diets at 90% of *ad libitum* energy intake and had 24 h access to their food. The aims of this experiment were to determine the effect on ileal sample dry weight and nitrogen digestibility of 1) adding starch to a sugar-based diet, and 2) feeding/ collection time.

The experiment was designed as a 2 × 3 factorial with 2 diets each containing 315 g/kg soya bean meal and either 591 g/kg sucrose or 295.5 g/kg of both sucrose and wheat starch. Both diets contained chromic oxide as a marker. There were 3 feeding/ collection times (1500/0800 h, 0800/1000 h and 0800/1330 h). The experimental diets were fed as a dry mash to six individually housed pigs. Water was provided *ad libitum* from nipple drinkers. Each pig was offered the experimental diet at 3 × maintenance digestible energy intake and was given 24 h to consume the ration. Feeding continued for 7 d after each pig reached 16 kg live-weight; it was then sedated with an intra-muscular injection of ketamine/xylazine (16 mg and 0.8 mg/kg live-weight respectively) and anaesthetised with a gas mixture of oxygen and halothane. The contents of the ileum (0 to 150 cm anterior to the ileo-caecal junction) were then removed, prior to the pig being killed with an overdose of sodium pentobarbitone. Ileal collections were immediately frozen (-15°C), freeze dried, ground and then analysed for nitrogen and chromium (Table 1).

Table 1. Effect of energy source and feeding/collection time on mean (± SEM) ileal sample dry matter (DM) and nitrogen digestibility (N dig) of weaner pigs (18 kg live-weight).

	n <sup>1</sup>	Energy source		Feeding/collection times (h)		
		Sucrose	Sucrose/starch	1500/0800	0800/1000	0800/1330
DM (g)	36	5.4 ± 0.61	5.5 ± 0.59	4.6 ± 0.72	5.5 ± 0.72	6.3 ± 0.75
N dig (%)	30	69 ± 2.6	70 ± 2.6	73 ± 2.8	69 ± 3.3	65 ± 3.3

<sup>1</sup>n = number of samples included in analyses.

Six samples were discarded from the 0800/1000 h and 0800/1330 h feeding/ collection treatments either because of low nitrogen digestibility (less than 20%) or because no sample was obtained when the collection was made.

The addition of 30% wheat starch to a sucrose diet did not increase the sample DM nor did it alter the nitrogen digestibility of soya bean meal fed to weaner pigs. The 1500/0800 h feeding/ collection treatment appeared to maintain ileal flow and provided uniform nitrogen digestibility when weaner pigs were offered a sugar-based diet as a dry mash.

## DETERMINATION OF APPARENT ILEAL PROTEIN AND AMINO ACID DIGESTIBILITY IN FEED FOR PIGS USING THE "MOBILE NYLON BAG TECHNIQUE"

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It is generally agreed that the ileal rather than the traditional faecal analysis method should be used for determining amino acid digestibility in pig diets. However, conventional digestibility methods that require either total collection of ileal digesta or the use of a marker are expensive and time consuming. A more practical method may be provided by the "mobile nylon bag technique" (MNBT), originally developed by Sauer *et al.* (1983) to determine faecal protein digestibility. The objective of these studies was to evaluate the use of the MNBT for determination and prediction of apparent ileal protein and amino acid digestibility in a variety of feeds for pigs.

For the MNBT studies, four barrows were surgically fitted with a simple T-piece cannula in the duodenum and with a post valve T-caecum (PVTC) cannula, distal to the ileocaecal valve (van Leeuwen *et al.*, 1991). One-gram samples of each of 24 different feeds, ground through a 1.0 mm mesh screen, were enclosed in nylon bags (25 × 40 mm; 48 mm mesh). Following pre-digestion (4 h; 37°C; 0.01 M HCl; 4,000 Folin Intestinal Pepsin Units/L) the bags were inserted via the duodenal cannula and subsequently collected from the PVTC cannula. The undigested contents were pooled within pig and feedstuff prior to analyses. The apparent ileal protein and amino acid digestibilities of each feedstuff were also determined in conventional digestibility trials using barrows fitted with simple T-piece cannulae at the distal ileum.

**Table 1. Apparent ileal protein and lysine digestibility of feed determined with the conventional method (CM) and the mobile nylon bag technique (MNBT)<sup>1</sup>.**

	Protein		Lysine	
	CM	MNBT	CM	MNBT
Cereals (n=11)	70.4 ± 9.1	76.1 ± 7.6***	61.1 ± 9.1	81.7 ± 5.3***
Peas (n=4)	74.2 ± 2.3	80.5 ± 5.2*	82.7 ± 1.6	91.5 ± 1.2***
Oilseed products (n=4)	71.4 ± 3.7	50.2 ± 13.0	77.3 ± 3.3	70.5 ± 7.3

<sup>1</sup> Data presented as mean ± SD. Means in the same row, within protein and lysine digestibility, differ at \* P<0.05, \*\*\* P<0.001.

With the exception of products from oilseeds, the apparent ileal protein and amino acid digestibility, determined with the MNBT, were significantly (P<0.001; P<0.05) higher than those determined with the conventional method. Simple regression analysis showed a correlation of  $r=0.24$  (P>0.05) between both methods for apparent ileal protein digestibility. An improvement (P<0.05) in the correlation coefficient,  $r=0.69$ , was obtained when the protein content of the feed was included as the second covariable in multiple regression analysis. In conclusion, the suitability of the MNBT for determining or predicting *in vivo* apparent ileal protein and amino acid digestibility is limited.

### References

- SAUER, W.C., JORGENSEN, H. and BERZINS, R. (1983). *Canadian Journal of Animal Science*. 63:233-237.  
 van LEEUWEN, P., van KLEEF, D.J., van KEMPEN, G.J.M., HUISMAN, J. and VERSTEGEN, M.W.A. (1991). *Journal of Animal Physiology and Animal Nutrition*. 65:183-193.

## AN *IN VITRO* METHOD FOR PREDICTING DIGESTIBLE ENERGY APPLIED TO MILLING BY-PRODUCTS

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The wheat milling by-products, bran and pollard, are widely used as feed ingredients in the pig industry. Because there is variation in the digestible energy content of these products (from 10.9 to 14.1 MJDE/kg, Batterham *et al.*, 1980), and since *in vivo* methods are time consuming and expensive, there is a need for a fast and reliable *in vitro* method to estimate energy digestibility. The *in vivo* apparent digestible energy (ADE) and the *in vitro* digestibility of dry matter (DDM) in 5 samples of both pollard and bran were determined. The relationship between the *in vivo* and *in vitro* results was investigated.

*In vivo* digestibility was based on the total faecal collection method (den Hartog *et al.*, 1988). For each by-product six 35 kg body-weight (BW) Large White  $\times$  Landrace male pigs were included in a 6  $\times$  6 randomised Latin Square Design. A basal diet (80% barley, 10% Chilean fish meal, 10% casein, plus a vitamin and mineral supplement) and the five samples each combined with the basal diet (70%/30%) were fed (8% BW<sup>0.75</sup>) to each pig for a 10 d period. *In vivo* digestibility was determined by correcting the determined digestibility of the experimental diet for the contribution of the basal diet.

The *in vitro* DDM was estimated using a three-step enzymatic method (Boisen, 1991). The samples (0.5 g of finely ground material) were incubated at 40°C with pepsin at pH 2 for 75 min, then with pancreatin at pH 6.8 for 3.5 h, and finally with Viscozyme (Novo, Denmark) at pH 4.8 for 18 h. After incubation the residue was filtered to remove the digested material and the residue was dried overnight at 80°C. A blank containing enzyme only was included. *In vitro* DDM was calculated from the dry matter in the sample and the undigested residue, after correction for the dry matter in the blank.

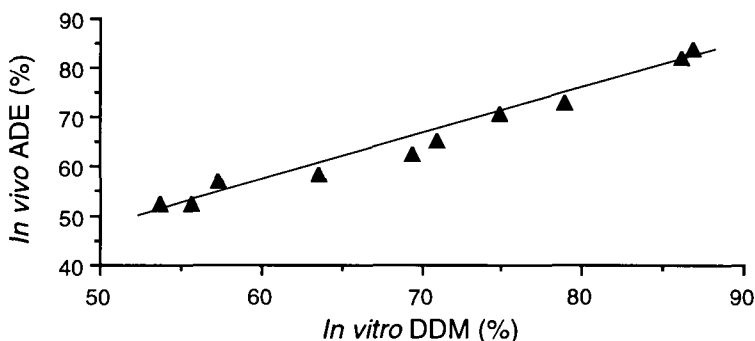


Figure 1. Relationship between *in vivo* apparent digestibility of energy and *in vitro* dry matter digestibility.

As shown in Figure 1, the *in vitro* DDM by the three-step method was significantly related to the *in vivo* ADE. The prediction equation generated from regression analysis was:  $ADE (\%) = 1.42 + 0.93 DDM (\%)$  ( $r^2=0.97$ ,  $rsd=2.05$ ). It is concluded that the *in vitro* value of DDM is a good predictor of *in vivo* ADE for milling by-products.

### References

- BATTERHAM, E.S., LEWIS, C.E., LOWE, R.F. and McMILLAN, C.J. (1980). *Animal Production*. 31:259-271.  
 BOISEN, S. (1991). In "In vitro Digestion for Pigs and Poultry", pp. 135-145, ed. M.F. Fuller. (CABI: Wallingford).  
 den HARTOG, L.A., VERSTEGEN, M.A.W., BOER, H. and LINDERS, P.B.J. (1988). *Journal of Animal Science*. 65 (Supplement 1):311.

## A SYMPOSIUM - NOVEL METHODS TO ENHANCE GROWTH

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### Introduction

Two factors that have had a significant influence on pig production, particularly over the last 10-20 years, are, first, the necessity to continually improve the efficiency of production and, second, the need to respond to the continual pressures of consumer demand. The drive to improve production efficiency is based on declining returns for pig meat and pig meat products and the increase in the size of production units with a reduced number of staff per sow (Campbell, 1995). The strength of consumer demand is best seen by the marked decrease in the backfat of pigs over the last 10-20 years (of at least 0.5 mm per year) in response to the demand for leaner meat. In the past, the efficiency of pig production has been increased by improving the match between the supply of nutrients in the diet with the pigs requirements, by improving climatic control in pig housing, by using improved genotypes that produce more lean meat per unit of feed intake, and managing pigs to improve herd health. These management strategies are still important factors in modern pig production but, as described by Campbell (1995), there has been no reduction in the average cost of production in the Australian pig industry over the last five years. This suggests that the pig industry may need to develop and adopt new methods to improve the efficiency of growth in pigs.

As our understanding of pig nutrition, metabolism and physiology has improved, opportunities have arisen for using novel methods to improve growth performance. The term 'novel' is used in this context to describe technologies that aim to manipulate specific metabolic processes by either increasing the potential growth rate or by removing specific limitations to productivity. The first two papers in this symposium (Dunshea and Walton, 1995; McCauley *et al.*, 1995) will address the scientific basis behind two strategies to improve growth that have been the focus of recent research. First, the use of exogenous metabolic modifiers and, second, the manipulation of endogenous hormones. These papers do not provide an exhaustive examination of novel growth enhancers, but they clearly demonstrate some recent advances in the manipulation of pig growth.

A discussion within the context of the entire pig industry on the potential of novel methods to enhance growth would be incomplete without considering the factors that contribute to the ultimate adoption, or rejection, of growth promoters by pig producers. The translation of novel laboratory techniques into methods that are adopted and integrated into production systems can be a difficult route with several hurdles to be cleared. The hurdles include regulatory approval by legislature, the scientific criteria of safety, quality and efficacy, and the more subjective political considerations of ethics, animal welfare and socio-economic impacts. The final paper in this symposium (Bent, 1995a) will address these issues.

The scientific research that aims to improve our understanding of the physiological mechanisms by which a novel method enhances growth may seem a long way from the market place for pigmeat. It is, however, part of the long process of converting an original idea into a technique that is adopted by the industry. For example, only rigorous scientific research allows us to determine the most appropriate methods of adopting the new technology, the likely impact on the costs of production, and the consequences on the quality of the final product. Coupling this work with a socio-economic evaluation is often neglected, perhaps because the two areas of research, i.e., scientific vs socio-economic, are generally carried out by different groups of people, i.e., physiologists vs economists. This, however, does not lessen the importance of combining scientific studies with an evaluation of the likely impact of new technologies on the industry as a whole.

*Symposium continued on next page*



## POTENTIAL OF EXOGENOUS METABOLIC MODIFIERS FOR THE PIG INDUSTRY

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### Introduction

Genetic selection and increased understanding of nutrition over the last decade or so has led to tremendous improvements in the efficiency of pig production. A characteristic of the improved pig is that it has high circulating levels of the naturally occurring hormone, somatotropin (or growth hormone) (Wangsness *et al.* 1977). It has been known for 40 years that injection of pigs with pituitary tissue extracts containing porcine somatotropin (pST) results in increased lean tissue deposition and decreased fat accretion in growing pigs (Turman and Andrews, 1955). Advances in biotechnology have now provided a means of producing pST on a commercial scale and the efficacy of daily injection of recombinantly-derived pST for improving productive performance of swine is beyond doubt. Recently pST was approved for use in the Australian pig industry as a daily injectable metabolic modifier. The effects of pST on muscle and bone deposition are thought to be mediated via insulin-like growth factor-I (IGF-I). This peptide hormone and its analogues, which can also be produced using recombinant technology, are being investigated as growth promoters for the pig industry.

In addition to the injectable somatotropins and IGF's, other orally active compounds which alter the ratio of lean to fat deposition have recently become available for research and, in some cases, commercial purposes. The first group of compounds are broadly called  $\beta$ -agonists because they all act through the  $\beta$ -receptors on the target tissues. These  $\beta$ -agonists which act predominantly on skeletal muscle to increase protein deposition and have little effect on bone or fat deposition, are currently being reviewed by authorities overseas for use in the animal industries. One orally active metabolic modifier which is approved for use in the pig and poultry industries is betaine. Betaine has been shown under some circumstances to decrease backfat depth and improve growth performance in pigs. Another feed additive, chromium picolinate, has also been suggested as a possible metabolic modifier for pigs. In this paper the role of metabolic modifiers in the grower/finisher pig and the factors affecting their efficacy will be examined. The possibility of manipulating the growth of the neonatal piglet, whose performance is far below potential, will also be discussed.

### Somatotropin

Exogenous pST treatment consistently improves average daily gain (ADG) and feed conversion efficiency (FCE) and its efficacy is unquestioned (Etherton *et al.*, 1987; Campbell *et al.*, 1988, 1989a, 1990ab, 1991). Dose-dependent increases in lean deposition and reductions in fat deposition and carcass fat have been observed (Boyd and Bauman, 1989; Etherton *et al.*, 1987; Evock *et al.*, 1988; Krick *et al.*, 1992). Porcine somatotropin is effective in increasing protein deposition and decreasing fat deposition in boars, gilts and barrows (Figure 1, Campbell *et al.*, 1989a) of both poor and improved genotypes (Campbell *et al.*, 1990a, 1991; Krick *et al.*, 1992). Although the greatest responses occur in finisher pigs (60 to 100 kg), exogenous pST also improves performance in younger pigs (30-60 kg) (Campbell *et al.*, 1989b, 1990b; Krick *et al.*, 1993). As a result of the reduction in fat deposition, there is a consistent dose-dependent reduction in feed intake (Boyd and Bauman, 1989; Krick *et al.*, 1992). While this reduction in feed intake is sometimes associated with improved digestibility (Wray-Cahen *et al.*, 1991), these differences disappear when pigs are pair-fed (Verstegen *et al.*, 1990). Since pST stimulates protein deposition in all tissues, there are dose-dependent increases in visceral mass and reductions in dressing percentage (Etherton *et al.*, 1987; Evock *et al.*, 1988). The effects of metabolic modifiers such as pST and the  $\beta$ -agonists have been reviewed recently (Reeds and Mersmann, 1991; Dunshea, 1994; NRC, 1994) and will be briefly discussed.

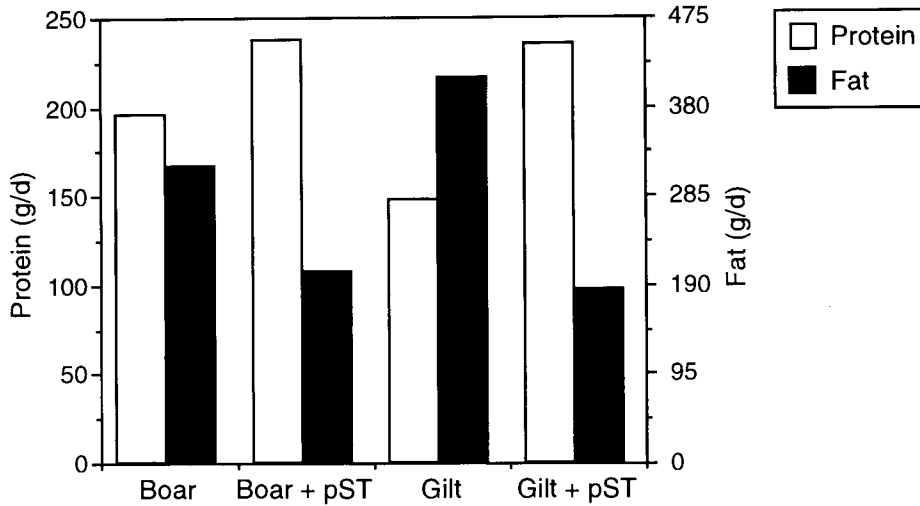


Figure 1. Effect of pST on protein and fat deposition in finisher gilts and boars (Campbell *et al.*, 1989a).

Given that there is relatively little effect of pST upon protein digestibility, the increased protein deposition observed in pST treated pigs must be due to either an increase in the efficiency of use of dietary protein and/or an increase in the requirement of dietary protein to support the increased protein deposition. In the grower pig (30-60 kg) it appears that pST has very little, if any, effect on dietary protein requirements but there is an increase in the efficiency of utilization of dietary protein (Campbell *et al.*, 1990b; Caperna *et al.*, 1990; Krick *et al.*, 1993). For example, both Campbell *et al.* (1990b) and Krick *et al.* (1993) conducted experiments where grower pigs were fed diets containing varying levels of "ideal" protein and were also treated with excipient or pST. These data suggest only a marginal increase in the dietary protein requirement, but a substantial increase in the efficiency of use of dietary protein of approximately 25%. Therefore, it appears that conventional starter/grower diets containing 17-18% crude protein may be sufficient to allow the expression of the benefits on pST in grower pigs over the live-weight range of 25-60 kg. In this class of pigs, energy intake is more likely to be limiting than protein intake.

The effects of pST on the protein requirements of finisher pigs (60-90 kg), in which increases in protein deposition are greater, are less clear. In one study, both the efficiency of use of dietary lysine (formulated to be the first limiting amino acid) and the lysine requirement were increased in gilts and barrows treated with pST (Boyd *et al.*, 1991). In this experiment, efficiency was increased by 50% (from 40 to 60%) whereas maximum protein deposition increased by 74%. The combined effect was an increase in protein requirement of 17%. However, Campbell *et al.* (1991) found that there was no change in the efficiency of use of dietary protein in pST treated-boars, but the dietary protein requirements increased from 11 to 18% to support an increase in protein deposition from 119 to 215 g/d. This is clearly demonstrated in Figure 2 where the rates of protein and fat deposition are given for boars fed diets containing 11 or 18% CP and treated with excipient or pST. For boars fed low levels of protein there were no benefits of treating with pST, and in fact overall performance was reduced since fat deposition was decreased. Also, feeding additional protein to the control boars did not improve lean tissue deposition or growth. Feeding additional protein to pST treated boars however, caused quite substantial increases in protein deposition. Differences in response between the two studies may reflect the relative efficiencies with which boars, barrows and gilts deposit dietary protein. Thus, in the study by Campbell *et al.* (1991) conducted in boars, the control animals were already depositing dietary protein with an efficiency of 62% and pST did not improve it. In the study reported by Boyd *et al.* (1991), involving barrows and gilts, the efficiency of utilization for the controls was lower (40%). Treatment with

pST increased efficiency of use of dietary protein to 60%, similar to that found by Campbell *et al.* (1991). Therefore, in boars of improved genotype at the finisher stage which already retain dietary protein with a high efficiency, improvements in protein deposition during pST treatment may only be sustained by increasing dietary protein intake. Conversely, in finisher barrows and gilts with lower efficiency of use of dietary protein, pST treatment can increase both the efficiency of use of dietary protein and the dietary protein requirement.

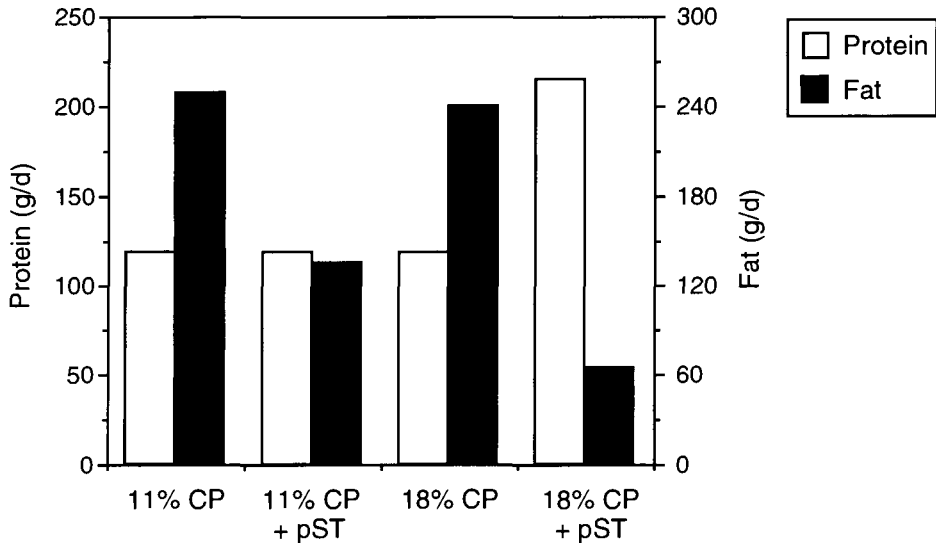


Figure 2. Effect of pST and dietary protein on protein and fat deposition in finisher boars (after Campbell *et al.*, 1991).

The relationship between energy intake and protein deposition is linear in the grower pig fed protein adequate diets. This was confirmed in the study of Campbell *et al.* (1988) which demonstrated that protein deposition increased with energy intake in young pigs treated with excipient or pST. At all levels of energy intake, protein deposition was higher and fat and total energy deposition lower in the pigs treated with pST. Extrapolation of the relationship between total energy deposition and energy intake to zero energy retention suggests that maintenance energy requirement is increased during pST treatment. The increases in energy requirements are at least in part due to increased protein synthesis in muscle and in visceral tissues, such as gut and liver, which are increased in mass during pST treatment. Also, *ad libitum* energy intake was 10% lower in pigs treated with pST. In older pigs, where *ad libitum* feed intake is greater, the relationship between energy intake and protein deposition is more typically a linear-plateau one. Campbell *et al.* (1991) reported data depicting the relationship between protein deposition and energy intake in finisher boars and gilts treated with excipient or pST. The control gilts exhibited the expected linear-plateau relationship with protein deposition plateauing at 112 g/d at an energy intake of approximately 30 MJ DE/d. While gilts treated with pST still exhibited the linear-plateau response in protein deposition to energy intake, they had increased protein deposition at every level of energy intake. Protein deposition plateaued at 203 g/d at an energy intake of 33 MJ DE/d. *Ad libitum* feed intake was decreased by almost 20% and maintenance energy requirement increased by 27%. The benefits of providing additional energy to the pST-treated gilts and the penalties for doing likewise for the control gilts are demonstrated in Figure 3. Increasing the energy intake from the inflection point (30 MJ DE/d) for the control gilts up to what might be considered to be commercial feed intakes (30 MJ DE/d) resulted in a considerable increase in fat deposition with no change in protein deposition for the control gilts whereas it caused an increase in protein deposition and a modest increase in fat deposition in the pST-treated gilts. For control boars there was the expected linear-

plateau relationship between protein deposition and energy intake whereas for pST-treated males the relationship was linear (see Campbell *et al.*, 1991). Therefore, if the full benefits of exogenous pST are to be achieved commercially then feed intake needs to be maximised, particularly for improved boars.

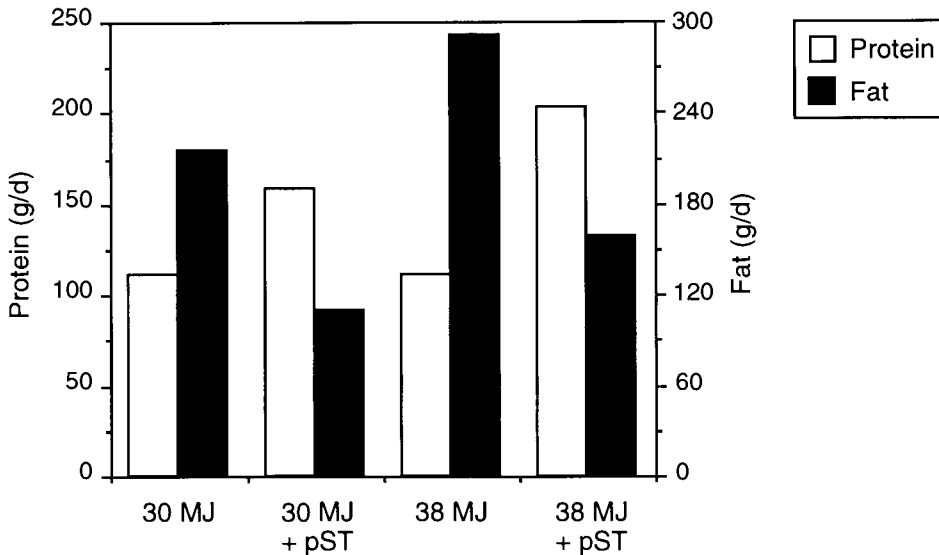


Figure 3. Effect of pST and dietary energy on the rate of protein and fat deposition in finisher gilts (after Campbell *et al.* 1991).

### $\beta$ -agonists

Treatment of pigs with  $\beta$ -agonists, particularly ractopamine, generally has resulted in dose-dependent increases in ADG, FCE and carcass lean content (see Dunshea 1991; 1993). Contrary to what happens during pST treatment, feed intake is typically unchanged (Adeola *et al.*, 1990; Gu *et al.*, 1991; Yen *et al.*, 1991) or decreased slightly (Adeola *et al.*, 1990; Watkins *et al.*, 1990; Mitchell *et al.*, 1991) during  $\beta$ -agonist treatment. While there is general agreement that protein deposition is increased during  $\beta$ -agonist treatment, effects on fat deposition have been more equivocal. For example, while ractopamine increased protein deposition in boars, gilts and castrates of an improved genotype by 15, 42 and 41% respectively, there was little effect on fat deposition (Figure 4; Dunshea *et al.*, 1993a). Other  $\beta$ -agonists which have improved performance in finisher pigs are salbutamol, cimaterol, clenbuterol, Ro 16-8714, BRL- 47672 and L-644,969 (see Dunshea, 1991, 1993, 1994; Dunshea and Gannon, 1995).

As for pST-treated pigs, the  $\beta$ -agonist ractopamine has no effect on nutrient digestibility and so effects must be predominantly post-absorptive. The limited information, obtained only in finisher pigs, suggests that the increased protein deposition rates observed in response to  $\beta$ -agonists increase the dietary requirement for protein, since ractopamine and other  $\beta$ -agonists are not effective in pigs fed low levels of dietary protein. Thus, Anderson *et al.* (1987) found that ractopamine increased nitrogen retention in pigs of a moderate genotype which were fed a 16% protein diet, whereas nitrogen retention was decreased in pigs fed a 12% protein diet. Likewise, Bracher-Jakob and Blum (1990) found that the  $\beta$ -agonist Ro 16-8714 increased protein deposition in pigs receiving diets containing 14% but not 11% protein. The increased dietary protein requirement during dietary ractopamine treatment has been confirmed in an experiment in which six levels of dietary protein were fed in restricted amounts (30 MJ DE/d) to finisher gilts (Dunshea *et al.*, 1993b). Efficiency of use of dietary protein was not altered by ractopamine since protein deposition increased with protein content at a similar rate for both the control and ractopamine-treated pigs over at least the two lowest levels of

dietary protein (<11% protein). However, at higher dietary protein contents, the plateau or maximal protein deposition rate was 23% higher in the gilts receiving ractopamine (96 vs 118 g/d for control and ractopamine-treated pigs, respectively). The levels of dietary protein required to support maximum protein deposition were 12.7% for the control and 15.8% for the ractopamine-treated pigs. At dietary protein contents which maximise protein deposition in the control gilts there is no difference in performance between the control and the ractopamine-treated gilts. The benefits of providing additional dietary protein are shown in Figure 5. As dietary protein level is increased there is no further improvement in protein deposition in the control gilts. However, protein deposition is increased in gilts treated with dietary ractopamine. When discussing protein and energy requirements it is important to realise that ractopamine and other  $\beta$ -agonists stimulate protein deposition only in skeletal muscle whereas pST stimulates protein deposition in all tissues. Metabolically active visceral tissue such as gut, heart and liver are often reduced in mass. These relative differences in carcass and non-carcass protein may have some impact upon the "ideal" profile of amino acids for pigs treated with  $\beta$ -agonists (Reeds and Mersmann, 1991).

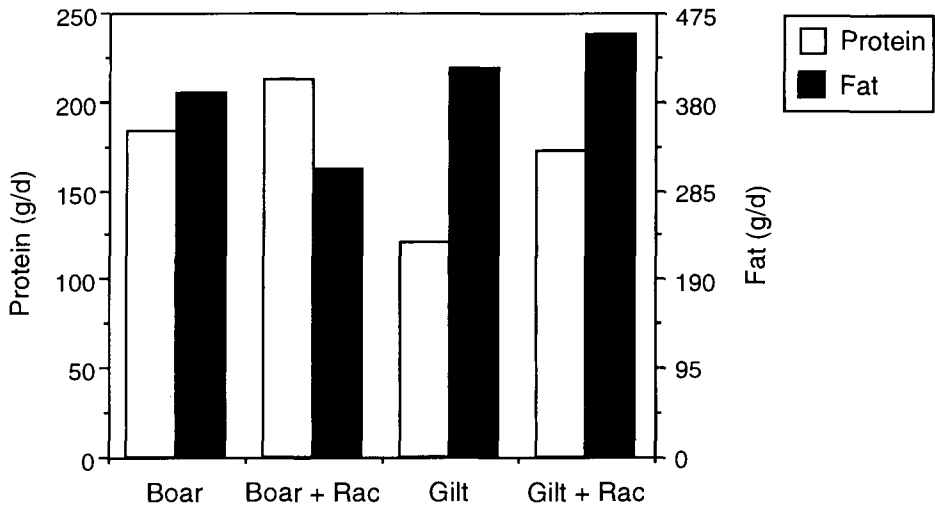


Figure 4. Effect of dietary ractopamine (Rac) on protein and fat deposition in finisher gilts and boars (Dunshea *et al.*, 1993a).

When the data for gilts presented in Figures 4 and 5 are compared it becomes obvious that even when protein is adequate, the responses to dietary ractopamine is also limited by dietary energy intake. Therefore, it was decided to investigate the interactions between dietary energy intake and protein and fat deposition in ractopamine treated finisher gilts and boars (Dunshea *et al.*, 1993c). The relationship between protein deposition and DE intake for the control gilts was of the linear-plateau form with carcass protein deposition reaching a plateau at 140 g/d at an energy intake of 36 MJ DE/d. However, in ractopamine-treated gilts protein deposition increased linearly with increasing energy intake up to a maximum of 191 g/d at an *ad libitum* DE intake of 47.2 MJ DE/d. Supplementation of the diet with ractopamine increased protein deposition at every level of energy intake. The slope of the linear ascending portions of the curve were not different and the improvement in protein deposition due to dietary ractopamine was 21 g/d up until a DE intake of 36 MJ/d. For boars receiving either 0 or 20 ppm of

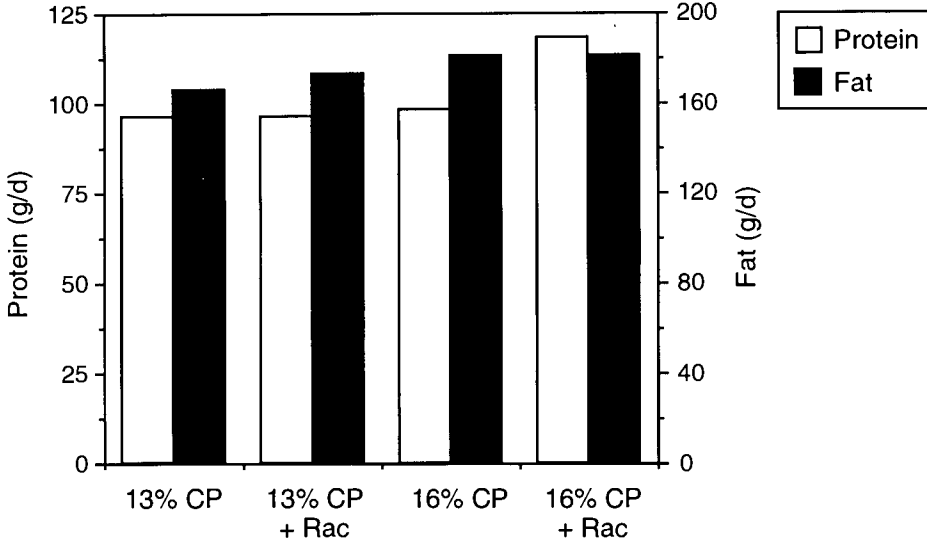


Figure 5. Effect of dietary protein and ractopamine (Rac) on protein and fat deposition in restrictively-fed (30 MJ DE/d) finisher gilts (Dunshea et al., 1993b).

ractopamine the relationship between protein deposition and energy intake was linear up until *ad libitum* DE intakes of approximately 45 MJ/d. While the slopes of these lines were the same, the benefit to protein deposition in boars (19 g/d) was similar to that observed in gilts. Therefore, dietary ractopamine increases protein deposition in both gilts and boars at every level of energy intake, but *ad libitum* intakes are necessary to maximise protein deposition in improved genotypes treated with ractopamine. Also, the differences in protein deposition between boars and gilts are still evident during ractopamine treatment. Despite an increase in protein deposition dietary ractopamine had no effect on maintenance energy requirement. This may be due to the energy requirements of an increased skeletal muscle mass being offset by a reduced visceral mass.

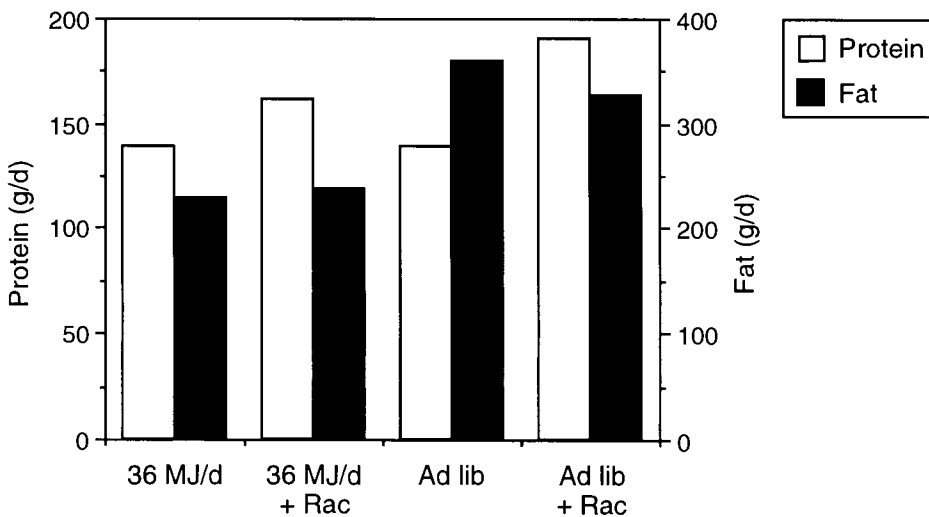


Figure 6. Effect of dietary energy and ractopamine (Rac) on protein and fat deposition in finisher gilts (Dunshea et al., 1993a).

## Betaine

Betaine is a methyl donor which has been investigated as a partial replacement for choline in pig and poultry diets since it is less expensive and less corrosive than choline. As an additional benefit it was found that carcass fat was reduced in chickens receiving supplemental betaine but not when receiving supplemental choline or methionine (Saunderson and MacKinlay, 1990). More recently, the effects in pigs have been investigated. Cadogan *et al.* (1993) found that although betaine had no effect on growth performance of finisher gilts, backfat at the P2 region was decreased from 17.6 to 15.0 mm. Given that there was no change in ADG or feed intake this suggests that there was a repartitioning of adipose tissue fat. On the other hand, Mooney and Cromwell (1994a) found that although there was no effect of betaine on back fat at the 10th rib there was an increase in loin eye area at this site. Again growth performance was not altered, suggesting a change in body conformation rather than rates of lean and fat tissue deposition. In another study, Smith *et al.* (1994) found that betaine did not alter growth performance, loin eye area or backfat. These types of findings and variable responses on farm have somewhat confused researchers and producers alike.

In an effort to clarify the situation, Henman (1995) conducted a comprehensive study to investigate the interactions among gender, dietary methionine and betaine in finisher pigs. They found that while there were no significant main effects of betaine on growth performance, P2 or loin eye area there were some significant interactions between methionine and betaine for FCR and loin eye area. These data suggest that at low levels of dietary methionine at least partial substitution of betaine for methionine can occur and that there can be beneficial effects of supplementation with betaine. These data also suggest that methionine has an important metabolic role as a methyl donor, not necessarily related to protein metabolism.

## Chromium picolinate

It has been suggested that dietary chromium may increase insulin sensitivity (Steele *et al.*, 1977) and over the last two decades it has been investigated as a potential means of manipulating fat deposition in humans and farm animals. While chromium chloride has been successful in improving the growth rate of turkeys (Steele and Rosbrough, 1979) or protein deficient rats (Mertz and Roginski, 1969), effects in pigs are more equivocal (Page *et al.*, 1993; Mooney and Cromwell, 1994b). In part, this may reflect inefficient absorption of chromium since the absorption and utilisation of chromium may be dependent upon incorporation into an organic molecule such as picolinate (Evans and Johnson, 1980). There has been a recent flurry of activity in the US to assess the efficacy of chromium picolinate for improving growth and carcass composition in pigs but still the data are confusing. Page *et al.* (1993) reported the data from three experiments conducted to determine the efficacy of chromium chloride, chromium picolinate and picolinate alone in grower/finisher pigs. While there were no effects of chromium or picolinate on growth rate or FCR there were dose dependent effects of chromium (as picolinate) on loin eye area and backfat depth (Figure 7). Others have also seen little or no effect of chromium picolinate on growth rate or FCR (Evock-Clover and Steele, 1994; Mooney and Cromwell, 1994ab; Smith *et al.*, 1994) except perhaps an improvement in performance in weanling piglets (van Heugten and Spears, 1994). To confuse the issue, Boleman *et al.* (1994) found that chromium picolinate depressed growth when fed to pigs from 20 to 103 kg but not when introduced at 55 kg. Lean tissue deposition or loin eye area was increased and fat deposition or backfat decreased by chromium picolinate in some studies (Page *et al.*, 1993; Mooney and Cromwell, 1993, 1994a; Boleman *et al.*, 1994) but not in others (Evock-Clover and Steele, 1994; Mooney and Cromwell, 1994b; Smith *et al.*, 1994). Clearly further work is required but the general consensus appears to be that growth rate and FCR remain unaltered whereas carcass lean content can be increased by chromium picolinate. Alternatively, there may be changes in body conformation or the partitioning of nutrients between carcass and non-carcass components. Indications are that the latter could be the case since liver size is decreased (Boleman *et al.*, 1994) and dressing percentage increased (Page *et al.*, 1993) by chromium picolinate.

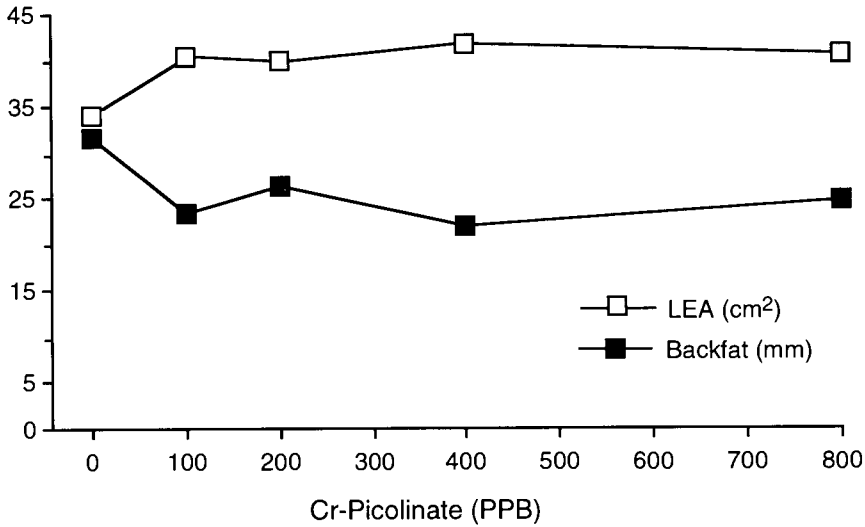


Figure 7. Relationship between dietary chromium picolinate concentration and loin eye area (LEA) and backfat depth at the 10th rib in finisher pigs (Page *et al.*, 1993).

### IGF-I

The growth promoting effects of pST on lean tissue deposition are thought to be mediated via the insulin-like growth factors (IGF's). This is in part based upon their ability to stimulate protein synthesis in *in vitro* culture systems. Also, IGF-I and its analogues have been shown to increase lean tissue and whole body growth in the rat. Finally, treatment of pigs with exogenous pST causes an increase in circulating levels of IGF-I and lean tissue growth.

While IGF-I has been shown to acutely improve whole body and hindlimb nitrogen balance in protein restricted pigs (Malmoff *et al.*, 1994), when IGF-I has been administered for longer periods to finisher pigs fed protein adequate diets they have had little effect upon growth (Walton, *et al.*, 1994). Furthermore, LR<sup>3</sup>IGF-I (a potent analogue of IGF-I) reduces growth rate and the plasma concentrations of IGF-I and the major IGF-binding proteins in finisher pigs fed protein adequate diets (Walton *et al.*, 1994; Dunaiski *et al.*, 1994). Under these conditions LR<sup>3</sup>IGF-I treatment also reduces the amplitude of endogenous pST pulsatile release Dunaiski *et al.* (1994). Therefore, while it appears that IGF-I can increase or improve lean tissue growth in rats or protein-restricted finisher pigs, negative feed back mechanisms limit its usefulness in the finisher pigs fed adequate dietary protein.

So what is different between rats and the finisher pig? The clue might lay in their relative sensitivity and responsiveness to exogenous ST. The rat is relatively insensitive and unresponsive to exogenous ST but sensitive to the growth promoting properties of IGF-I and especially IGF-I analogues. On the other hand, the finisher pig is highly sensitive and responsive to pST, relatively unresponsive to exogenous IGF-I and responds negatively to potent analogues of IGF-I. However, there is one class of pig which is unresponsive to pST but whose potential for growth is relatively untapped; the neonate. The young pig is unresponsive to exogenous pST up to at least 3-4 weeks of age (Harrell *et al.*, 1994; Walton and Dunshea, unpublished) at a time when endogenous production of pST and the number of tissue pST receptors are low. Therefore, it is possible that exogenous IGF-I or analogue treatment of baby pigs may not result in negative feed back inhibition of endogenous hormone secretion and will allow the growth promoting properties of IGF-I to be expressed. This was confirmed when Schoknecht *et al.* (1993) reported that treatment of sucking pigs for 7 days from 3 days of age increased growth rate by about 10%. More recently, the effects of IGF-I and LR<sup>3</sup>IGF-I have been investigated in an experiment where piglets were removed from the sow after obtaining



colostrum and then artificially reared on whole-milk from cows. Cows' milk was chosen since it has been demonstrated to have a protein:energy content in excess of that which maximises growth in the piglet (Williams, 1995). The first thing to note is that just by allowing the piglet *ad libitum* access to a high protein milk (the protein:energy ratio of sows' milk is only 70% that of cows' milk) growth rate is increased to almost double what is generally seen in the sucking piglet (220 g/d). Importantly, this performance can be further enhanced by IGF-I or the analogue LR<sup>3</sup>IGF-I. The increase in growth in IGF- or analogue-treated piglets was not immediate and did not become apparent until the latter half of the experiment. While this may be a delayed response it may be that the visceral responses precede or are actually causative of the increase in peripheral tissue growth, particularly for LR<sup>3</sup>IGF-I. Thus, with LR<sup>3</sup>IGF-I treatment the weights of the liver, small intestine and spleen increased by 12, 16 and 47%, respectively.

**Table 1. Effect of IGF-I or LR<sup>3</sup>IGF-I infusion on growth performance and organ size in artificially-reared piglets (Walton and Dunshea, unpublished).**

	Control	IGF-I	LR <sup>3</sup> IGF-I	SED	P value
ADG, 0-18 d (g/d)	378	397	412	14.5	0.13
ADG, 9-18 d (g/d)	386	423	457	17.6	0.01
Small intestine (g)	329	349	380	33.0	0.34
Liver (g)	286	303	321	17.5	0.19
Spleen (g)	19	16	28	2.1	0.001

In a subsequent experiment the interactions between nutrient intake (manipulated through establishing litter sizes of six and 12 piglets) and LR<sup>3</sup>IGF-I infusion in sucking piglets was investigated. Again, although an increase in growth during LR<sup>3</sup>IGF-I treatment was observed, this did not become manifest until the latter stages of the experiment (Table 2). Also, growth responses to LR<sup>3</sup>IGF-I were greatest in the piglets from litters of 12. Infusion of LR<sup>3</sup>IGF-I increased the growth of the small intestine, liver and spleen. As was the case for growth rate, the responses were greatest in the piglets from the large litters. In addition, two pigs from each litter received daily injections of either saline or pST. There was no effect of pST on pre-weaning growth rate (Walton and Dunshea, unpublished).

**Table 2. Effect of LR<sup>3</sup>IGF-I infusion on growth performance and organ size in sucking piglets from litters of six or 12 piglets (Walton and Dunshea, unpublished).**

Litter size	Six		Twelve		SED	P value	
	Control	LR <sup>3</sup> IGF-I	Control	LR <sup>3</sup> IGF-I		L <sup>a</sup>	T <sup>a</sup>
ADG, 0-27 d (g/d)	299	304	187	199	19.0	<0.001	0.432
ADG, 18-27 d (g/d)	294	325	114	167	32.0	<0.001	0.036
Small intestine (g)	359	373	247	311	34.0	0.011	0.047
Liver (g)	263	312	168	221	23.0	<0.001	0.002
Spleen (g)	29	53	16	40	6.3	0.033	<0.001

<sup>a</sup>L, litter size; T, LR<sup>3</sup>IGF-I treatment.

While the growth responses in themselves are exciting the discussion will focus on the effects upon visceral and gut development. Growth factors are present in relatively high quantities in colostrum and play an important part in gut development (Widdowson *et al.* 1976). For example, piglets which were not allowed access to colostrum but received milk replacer supplemented with IGF-I for 4 d had greater small intestinal weight (+28%) and jejunum villus height (+74%) than their control counterparts (Burrin *et al.*, 1995). Using lower doses of IGF-I, Xu *et al.* (1994) found no effect on piglet gut growth but a significant increase in the size of the pancreas after the animals received infant formula supplemented with IGF-I for 24 h. Gut development and growth in the piglet is essential

for efficient nutrient absorption and protection against bacterial invasion. The growth factor content of milk decreases with advancing lactation and so an opportunity exists to enhance gut growth and development through supplementation with exogenous growth factors. This is particularly pertinent given that the pig gut can be relatively immature at weaning and the pig suffers a growth check at or around this time. The findings that systemic infusion of LR<sup>3</sup>IGF-I increases gut and visceral growth, particularly in piglets from large litters, which are presumably under greater nutritional stress than those from small litters, suggests that this peptide may confer an ability to better handle the nutritional and disease challenges that can occur around weaning. For example, the gut is the route of absorption of many enteric bacteria and is also the site of inflammation caused by these bacteria. In addition, the liver is a major metabolic tissue and is the site of detoxification and synthesis of many metabolic enzymes. Finally, the spleen is a major immune tissue and an increase in size and lymphocyte proliferation (Jardieu *et al.*, 1994) during growth factor treatment may augment immune response. The results of the systemic IGF-I and analogue infusion studies lend support for a role of IGF-I and analogues to stimulate growth and visceral development in the piglet. Since there is some evidence that these growth factors are orally active in neonatal pigs, gut and visceral development may be achieved by incorporation of growth factors into the diets of early weaned or supplementally fed piglets. Furthermore, it is possible that these growth factors may be further modified to protect them from digestion and be absorbed from the gut in older piglets.

### Conclusion

There are a number of options available now or in the near future to manipulate growth or body composition in the finisher pig. Porcine somatotropin has already been approved for use as a daily injectable metabolic modifier in the Australian pig industry. Use of pST will probably be strategic, with producers concentrating on treating gilts. Alternatively, pST may be used in both boars and gilts and allow producers to produce heavier carcasses without being penalised for over-fat carcasses. If pST is used then producers will need to provide adequate dietary protein to allow the growth promoting effects to be expressed. Energy dense diets or management techniques to maximise feed intake should also be employed by producers to maximise returns on their investment in pST. As yet none of the  $\beta$ -agonists have been approved for commercial use in Australia but at least some are currently under review by authorities in the USA. If  $\beta$ -agonists such as ractopamine are approved for use then again attention will have to be paid to both the protein and energy intakes of pigs receiving dietary  $\beta$ -agonists. Betaine and chromium picolinate have the potential to improve carcass composition and may be beneficial under some circumstances. Producers are advised to assess these metabolic modifiers under their own conditions. While the IGF-I family of peptides do not stimulate growth in the finisher pig there may be opportunities to use them to manipulate growth and development of the neonate or early weaned pig.

## MANIPULATION OF ENDOGENOUS HORMONES TO INCREASE GROWTH OF PIGS

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### Introduction

The close link between the growth of the pig and economic returns has provided the impetus for a considerable amount of research into ways to maximise growth rate, while maintaining a viable production system. Considerable advances have been made to develop the use and show the value of administration of exogenous agents such as porcine somatotrophin (pST) and  $\beta$ -agonists in increasing pig growth. In particular pST is likely to soon be in use in the Australian pig industry. However, there are two particular constraints to the widespread use of exogenous treatments. First, the problems in maintaining continuous or frequent delivery of the agents for a prolonged period of time, especially in the case of hormones, has not been satisfactorily solved. Second, the regulatory requirements for withdrawal and public perceptions of the use of these agents limits the range and duration of their application.

Consequently, there has been a desire to investigate alternative strategies to enhance growth which have targeted manipulation of the endogenous hormone levels of animals. The two major approaches have been genetic manipulation and immunomanipulation of hormones. Genetic manipulation involves the alteration of the genome of the animal in such a way as to permanently alter its capacity for growth. Immunomodulation uses the development of an immune response against endogenous hormones to produce long term alteration of hormone levels. Neither of these methods is completely free of the problems associated with endogenous administration, but they either remove (genetic manipulation) or reduce (immunomanipulation) the need for long-term administration.

### Genetic manipulation of growth.

The isolation and sequencing of the genes associated with growth functions from a number of species, including the pig, in combination with advances in recombinant DNA technology has presented the opportunity of directly manipulating the genetic capacity for growth. The production of transgenic pigs is difficult and costly, but a successful outcome offers the opportunity to make large genetic gains in a short time.

Initial work in this area involved the introduction of the genes for porcine (Ebert *et al.*, 1990), human (Brem *et al.*, 1985), murine (Ebert *et al.*, 1988) and bovine (Pursel *et al.*, 1987) somatotrophin (ST) in combination with various promoter/regulatory sequences into pigs. While the frequency with which successful expression of the transgenes produced was low, both in absolute terms and relative to that obtained in mice, sufficient numbers have been produced to show that at present this is not a viable technology. The overall growth response in some pigs was favourable, with higher growth rates and higher feed conversions (Pursel *et al.*, 1990) indicating that the extra ST was acting in a similar fashion to the endogenous hormone with respect to tissue growth. The amount of ST in serum varied widely but those with prolonged elevations had a number of undesirable side effects including bone weakness, infertility and gastric ulcers. These ranged from mild to severe, and for this reason, the transgenic approach appears to be non-viable.

The principal reasons for these problems appears to be the result of the continuous expression of the transgenic ST genes throughout development, and the prolonged increase in ST levels in the circulation. Model systems have been developed in mice where transgenes are controlled by addition of inducers in the diet (Shanahan *et al.*, 1989) and, in such systems, expression can be regulated to defined periods. This has the potential to minimise or eliminate the health and reproductive problems. The genetic constructs used in pigs seem much less responsive to such manipulations. However, if the technology

improves both in the control of the insertion and expression of transgenes, transgenic pigs containing ST transgenes may become a viable means of manipulating growth.

Extra copies of the genes of two other growth related hormones, growth hormone releasing factor (GHRF) and insulin-like growth factor I (IGF-I), have been inserted into pigs. The former produced elevated levels of GHRF, but not of the native hormone, and growth hormone was not elevated (Pursel *et al.*, 1989a). The IGF-I transgenic pigs had a low proportion expressing the hormone, but none survived long enough for performance testing (Pursel *et al.*, 1989b).

General application of this technology for increasing growth awaits advances in the technology of transgenics. An alternative approach is to accelerate the growth rate of young pigs by modification of sow lactation. This has the advantage that, correctly expressed, the effect of the transgenesis is limited to a restricted time frame and to a particular group of tissues. That this is a feasible approach has been demonstrated by the production of transgenic pigs expressing the mouse whey acidic protein in milk (Wall *et al.*, 1991). Further development of this approach awaits the identification of candidate genes which can improve the quality or quantity of sow's milk with reasonable expectation that this will also produce an increase in growth rate of the piglets.

### Immunomodulation

Another method of altering the activity of endogenous hormones is the use of immunomodulation. In this technique, specific immunity is developed in the animal by either vaccination (active immunity) or by administering concentrated antibodies by injection (passive immunity) directed against a particular hormone. The latter method is used principally as a research tool. The presence of antibodies that bind to the hormone may result in either a decrease or increase in the physiological responses that are modulated by the hormone. This technique has the attractions of requiring less frequent administration than exogenous hormone treatments, and that vaccination is an accepted practice in animal production. However, actively immunizing animals usually requires several treatments and the effects may be delayed until sufficient immunity develops. It also generally produces, at most, moderate changes in activity because of the capacity of the endocrine system to maintain the normal concentrations by increasing or decreasing secretion in response to feedback regulation.

#### *Mechanisms of immunomodulation*

Active immunization against hormones may alter the physiological responses governed by the hormones in either a positive or negative direction. It is also possible that a combination of the two effects, acting together, may result in little change in the physiological state of the animal.

#### *Enhancing antibodies*

Immunization against a hormone can potentiate its activity and lead to an enhanced expression in its activity. Several mechanisms may contribute to this behaviour (Aston *et al.*, 1989). The antibodies produced may bind to the hormone and transport it in the circulation in a form where it is still able to bind to the receptor. This can be the result of the binding activity of the antibody being directed to regions of the hormone not involved with direct interaction with the receptor. Alternatively, the antibody may have sufficiently low affinity that it does not interfere with the binding to the receptor. A number of mechanisms may contribute to the ability of this "binding protein" behaviour of antibodies to enhance hormone activity. The metabolism and clearance of the hormone in the bound form is often longer, and this increase in the plasma half-life and duration of action produces a stronger response (Mihara *et al.*, 1991). The action of binding to the hormone may alter the nature of the interaction with the receptor, preventing events such as intracellular translocation and prolonging the interaction with the receptor. Certain aspects of the activity of a hormone may be enhanced if different receptors are involved and the binding of the antibodies favours the interaction with a subset of the receptors (Aston *et al.*, 1989).

The potentiating ability of immunization to hormones has been demonstrated in a number of studies. Use of certain monoclonal antibodies raised against ST and active or passive immunization against peptide fragments of ST has enhanced the activity of the hormone in rats (Wang *et al.*, 1990), mice (Holder *et al.*, 1988), sheep (Pell *et al.*, 1989) and pigs (Kraft and Wang, 1994; van der Hel *et al.*, 1994). Antibodies against certain fragments of GHRF potentiate the release of GH in sheep following injection with the releasing hormone (James and Pell, 1991). Antisera to IGF-I enhances the growth stimulating effects of exogenous IGF-I in dwarf mice (Stewart *et al.*, 1993). These studies suggest that if appropriate antigens can be identified, growth can be promoted in pigs by immunization against hormones that promote growth.

#### *Inhibiting antibodies*

The binding of the antibody to the hormone can block its activity either by preventing its interaction with the receptor or by accelerating the removal of the hormone from the circulation. In the first case the presence of sufficient high affinity antibodies to bind to all or a large proportion of the circulating hormone prevents it from interacting with the receptor (Van Oers and Tilders, 1991). The amount of hormone can also be reduced if the complex of hormone and antibody produce immune complexes which are rapidly cleared from the circulation, principally by the liver. The binding of antibodies to multivalent antigens such as the larger peptide hormones is more likely to produce immune complexes. Certain aspects of the activity of a hormone may also be inhibited if the binding of the antibody redirects the binding of the hormone, favouring interaction with one class of receptors at the expense of another class (Aston *et al.*, 1989). Immunization of animals with peptide fragments of hormones may tend to favour this form of response. For such an inhibition of hormone to increase growth the activity of the hormone must be to inhibit growth. Two hormones considered to have such an activity are cholecystokinin and somatostatin.

#### *Immunomodulation of cholecystokinin*

Results of experiments in which pigs were subjected to super-alimentation (Pekas, 1985) indicate that the capacity of the pig for growth is limited by appetite. Low appetite may be a result of continued selection for lean growth (Kanis 1990). If appetite is a limit to growth, and extra nutrients (administered by super-alimentation) are partitioned in the same proportion as that attained during *ad libitum* growth (Kanis and de Vries, 1992), then strategies to increase feed intake may have benefit in producing heavier pigs at slaughter or shortening the time taken to reach slaughter weight.

Studies in a variety of species, including the pig, indicate that one of the important satiety agents is cholecystokinin (CCK) (Baldwin *et al.*, 1983). The most common active form of the hormone in the circulation is the octapeptide (CCK-8), formed from the carboxyl terminus of CCK. In its native form, CCK-8 is sulphated on the tyrosine residue; the non-sulphated form has approximately a ten times lower biological activity. Structurally, CCK-8 is related to gastrin as they share the same five carboxy terminal amino acids (Figure 8).

The above observations have led to an approach to actively immunize pigs and other animals against CCK-8 on the basis that inhibiting the hormone will lessen its satiety activity. As a consequence, the animal would consume more feed and gain weight more rapidly. The results of experiments on rats (McLaughlin *et al.*, 1985) indicated that both feed intake and weight gain were increased in lean rats immunized against CCK-8. Immunization of sheep (Trout *et al.*, 1989) with the non-sulphated form of CCK (NS-CCK-8) however, did not alter feed intake or weight gain. Although these studies did not use the most active (sulphated) form of the hormone, subsequent use of a NS-CCK-8 antigen in pigs did promote growth and feed consumption (Pekas and Trout, 1990). The sulphated form of CCK-8 has also been tested in trials in sheep (Spencer, 1992) where rather than an increase in growth and feed intake, there was a decrease. It is possible that differences in the control of feed intake between ruminants and monogastric animals extend to the role and mechanism whereby CCK suppresses appetite.

The most striking success with CCK modulation of growth has been in studies on the pig by Pekas. Initial work by Pekas and Trout (1990) showed an approximately 11%

increase in body-weight and an 8% increase in feed consumption after immunization using NS-CCK-8 conjugated to human serum globulin (HSG) as the antigen. Over the period of the study, weight gains varied, slowing after immunizations presumably because of a reaction to the components of the vaccine. Once the anti-CCK titres had increased substantially, following the second booster immunization, the group immunized with NS-CCK-8 began to grow faster. Although the immunized group had heavier carcasses, their composition was not significantly different from that of controls (Pekas, 1991).

As the initial work had been conducted with individually housed barrows, another experiment was conducted using group-fed barrows and gilts (Pekas, 1993). Groups of barrows and gilts were immunized with NS-CCK-8, with carrier alone or not immunized. Pigs from each group shared the same pen and competed with each other for feed. Both immunized groups, barrows and gilts, gained weight faster than the controls.

During these studies, the variability in responses has been considerable and only relatively low anti-CCK titres were produced, despite multiple immunizations. As the two growth experiments showed that there was a correlation between antibody titre and weight gain, a study was conducted into the effect of the carrier protein on the nature of the response. The original carrier, human serum globulin (HSG) was compared with bovine serum albumin (BSA), keyhole limpet hemocyanin (KLH) and purified protein derivative (PPD). Of these, BSA was inferior while the KLH conjugate gave more consistent titres. The best response was from the PPD conjugate from which over 80% of the pigs had titres over 1:100 compared to 10% using the HSG conjugate.

A number of further requirements would have to be met to produce a practical vaccine. Most importantly, the response would need to be elicited by at most two immunizations, using an adjuvant, other than Freund's adjuvant, that is acceptable for animal usage. This may be achieved by using alternative forms of the vaccine which produce a much stronger immune response to CCK. As the cost of peptide is a major component of these vaccines, a second important requirement is the design of a vaccine that is cost effective and reproducible. The glutaraldehyde procedure used by Pekas uses a relatively large amount of peptide and is difficult to standardize.

#### *Immunomodulation of cholecystokinin using peptide analogues*

On the basis that developing the greatest titres to CCK-8 was the approach most likely to give practical growth responses, vaccines were designed to address problems of producing defined conjugates. This was based on the synthesis of a number of analogues of CCK-8. The principle features of these analogues was that they have a group that would facilitate the formation of a conjugate with a carrier protein, that the analogues have the sulphated form similar to native CCK-8 and that the orientation of coupling is controllable.

The structure of CCK, and the analogues used in the trials, are illustrated in Figure 8. The analogues maintain the sulphated tyrosine found in CCK-8. The methionine molecules in the native peptide were replaced with norleucine to enhance the stability of the analogues to oxidative processes. A large body of work shows that this change does not alter the ability of a number of peptides, including CCK, to interact with their receptor (Moran *et al.*, 1992). In each analogue, provision is made for the addition of a protected sulphhydryl group (S-acetyl thioacetic acid, SATA) which can be activated immediately prior to conjugation with the carrier protein. The SATA group is linked to the N-terminal (amino terminus) end of the analogue designated N-CCK-8, and to a lysine residue added to the C-terminal (carboxyl terminus) of the C-CCK-8 analogue.

These features enable the synthesis of conjugates with a defined orientation of the peptide, particularly with respect to the important sulphated group and the C-terminal pentapeptide which CCK shares with gastrin (pentagastrin). The orientation of peptides coupled to carrier protein can have a critical effect on the nature of the immune response. For example, antibodies raised against somatostatin antigens with either C- or N-terminal coupling only reacted with the natural material when the amino-terminal coupling was used (Lipkin *et al.*, 1988). A conjugate formed with the C-CCK analogue would present an unmodified form of the region of the peptide, around the sulphated tyrosine, that is characteristic of CCK and different to that of gastrin. The other analogue presents the peptide in a form where the linkage is through the N-terminal end, in the region around the sulphated tyrosine while the other terminus and the gastrin sequence is not modified.

Although the coupling chemistry is different from the previous studies in pigs, where glutaraldehyde was used to make the linkage to the carrier protein, the orientation of the N-CCK analogue is the same as in the previous work.

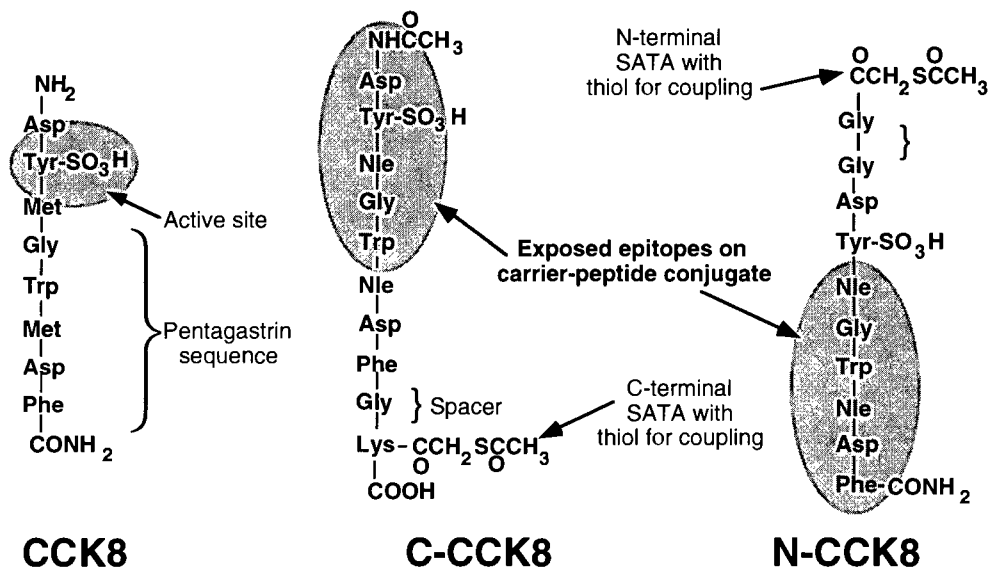


Figure 8. Structure of CCK8 and C- and N-terminally modified analogues used in immunization studies.

#### *Immunomodulation with cholecystokinin analogues in rats*

Initial trials with the CCK analogues were conducted in rats, and for technical reasons began with non-sulphated analogues. The carrier protein, ovalbumin, was modified by the addition of maleimide groups which react rapidly and strongly with free sulphhydryl groups (Peeters *et al.*, 1989). The C-(NS)CCK and N-(NS)CCK analogues were each coupled to the modified ovalbumin. Groups of female weanling rats were immunized with each of the CCK vaccines, and the ovalbumin carrier. A further group was not immunized. Three doses of the vaccine were administered at 3-week intervals, the first emulsified in Freund's complete adjuvant and subsequent boosters in Freund's incomplete adjuvant. Although rats were assigned to groups randomly on the basis of weight, there was a wide range of starting weights. This spread was maintained through the study and hence some analyses in this, and later studies, were conducted on data normalised for starting weights.

Table 3. Growth response of rats to immunization with (NS)CCK analogues.

Treatment	n	Gain/d <sup>1</sup>		Titre <sup>3</sup>
		(g)	(%) <sup>2</sup>	
Non-immunized	9	1.59 ± 0.11	0.83 ± 0.07	UD
Carrier immunized	10	1.41 ± 0.08	0.72 ± 0.08	UD
C-(NS)CCK	9	1.75 ± 0.13	0.96 ± 0.08	790
N-(NS)CCK	8	1.66 ± 0.11	0.91 ± 0.05	3830

<sup>1</sup>Weight gain over 21 d following final immunization. All results are mean ± SEM.

<sup>2</sup>Increase as a percentage of weight at final immunization. <sup>3</sup>Geometric mean of reciprocal of binding equivalents to standard rat anti-CCK pool on final bleed. UD = undetectable.

Overall, there was no significant difference in the final weight of either immunized group compared to controls (Table 3). However, when expressed as a percentage of initial weight there was a trend for the C-(NS)CCK group to grow faster in the latter stages of the study (Figure 9). However, titres to CCK were higher in the N-(NS)CCK group but, for both vaccines, there was no significant correlation with weight gain.

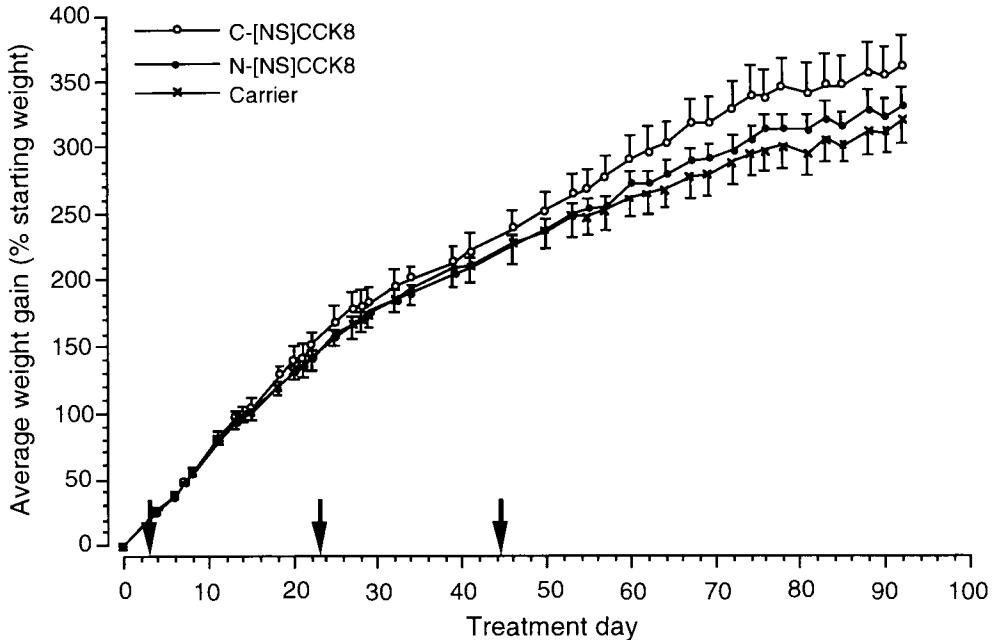


Figure 9. Growth rate of rats immunised with analogues of (NS)CCK8 over the duration of treatment. Data is represented as mean  $\pm$  SEM ( $n=10$ ). Arrows indicate day of immunization.

Another trial was conducted in rats using conjugates made with the sulphated form of the CCK analogues (C-CCK and N-CCK), and included the C-(NS)CCK from the previous trial. The hypothesis of this study was that a stronger response would be generated against CCK by using analogues that contained the sulphated tyrosine present in the native hormone. Rats in this trial achieved their maximum weight much earlier than in the previous trial, and gains following the last immunization were lower than in the previous trial. The final weights of the immunized groups were not significantly different from the controls either in absolute terms, or when expressed as a percentage of initial weight (Table 4). However, the greatest weight gain following the final immunization was in the N-CCK group,  $0.88 \pm 0.05$  g/d (mean  $\pm$  SEM) compared to  $0.59 \pm 0.06$  g/d in the control group.

Table 4. Growth response of rats to immunization with CCK analogues.

Group	n	Gain/d <sup>1</sup>		Final weight (% of start)	Titre <sup>3</sup>
		(g)	(%) <sup>2</sup>		
Carrier immunized	8	$0.59 \pm 0.06$	$0.25 \pm 0.03$	$350 \pm 10$	UD
C-(NS)CCK	7	$0.68 \pm 0.17$	$0.28 \pm 0.06$	$336 \pm 19$	692
C-CCK	8	$0.74 \pm 0.07$	$0.31 \pm 0.06$	$328 \pm 12$	271
N-CCK	8	$0.88 \pm 0.05$	$0.36 \pm 0.02$	$338 \pm 11$	1120

<sup>1</sup>Weight gain over 21 d following final immunization. All results are mean  $\pm$  SEM.

<sup>2</sup>Increase as a percentage of weight at final immunization. <sup>3</sup>Geometric mean of reciprocal of binding equivalents to standard rat anti-CCK pool on final bleed; UD = undetectable.



Titres against CCK were, again, highest in the N-CCK group but were only approximately one third as great as that in the previous study with non-sulphated analogues. This was also seen in the C-CCK group. The reason for this decline is not clear. The quality of the antigen had been monitored before use, and similar amounts were administered as in the previous trial. Modifications to the C terminal end of CCK analogues can reduce their biological activity which may reflect a shift in the conformation of the peptide. This change should also be reflected in a change in the ability to bind gastrin which shares the same five C-terminal amino acids. Indeed, antisera raised against both sulphated and non-sulphated N-terminal analogues bound gastrin, while the sera from the C-(NS)CCK animals did not bind any gastrin. Hence the C-(NS)CCK antisera must bind to the N-terminal region of the radio-iodinated CCK used in the antibody titrations. As this labelled CCK is a modified form of the non-sulphated form of CCK, it may bind less effectively to the C-CCK antisera which will recognise the sulphated form of the N-terminal region of CCK. This may also explain the lower titre that was detected when comparing N-CCK with previous N-(NS)CCK immunizations.

*Immunomodulation with cholecystokinin analogues in pigs*

The possibility that the immunological and hormonal responses to immunizations may differ between rats and pigs made it important to conduct equivalent trials in pigs. Young male pigs weighing between 10 and 15 kg were blocked by weight and randomly assigned to four treatments - non-immunized controls, carrier-only immunized controls, and immunized with the C-CCK or N-CCK vaccines. The first immunization was delivered approximately a week after weaning, when average weights were about 10 kg. Two booster doses were administered at four week intervals. Weight gains and antibody titres were measured regularly during the trial, and individual feed consumption was recorded after the last immunization.

The final weight and weight gains over the period following the last immunization did not differ among any of the treatment groups (Table 5). There was also no difference in the feed consumption and feed conversion efficiency. Initial antibody titres for the C-CCK group were much lower than that for the N-CCK group as was found in the previous rat trial. However, average titres in the N-CCK treatment increased after initial immunization, but did not increase significantly following the subsequent booster doses, rather, they remained at similar or slightly lower levels to that achieved early in the trial. Titres increased after each boost, but declined over the period to the next immunization to levels similar to that prior to the last immunization. This apparent failure of the vaccination process was not due to the quality of the antigen, as at least one pig had a "classical" response, similar to that seen in previous rat trials, with comparable final titres. At the end of the pig trial, measurable titres varied over a range of about 100-fold, compared to less than ten-fold in the rat trials. This inconsistent and generally weak immune response is the likely reason for the lack of any physiological response.

Table 5. Growth response of pigs to immunization with CCK analogues.

Group	n	Gain/d <sup>1</sup>		Final weight (% of start)	Titre <sup>3</sup>
		(kg)	(%) <sup>2</sup>		
Non-immunized	10	1.09 ± 0.03	2.24 ± 0.06	544 ± 16	UD
Carrier immunized	10	1.06 ± 0.04	2.21 ± 0.11	525 ± 9	UD
C-CCK	10	0.97 ± 0.04	1.95 ± 0.10	529 ± 19	UD <sup>4</sup>
N-CCK	10	1.03 ± 0.05	2.06 ± 0.12	542 ± 19	74

<sup>1</sup>Weight gain over 36 d following final immunization. All results are mean ± SEM.

<sup>2</sup>Increase as a percentage of weight at final immunization. <sup>3</sup>Geometric mean of reciprocal of binding equivalents to standard pig anti-CCK pool; UD= undetectable. <sup>4</sup>Two out of 10 animals had very weak titres, the others were undetectable.

There are a number of possible explanations for this disappointing immune response. The higher variability and lower response in pigs compared with rats may be due to intrinsic species differences, either in response to the antigen or to the adjuvant. The dose of antigen may not have been sufficient to trigger a sustained increase in antibody levels. Alternatively, the low titres may be the result of differences in the hormonal responses to the vaccination. If the rise in antibody levels and consequent removal of CCK triggers a hyper-secretion of the hormone, then, titres will be apparently decreased because of the saturation of antibody binding sites. The magnitude and variability of this hormone response may mask the size of the immune response, particularly if it is relatively weak. These, and other aspects of the hormonal and immune response, are currently under investigation.

The results to date have not provided unequivocal evidence that this treatment can increase growth. The current focus is to produce a stronger, more uniform immune response. This will involve using larger amounts of antigen in the vaccine and using a CCK analogue containing the native methionine residues.

#### *Immunomodulation of somatostatin*

The secretion of somatotrophin from the pituitary is subject to control from two hypothalamic hormones: growth hormone releasing factor (GHRF) which stimulates, and somatostatin (SST), which inhibits its release. The increase in growth rate and feed efficiency achieved with exogenous treatments of somatotrophin, coupled with the demonstration that SST inhibits the release of somatotrophin from the pituitary, makes the immunomodulation of SST a plausible means of increasing growth rate. That immunization against somatostatin can influence somatotrophin has been shown in studies in rats (Terry and Martin, 1981), sheep (Spencer *et al.*, 1983), cattle (Peticlerc *et al.*, 1988) and pigs (Dubreuil, *et al.*, 1989). The results of these studies suggest that a reduction in SST leads to higher basal levels of somatotrophin and reduce latency to repeated stimulation with GHRF.

While the influence of SST on somatotrophin is a direct action theoretically capable of influencing growth, somatostatin also influences other hormones and physiological systems. Indeed, as the effects of SST-immunisation on GH are inconsistent, the effect on growth may well be mediated by some other effect on the endocrine system. Somatostatin influences a number of hormones including thyrotrophin, CCK and gastrin (Dubreuil *et al.*, 1994). It is an important neurotransmitter in the gut and also affects gastric motility (Fadlalla *et al.*, 1985) in sheep. Alterations in gut physiology could lead to an increase in digestibility and absorption of nutrients and consequent increase in growth rate without direct involvement of somatotrophin (Sun *et al.*, 1990a).

Demonstration that immunomodulation of SST can increase growth rate has been inconsistent. While growth promotion has been achieved in sheep (Spencer and Garssen, 1983), cattle (Ingvarlsen and Sejrsen, 1992) and pigs (Huang *et al.*, 1990), there have been a number of studies, including pigs (Dubreuil *et al.*, 1989), that have not shown any beneficial effects on growth in the presence of a measurable immune response to SST.

#### *Immunomodulation with somatostatin in the pig*

A study in pigs was conducted using two forms of SST antigen and routes of administration. The principle antigen used in this study was a bovine serum albumin (BSA)-SST conjugate as used in previous work in sheep (Westbrook *et al.*, 1993). The other antigen tested was an ovalbumin OVA-SST conjugate formed using coupling chemistry, similar to that used for CCK, designed to favour the coupling of SST to the carrier protein by its N-terminus. Vaccines were administered to groups of female pigs at 3-week intervals on three occasions. Overall, there was no significant effect on growth rate in vaccinated pigs (Table 6). The immune response to SST was weak in both groups and, overall, was much lower than in studies with similar antigens in sheep.

**Table 6. Growth response of female pigs immunized against somatostatin (SST).**

Treatment	n	Weight (kg) <sup>1</sup>		Gain (kg/d) <sup>2</sup>
		Week 0	Week 14	Week 14
Carrier immunized	5	7.44 ± 0.34	74.1 ± 1.2	0.80 ± 0.03
BSA-SST	5	7.84 ± 0.51	72.8 ± 1.3	0.79 ± 0.01
OVA-SST	6	7.47 ± 0.54	78.9 ± 2.3	0.86 ± 0.04

<sup>1</sup>All results are mean ± SEM. <sup>2</sup>Weight gain over 56 d following final immunization.

The usual reason cited for lack of effect on growth is the development of inadequate titres to SST, and a common finding with immunisation of large animals including pigs, is a relatively low titre compared to studies in rats. Similar considerations apply to SST as to other forms of immunomodulation, in that certain types of immune response may act to increase the activity of SST. In addition, feedback stimulation and hyper-secretion of SST may also overwhelm the capacity of the immune response to block SST activity. Finally, the period over which an altered hormone response acts may be too short to allow for the detection of a significant increase in growth rate.

Previous work in sheep has shown that the lambs born to ewes actively immunized against SST are heavier (Westbrook *et al.*, 1993) and grow at a faster rate (Westbrook *et al.*, 1994) than those from control ewes. The factors responsible for the increased birth weight have not been determined, although differences in plasma metabolites between control and immunized ewes indicate that basic aspects of metabolism are affected by the treatment and may affect the provision of nutrients to the growing foetus.

The increased growth rate could also be explained by an increased consumption of milk. Somatostatin-immunized ewes produce 11% more milk when mechanically milked (Sun *et al.*, 1990b) and 20-30% more milk, when they suckled lambs (Westbrook *et al.*, 1993). The latter effect is at least partly due to an increase in the appetite of the lambs. Lambs fed milk harvested from immunized ewes acquired antibodies against SST and had a higher milk consumption and growth rate than those fed milk from non-immunized ewes. This effect was seen regardless of the immune status of the dam from which the lamb was born. The mechanisms responsible for changes in appetite have not been determined.

A preliminary study has recently been conducted on the effect of immunization of pregnant gilts against SST on the subsequent performance of their litters. Five gilts previously immunized against SST during the grower phase were boosted at 40, 60 and 91 days of pregnancy. Five control gilts were similarly immunized against the carrier protein alone. The weight of the gilts at farrowing and the number and weight of piglets born did not differ between each group. Immediately after birth, litter sizes were adjusted to approximately eight by the removal of either low-birth-weight piglets or those of equal weight to the mean for the litter. Litters were not mixed. The rate of growth (Figure 10) of the piglets fed from the SST-immunized sows was greater over the four weeks of the study and, from the first week onwards, the weight of the piglets from the immunized litters was significantly greater than controls. The final body-weight of the piglets suckled by the SST-immunized sows was nearly 20% greater ( $8.9 \pm 1.3$  kg, mean ± SD) than those suckled by non-immunized sows ( $7.6 \pm 0.7$  kg). A similar study conducted by Farmer *et al.* (1990) also found that there was no difference in the birth weight of pigs born to SST-immunized gilts. Antibody titres were passively transferred to the piglets and declined slowly over the four weeks of the study. Despite the higher growth rate of the piglets, SST immunized sows did not lose a significantly greater amount of weight than the control sows.

At present, it is only possible to speculate on the mechanisms responsible for the higher growth rates of the piglets. The physiology of the sow may have been altered to increase either the quality or quantity of the milk produced. Alternatively, anti-SST may alter the appetite of the piglet and indirectly increase milk production in the sow. As SST has numerous activities in the gut, consumption of milk containing anti-SST may alter gut function or the rate at which the gut matures in piglets. An indication that piglet factors

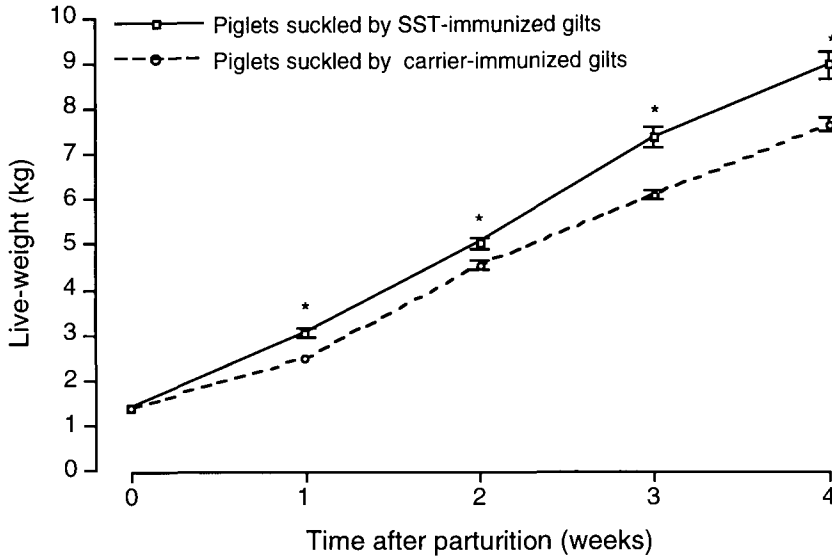


Figure 10. Growth of piglets suckled by carrier-immunized and SST-immunized gilts. Data is shown as mean  $\pm$  SEM ( $n=37$  and  $38$  respectively). Piglets were weaned 3 weeks after birth. \* Differences between mean live-weights at the same age are significant ( $P<0.05$ )

are involved in this growth effect comes from preliminary studies on the response of the stomach to pentagastrin. In piglets suckled by unimmunized sows, the stomach produces gastric acid in response to intravenous infusion of pentagastrin, the same response as seen in normal pigs. In those fed from immunized sows, this response is entirely absent. While it is not clear how this particular response might increase growth rate, it indicates that consumption of anti-SST antibodies can influence gastro-intestinal function. Whatever the mechanism, if the early gains of between 1.0 and 1.5 kg in piglet weight can be maintained for the subsequent growing period, a significant gain could be made in either final weight or in a shorter duration of the finisher phase.

### Conclusions

Direct manipulation of endogenous hormones by both genetic manipulation and immunomodulation are capable of producing improvements in pig performance to a degree which could benefit producers. However, both approaches are not technically reliable. In the case of genetic manipulation, progress awaits improvements in the technology of the control of the site and degree of expression of modified genes. Immunomanipulation of growth in rapidly growing pigs will require either a better understanding of the immunological and endocrine response or the targeting of different aspects of growth. It must be recognised that these treatments need to be applied as early as possible for the changes in growth rate, which are likely to be modest, to be reflected in significant gains at slaughter. For the future, there is some potential in modifying the growth of the neonatal pig by passive transfer of immunity from immunized sows through colostrum and milk.

## COMMERCIALIZATION OF NOVEL GROWTH ENHANCERS IN PIGS - ECONOMIC AND POLITICAL CONSIDERATIONS

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### Introduction

The translation of a novel way to enhance livestock growth from a laboratory technique into a method that is adopted and integrated into practical production systems can be a tortuous route with numerous issues to be addressed. The issues fall into four main areas: adoption by farmers; acceptance of the end-products by the post-farmgate sectors of the food industry; recognition of a commercial opportunity by an industrial corporation; and legislative approval by the relevant regulatory bodies.

Although prior assessment of the likely success in these four areas is important, very few of these *ex-ante* evaluations are published. First, much of the evaluation of new products is carried out by, or on behalf of, commercial companies. The results have a commercial value and are usually kept confidential. Second, several of the classical productivity enhancers are by-products of the quest for high-value human pharmaceuticals. As such, their development costs have not needed to be evaluated and compared with their potential revenues. This is likely to change with advances in biotechnology which are leading to more specifically designed products in both human and animal health fields.

A third reason for few published evaluations is that the results of such studies are more context-specific and ephemeral than the results of technical studies. The technical effects of a new product will depend on factors such as genetics, housing, nutrition and management. The economic effects will depend on all these factors, but also on the values of all the inputs and products involved. These values will be subject to market and policy fluctuations.

In spite of the limitations of economic analyses, some general conclusions can be drawn regarding the likely costs and benefits of growth enhancers (Bent, 1993a). For illustrative purposes, the potential impacts of three types of growth enhancers in UK pig production are presented in the second section of this paper.

The third section discusses some of the economic issues surrounding a selection of the novel growth enhancing techniques presented in this symposium.

### Issues in commercialization

This section considers the four main areas of issues to be addressed in commercialisation of growth enhancers, namely,

- legislative approval;
- farmer adoption;
- food industry acceptance; and
- corporate commercial opportunities.

#### *Legislative approval*

In many countries the use of productivity enhancing substances is subject to legislative approval by regulatory authorities. The channels and criteria can vary from country to country and within a country depending on the substance, species and method of administration (Bent, 1993b). The formation and expansion of free-trade zones such as the European Union (EU) and the North America Free Trade Agreement (NAFTA), and the emphasis on trade liberalization embodied in the Uruguay Round of the General Agreement on Tariffs and Trade (GATT) have led to increasing harmonization and mutual recognition of regulatory procedures. Whilst the traditional criteria for approval have been safety, quality and efficacy, there may be some regional differences in definition, measurement and acceptable levels, of these criteria.

Two important departures from this approach have occurred in the European Community in recent years. The first was the banning in 1985 of steroid hormone implants in beef production with effect from 1 January 1988 (Directive 85/649/EEC). The second departure from traditional procedures has been the failure of the European Council of Ministers to approve the use of bovine somatotropin (bST) in dairy production. These two issues have had serious consequences.

#### *The socio-economic criterion*

The banning of steroid hormone implants and the failure to approve bST when the three criteria of safety, quality and efficiency have been satisfied imply an additional criterion in the approval process, which has been referred to as the "fourth hurdle".

The reasons for a hormone ban cited by the European Parliament included the alleged inferior quality of meat produced with hormones (EP Opinion, 1981), the costs of disposal of surplus beef stocks, and the possibility that some cocktails of some hormones might reduce the immunity of some animals to some diseases (EP Opinion, 1985). No evidence was provided to support these statements. The European Commission included in its reasons for introducing the ban the variable assessment of the effects of hormones on human health between Member States which had given rise to different legislation in different countries that resulted in imbalances in competition. The opinions of the Economic and Social Committee (ECOSOC) of the European Community (ECOSOC Opinion, 1985) and the suppressed findings of the Commission's Scientific Working Group on Anabolic Agents in Animal Protection (the Lamming Committee) were dismissed in adopting the ban (Brand and Ellerton, 1989).

Similar, ill-defined socio-economic considerations have been cited at various stages in the blocking of approval of bST in the European Union. In the USA, approval was delayed because of the need for further information concerning the three traditional criteria. Once sufficient information was supplied, bST was approved for use and licensed. In the EU, socio-economic considerations have been cited throughout the registration application as reasons for delaying or refusing approval. Following approval of bST by the Committee for Veterinary Medicinal Products (CVMP) in February 1994, the only remaining reasons for non-approval of bST in the EU are political.

Although there has been a political and bureaucratic desire to use socio-economic criteria for regulatory approval (Bent, 1995b), the techniques for *ex-ante* estimation of socio-economic impacts are recognised by economists to have limited application (Bent, 1993b; Buckwell, 1993; Burton, 1993; Bent and Buckwell, 1993). Variations in the results will occur depending on the methodologies employed, the parameters considered and the economic climate assumed. This lack of replicability makes a socio-economic criterion totally inappropriate for regulatory approval procedures.

#### *Relocation of research and development*

The uncertainty that has surrounded the regulatory processes has led to the restructuring and relocation of research and development (R & D) activities by multinational pharmaceutical companies (R. Deakin, and D. Hammond, personal communication).

Relocation of R & D to more accommodating countries will be beneficial to the new hosts because of increased employment in R & D, manufacturing and marketing. Potential export earnings from the new technology will replace the costs of imports. Trials and field development are likely to be conducted under local conditions which will increase the applicability of the technique to local rather than foreign producers. The corollary of these gains are losses in countries from which the R & D activities move.

Although relocation of these activities will result in losses in some areas and gains in others, there is likely to be a net loss because of the overall reduction in R & D that is likely to occur where there is uncertainty about access to a significant market. The European Union is a significant market, not only because of its size (more than 20% of world pig production), but because of its relative affluence and stability which guarantee returns in hard currency. Thus 20% of world pig production represents approximately 50% of the commercial world market for pharmaceutical pig products.

### *International trade implications*

A further consequence of the departure from scientific criteria for approval is that harmonization and mutual recognition of approval procedures between trading blocks are jeopardised. The European ban of steroid hormones in beef production effectively introduced a trade embargo on imports of beef from all the major beef producing regions of the world. Because it was necessary to check that controls of hormone non-use were equivalent to EC standards, trade was disrupted even with the countries where hormones were either not used or not allowed.

This has proved to be politically embarrassing as the EC record for control of banned growth enhancers has been poor in some regions (ECOSOC Opinion, 1985). Some countries were vociferous in their objections to the new, non-tariff barrier. The USA retaliated by raising tariffs on particularly sensitive Mediterranean products exported from the European Community. However, some producers (eg., Texan ranchers) who derived little direct benefit from using hormones recognised and exploited new market opportunities for "hormone-free" products. Similarly, although EC beef producers were placed at a comparative disadvantage by the ban, they were able to identify certain higher-value, niche markets.

### *Political prospects*

There can be little doubt that the recent failures to apply only the scientific criteria of safety, quality and efficacy have had significant and global consequences. Political issues of real or imaginary threats to human health, socio-economic impacts, animal welfare, ethics and environmental impacts are potentially involved in the approval processes in the European Union. This political involvement is unlikely to diminish in the foreseeable future. Whilst it is nearly impossible to predict the effects of these factors on the commercialization of new techniques to enhance growth, some techniques may be regarded as more "politically-correct" than others. These considerations should not impinge on fundamental research, but will be crucial to the development of new technologies in the field. These political issues can only be ignored if a country's producers are totally isolated from international competition, with access to domestic markets protected by, say, phytosanitary barriers and no requirement or desire to compete in export markets.

### *Farmer adoption*

The second stage in commercialisation is to persuade farmers to adopt the new technique. For a new technique to tempt a farmer, it must be perceived to either increase profitability, reduce risk or effort, or to increase job satisfaction. Techniques may still be attractive where there is a sufficient trade-off between positive and negative aspects of these factors, for example, reduced profitability may be acceptable if there is also a sufficient decline in effort or risk. Broadly speaking, new techniques will increase profitability by either increasing technical efficiency (reducing the amount of input required per unit of product) or altering allocative efficiency (changing the proportions of inputs required) or increasing the value of the product itself.

Whilst the change in profitability may be assessed by comparing the value of these benefits with the costs of the new technology and its administration, it is the farmer's perception of the comparison that is crucial to adoption. Benefits can be readily perceived and attributed to new technologies in more intensive systems, such as piggeries, where many variables can be kept constant within a controlled environment with homogeneous groups of animals. Attribution is more difficult in extensive systems where climate, nutrition and genetic characteristics are more heterogeneous. Even in closely controlled environments, some significant benefits, such as increased annual throughput, are less perceptible and quantifiable than other changes. Problems of perception and quantification are further complicated in *ex-ante* studies in that, first, the technical responses observed in trials may not be replicated under field conditions and, second, a large technical response may not result in a large economic response.

*Technical responses under field conditions*

Results recorded in controlled trials are rarely replicated in commercial use. A range of treatments will be tested in trials under closely controlled conditions. The spectrum of experimental treatments may not give a result which is representative of the eventual dosage, timing and duration of treatment in commercial practice. Commercial treatment will be applied in a range of conditions which will be different from the controlled conditions in trials.

In general, technical responses are lower under commercial conditions than in controlled trials. Failure to include this aspect in economic evaluation can produce misleading results. For example, early economic studies of bST assumed a 20% yield increase in all cows across the whole lactation, i.e., a 20% yield increase for the total herd (Fallert and Hallberg, 1992). However, it is now clear that the yield response to a slow-release or intermittent treatment under field conditions is likely to be 12-15% for treated animals. Furthermore, up to 25% of cows in a herd will be heifers that are still growing and hence will not be treated. Of the remaining 75% of the herd, cows will not be treated in the first three months of lactation because of potential problems with conception. During the remaining seven months of lactation, these cows will produce 60% of total lactation yield. Thus the likely herd yield increase will be 12-15% of 60% of the yield of 75% of the cows (i.e., 5.4 - 6.8%.) rather than the 20% assumed. This is the maximum possible herd response assuming all eligible cows are treated all the time, even though such blanket treatment may not be economically worthwhile (Bent and Buckwell, 1993).

Although it is generally recognised that responses in commercial practice will be lower than in controlled trials, there are exceptions to this rule. One of the most significant exceptions is response to in-feed, anti-microbial growth promoters. It is generally accepted (Gropp, 1991) that responses are higher in commercial use than under controlled trials as a result of greater bacterial challenge and feed variability.

A further problem with extrapolating controlled trials to commercial conditions is that the very essence of controlled trials is that as many parameters as possible are held constant to enable attribution of the response to the treatment. In commercial practice there are no such constraints, and the major economic benefit of a new technology may not be the response, with all other parameters held constant, but may lie in the opportunity it offers to change the production system. For example, the in-feed, anti-microbial growth promoters have benefits in terms of changing feed efficiency and growth rate, but it has also been suggested that they were crucial to the development of modern, intensive systems of production. Thus many of the economic benefits of large-scale, labour-efficient, intensive systems are attributable to this technology, even though the technology may no longer be crucial to the operation of these systems. Similar opportunities for systems adjustments have been postulated for some of the new techniques under review in this symposium.

*Economic implications of technical responses*

The main effects of growth enhancers are on daily live-weight gain, feed efficiency and product characteristics. These substances can also change levels of illness and mortality in treated animals. These effects can modify the impacts of livestock production on the environment. The economic implications of these effects in pig production are discussed in general terms in the following paragraphs.

An increase in daily live-weight gain (DLWG) will reduce the time taken to finish a pig, which will mean that more batches can be put through a finishing unit per year. Annual building costs and other time-related costs may be spread over a greater number of pigs and kilograms of pigmeat thus reducing the cost per pig and per kilogram.

An alternative option suggested by some authors is that a similar number of animals may be finished but at higher weights (Lemieux and Wohlgenant, 1989). Although this option will not reduce the time-related costs per animal, the costs will still be spread over a greater weight of meat. This option has little value in markets where tight weight specifications are stipulated and is discussed further in the section on food industry acceptance.

Improvements in food conversion ratio (FCR) will result in a reduction in the quantity of feed consumed per unit of output. To some extent improvements in FCR and



DLWG are inextricably linked. Dramatic changes in FCR can occur with some growth enhancers. However the effects on costs of feed and total production costs may be minimal. A 20% reduction in feed consumption may appear significant when feed costs can account for anything up to 80% of total costs of production. However if the treatment is only from 7-30 kg, the reduction in feed consumed will be roughly 13 kg. This represents a small reduction when compared to the 80 kg of feed consumed by the pig's parents and the 200 kg consumed at other times during its life. Some treatments (eg., pST) require feed of higher quality and cost per kilogram which can offset or negate the savings from reduced feed intake.

With some enhancers (eg., pST,  $\beta$ -agonists) these gains are achieved through a change in the partitioning of energy between fat and lean. In addition to changes in growth performance there will be changes in carcass values which are discussed below. With other enhancers (eg., in-feed anti-microbials, probiotics) there appear to be changes in digestive efficiency which has been attributed to modulation of the gut flora. If treatment is at a vulnerable stage in the development of a pig's gastro-intestinal tract, the benefits of a healthy intestine may be realised long after treatment ceases. Few trials appear to examine the performance of treated animals after treatment has ceased. Any such post-treatment effects must be considered in an economic analysis.

Changes in mortality and incidence of illnesses have been reported with the use of some growth enhancers. In-feed, anti-microbial enhancers can dramatically reduce the incidence of gastric disorders in small animals, particularly in unhygienic conditions (Gropp, 1991). This can lead to a marked reduction in mortality or remedial treatment costs. The benefits of healthy development at a sensitive stage can extend beyond the immediate treatment period. Similar benefits are reported for piglets borne to and suckled by gilts immunised against somatostatin (McCauley *et al.*, 1995).

There are reports of increases in some disorders which might be attributed to use of productivity enhancers. The levels of mastitis recorded in some bST-treated cows have been higher than in control animals. However, the incidence was considered to be similar to other high yielding cows (Elanco, personal communication, 1991). Pigs treated with high levels of certain  $\beta$ -agonists have shown increased incidence of foot lesions. With some  $\beta$ -agonist treatments, pigs have been reported to have a stiffer gait, though this has not been shown to be detrimental to the animal's health or welfare (T. Rolf, personal communication, 1991).

The volume of manure generated in the production of pigs will change with changes in feed efficiency or mortality. The economic consequences of this effect are complex. An immediately apparent benefit is a potential reduction in manure storage requirements and disposal costs. Benefits can be far more significant in regions where there are strict environmental controls such as in The Netherlands, Denmark and north-west France. The value of changes in manure output will depend on the nature of these controls.

Changes in the absolute levels of emissions will depend on the feeding and housing technologies employed. A growth enhancer may significantly reduce nitrogen and phosphate emissions under current production techniques, but have a less significant impact if more sophisticated techniques, such as the use of synthetic amino acids and phytase, were widespread.

The characteristics of the livestock product may be affected by the use of some growth enhancers. Steroid hormones and  $\beta$ -agonists can change the conformation of a carcass, typically increasing the proportion of high value cuts. With some substances there may be changes within the product itself, such as a change in the ratio of fat to lean. This change may occur either in isolation or in conjunction with a change in conformation. There may also be changes in taste, tenderness and in cutting, processing and eating characteristics. The values of these changes are discussed in more detail in the section on supply chain acceptance. With some growth enhancers, particularly those that act in the digestive process, there may be no change in carcass characteristics.

There are other characteristics of livestock products that constitute "value" or "quality". The value of a product is a vector of characteristics comprised of form, time and location. Although less relevant in pig production, growth enhancers may be used to take advantage of seasonal pricing. More uniform growth of batches of animals may have benefits in terms of the price obtained, or through a reduction in the number of "drafts" taken from a pen or through an increase in the percentage occupancy of accommodation.

### *Cost of growth enhancers*

The cost of the growth enhancer includes the cost of the active ingredient, and also the costs of administration. There are two broad methods of administration, either the active ingredient is incorporated into the feed, or it is injected, implanted or administered orally either daily or intermittently. The choice of method of administration is determined by the nature and mode of action of the substance and by the system of livestock production in which the substance is to be used.

Administration by incorporation into feed is particularly attractive as it can involve minimal additional work. If significant amounts of concentrates are prepared by compounders, it is more likely that dosage levels and methods of incorporation are correct and can be subjected to closer regulation. This method of administration is ideal in circumstances where the enhancer is gut-active, as with the antibacterial growth promoters, or where the substance is not adversely modified in the gut. Some substances, such as the somatotropins, and, to a lesser extent, the steroid hormones, are adversely affected by the digestive process and therefore are preferably injected or implanted.

The nature and level of administration cost will vary widely depending on the method of administration. Feed incorporation by large-scale compounders can be relatively inexpensive, in that the costs of registration with regulatory bodies, upkeep of statutory records and inspection visits by controlling authorities can be spread over a large volume. For the small-scale or on-farm compounder, these can be significant costs per tonne treated. In-feed administration rarely involves additional farm labour, as the animals have to be fed anyway.

The costs associated with administration by implantation, bolus or injection consist of labour and equipment. Generally labour will be required to collect or muster animals into a treatment area, the animals may need constraining in a crush or race, and may need further constraining and/or swabbing during treatment. Daily treatment of unconstrained pigs in a pen may involve no more work than if the pigs were implanted once a month, where implantation required withdrawal of each pig from the pen, constraint in a crush and a precise operation of subcutaneous implantation.

### *Food industry acceptance*

The third stage in commercialisation is the acceptance of the end-products by the processing and retailing sectors of the food industry. These parties will be interested in intrinsic characteristics of the product such as carcass weight, percentage meat, conformation, cutting and eating qualities, visual appearance and cooking and processing properties. These characteristics will affect yield of high-value products and processing efficiency. The value attached to these properties varies from country to country and between processors within countries.

This sector is also concerned with intangible characteristics, such as animal welfare and product image, and is particularly sensitive to consumer attitudes. Processors and retailers are constantly gauging, and frequently prompting, effective consumer responses. This section of the paper considers the values attached to certain carcass characteristics by the food industry and the attitudes of consumers to changes in technology in food production.

### *Carcass characteristic values*

Prices for finished pigs in Europe, North America and Australasia are increasingly being paid on the basis of carcass weight and content of lean meat or fat cover. The proportion of pigs slaughtered in the USA for which payment is based on fat cover has increased dramatically in the early 1990s. Grading of pigs on estimated percentage meat content has been widespread in the European Union for several years. Significant advances have been made, particularly in Denmark, with research into remote, non-invasive grading using readings from several carcass sites.

### *Meat percentage*

In general, higher prices are paid for more meat and less fat in the carcass although the value of this premium varies among countries. For example, Danish farmers produce

leaner carcasses than the Dutch. Dutch farmers have access to the same technology as Danish producers and could produce leaner carcasses, but the financial benefits are not sufficient. The incentives offered to farmers reflect the values to the processing sector of the amount of lean meat in pig carcasses. If Dutch processors valued higher meat percentages, they may be obtained from Dutch farmers by providing similar incentives to those offered in Denmark and France.

The explanation for the difference in meat contents and incentives among countries lies in the end-use of the meat. Bacon and ham are major products of the Danish industry. The consumers demands low visual fat content in these products. French pigmeat production is split between fresh and chilled joints and charcuterie products such as pâtés. Relatively low fat content is desirable in the fresh and chilled markets, whilst a maximum meat percentage of around 55-57% is acceptable for charcuterie products. This means that a wider range of carcass grades can be accepted but that the value of increased meat percentage declines after 58%. It is worth noting at this point that the French processing sector has been a significant importer of pig fat from the USA.

A large proportion of Dutch pig production is exported either as processed products or for processing in Germany into a range of sausages and salamis. Relatively high fat content in these products is not only acceptable, but may be desirable.

Some of the growth enhancers, such as pST and  $\beta$ -agonists, can increase the percentage of lean meat in carcasses. Part of the benefit of such techniques lies in the increased value of these leaner carcasses. However processors in The Netherlands and France can already obtain leaner carcasses if they wanted them by increasing the incentives. The premiums offered represent a finely-tuned balance between the costs (or difficulties) of producing lean meat for the producer and the values or benefits of lean meat to the processors in their particular markets. If a new technology, such as pST, makes it easier or cheaper for a producer to finish leaner pigs, then the price incentive will be reduced. Any such adjustment in price premiums will affect non-adopters as well as adopters.

#### *Carcass weight*

The second carcass characteristic for which a premium is paid is weight. In some circumstances the carcass is used to produce saleable portions such as chops and joints. The size of these portions is dictated by the consumers. These portion sizes will determine the optimum size of the carcass. Trimming of some cuts may be required to maximise value of a carcass, but is uneconomic if applied to too many cuts. Thus there is clear maximum economic carcass size depending on the market. Tight weight specification is less important where carcasses are used to produce processed products.

The use of growth enhancers that increase daily gain and finish pigs at higher weights would appear to have limited application. Growth enhancers that are to be used to finish animals at higher weights should have two characteristics. They should reduce the variability of growth rates in groups of pigs and they should at least maintain the lean:fat proportions at higher weights. Substances such as pST and  $\beta$ -agonists have a role in maintaining lean:fat proportions, but it is not clear that they reduce variability of growth rates. In some production systems, particularly where feed intake is restricted, variability may be increased through accelerated differentials in pig size and intra-group competition. Uniformity in early growth and development in young piglets is likely to have important interactions with later variability. In this respect, the economic benefits of some of the enhancing techniques used at the peri- and neo-natal stages may not be realised until the later stages of finishing.

#### *Other carcass characteristics*

Slaughterers and processors are concerned with carcass characteristics other than meat percentage and carcass weight. In discussions about the potential use of hormone and  $\beta$ -agonists, Danish abattoirs were of the opinion that increases in meat percentage above 63% were not particularly desirable because of deterioration in visual characteristics and increasing difficulty in cutting. There are also concerns about the effects of reductions in intra-muscular fat levels on visual appearance and cooking and eating qualities. Some French processors have also expressed concerns about possible

changes in the types of fat in the carcass which would affect cooking characteristics both in the home and in processing plants.

#### *Consumer responses*

The fear of adverse consumer reactions to new technologies has been observed in bureaucrats and politicians (Bent, 1993b) and in farmers and processors (Floriot *et al.*, 1994). Acceptability of production techniques to consumers can have dramatic effects on markets as illustrated by various food scares and by the growth of markets for products like free-range and barnyard eggs. Public reactions can also affect legislative regulation of production systems.

One way of gauging potential consumer reactions is through the use of consumer surveys. The potential use of bST in milk production is the livestock technology that has been most researched with consumers in the late 1980s and early 1990s. The general conclusion was that this technology was not welcome and its introduction would lead to dramatic hostile consumer reactions with significant effects on milk prices and farmer incomes. These conclusions have been readily accepted by the media, retailers, processors and farmers. However, the construction, execution and interpretation of these surveys need to be understood before such conclusions are drawn.

Ideally the best way to gauge consumer response would be to place the product in the market and measure uptake at various prices and presentations. This is not possible with production techniques that have not been authorised. One of the most significant problems with consumer surveys is that as soon as the subject under review is introduced, the concentration of the interviewee on the subject becomes atypical.

#### *Consumer awareness*

In a number of surveys conducted in North America and Europe, between one and five percent of respondents were aware of bST. Even though 95-99% of respondents were ignorant of the existence of the technology, their attitudes to bST were canvassed. Their stated responses to bST should have been regarded as invalid because their behaviour is unlikely to be affected by the introduction of a technology of which they are unaware.

In asking respondents about a technology of which they are unaware there are difficulties in introducing the technology in neutral terms. Surveys involving bST rarely avoided using the term "hormone" which is alarming to most people. Mention of yield increases has little relevance to consumers. Sophisticated respondents may have concerns about implications for animal welfare and costs of surplus production. None of the bST consumer surveys appeared to suggest any potential benefits to consumers through lower cost or improved product characteristics. It is not surprising that as the presentation introduced potential hazards and unfamiliar implications without any compensating benefits, consumers expressed risk-averse responses of antagonism to the technology.

#### *Response option bias*

A serious flaw in the majority of the ten surveys for which the questionnaires were available was a negative bias in the response options given to interviewees (Bent and Buckwell, 1993). Two of the ten surveys had symmetrical response options, i.e., the response options to a question such as "how would your consumption change if this technology were introduced?" were symmetrically distributed around the neutral "no change" so that "significant decrease" and "slight decrease" were matched by positive responses of similar and opposite magnitude "significant increase" and "slight increase". Four of the ten surveys had a negative bias in the response options and the remaining four surveys had no positive response options at all. An adverse average response from these surveys was statistically inevitable.

#### *Interpretation of results*

Even when the problems mentioned above have been overcome in consumer surveys, results need careful interpretation. There are numerous examples of inconsistency between the stated intentions of consumers and their actions. Consumers in one of the surveys on bST were asked about attitudes to other production techniques. Twenty-five percent said they would avoid milk from cows that were kept in yards and 38% would

avoid milk from cows feed on silage made with silage additives. Both these techniques are in widespread use. In the same survey 45% of consumers would avoid milk from cows treated with bST. This comparison puts headlines of "roughly half of consumers would avoid milk produced with bST" into context.

These results raise two of the problems of labelling milk according to production technique. The first problem is what should and should not be on the labels, and who decides? Should the label include details of dairy cow nutrition and housing? The second problem is verification that the labelling is accurate. Such controls would carry a significant cost and are unlikely to be comprehensive. For example, supplementary bST is virtually indistinguishable from naturally secreted bST. Complex labels and incomplete verification will reduce, not enhance, consumer confidence.

The second point to note when interpreting consumer reactions to food issues is that these reactions are usually transient. Consumption of various foods has frequently returned to pre-scare levels in a relatively short time. As each food scare and health concern emerges, the importance of previous problems appears to diminish.

Finally, in these surveys respondents were asked about consumption of liquid milk. However only 20% of milk is consumed in liquid form in Europe. Milk production technology is less of an issue with other dairy products. Similarly reactions to changing meat production technology are likely to be greatest with respect to fresh cuts and more diluted with respect to processed products. Extrapolation of results obtained in surveys about fresh milk and meat to processed products is difficult to justify.

#### *Corporate commercial opportunities*

The fourth issue that must be addressed in the commercialisation of new techniques is the recognition of commercial potential by an industrial corporation. A company needs to know that the new technique has a reasonable chance of success in the above three areas and can be sold in sufficient quantities in markets that can pay an appropriate price in hard currency. However, companies do invest in the development of new techniques without this prior evaluation. The company will also require an effective system of patents, protection of intellectual property rights and royalties to guarantee a sufficiently attractive return on research and development costs.

**Table 7. Assumed changes in UK pig production in response to three different types of growth enhancers.**

	In-feed growth promoter	In-feed $\beta$ -agonist	Implanted/injected hormone
Daily live-weight gain	+10% <sup>1</sup> +5% <sup>2</sup>	+5% <sup>4</sup>	+15% <sup>4</sup>
Feed conversion ratio	-5% <sup>1</sup> -3% <sup>2</sup>	-8% <sup>4</sup>	-15% <sup>4</sup>
Mortality	-20% <sup>1</sup> -15% <sup>2</sup> -10% <sup>3</sup>	--	--
Veterinary costs	-40% <sup>1</sup> -20% <sup>2</sup> -10% <sup>3</sup>	--	--
Feed prices (including enhancer)	+1%	+2%	+2%
Treatment cost/pig	--	--	\$0.75/week
Treatment labour	--	--	150 pigs/h daily
Meat percentage	--	+5% points	+5% points
Killing out percentage	--	--	-1% point

Response at different live-weights: <sup>1</sup>up to 25kg; <sup>2</sup>25-45kg; <sup>3</sup>45-68kg; <sup>4</sup>45-68kg for porkers, 68-85kg for cutters (institutional catering and processing), 68-92kg for baconers.

### Effects of productivity enhancers in the UK pig sector

This section uses a comparison of the potential impact of three different groups of productivity enhancers on UK pig production to illustrate the economic impacts that can be assessed using relatively simple budgeting models based on derived growth and carcass composition functions.

The first group of enhancers are the classical, in-feed, anti-microbial feed additives. The second group may be considered to represent  $\beta$ -agonists as they are administered in the feed in the latter stages of finishing, are relatively cheap and have an effect on carcass characteristics. The third group are similar to growth hormone requiring injection or implantation in the latter stages of finishing. They are more costly than the other two groups, and have an effect on carcass characteristics.

The assumptions about changes in live-weight gain, feed conversion efficiency, feed costs, mortality, and carcass characteristics are given in Table 7. These responses will vary depending on numerous factors including significant unknown factors such as approved dosage levels and product formulation. If responses to specific products are greater than the responses assumed here, then the economic impact is likely to be greater.

#### Incremental effects

A useful first stage in the analysis of the potential impact of these substances is to examine the effect of each of the responses in isolation. From this analysis, the significance of a response emerges, both in terms of magnitude and with respect to the specific cost items that are affected. This approach is particularly useful in *ex-ante* studies where there may be considerable uncertainty over likely levels of response, as it helps to filter out areas where detailed sensitivity analysis would be redundant.

The incremental effect of the various changes in performance are given in Table 8. From these results it can be seen that, where growth promoters are used, a relatively large reduction in young piglet mortality has a significant benefit, whilst later reductions in mortality will only increase the benefit by a small amount. When all the benefits and costs are considered, this represents about half of the net benefit of using growth promoters.

**Table 8. Effects of incremental performance changes on net margins from growth enhancers in UK pig production systems (converted from £ sterling @ AUS \$2.1/£).**

	In-feed growth promoter	In-feed $\beta$ -agonist	Implanted/ injected hormone	
Daily live-weight gain	+82¢ <sup>1</sup> +10¢ <sup>2</sup>	+92¢ <sup>4</sup>	+19¢	+44¢
Feed conversion ratio	+92¢ <sup>1</sup> +38¢ <sup>2</sup>	+132¢ <sup>4</sup>	+172¢	+326¢
Mortality	+155¢ <sup>1</sup> +8¢ <sup>2</sup> +6¢ <sup>3</sup>	+172¢ <sup>5</sup>	--	--
Veterinary costs	+4¢ <sup>1</sup> +2¢ <sup>2</sup> +0¢ <sup>3</sup>	+8¢ <sup>5</sup>	--	--
Feed prices (including enhancer)	-32¢	-42¢	-42¢	
Treatment cost per pig	--	--	--	-353¢
Treatment labour	--	--	--	-281¢
Meat percentage	--	+802¢	--	+802¢
Killing out percentage	--	--	--	-97¢

Effect of response at different live-weights: <sup>1</sup>up to 25kg; <sup>2</sup>25-45kg; <sup>3</sup>45-68kg; <sup>4</sup>up to 45kg; <sup>5</sup>up to 68 kg.

With all three enhancers, improvements in daily live-weight gain produce benefits as a result of fixed cost reductions per pig. There are additional benefits to be obtained, which have not been shown, through increases in number of pigs per year finished per pig place. This benefit will be highly dependant on the margins being obtained. A 5% increase where margins are AUS \$10 per pig will be worth an additional 90 cents per pig-place per annum.

For both the  $\beta$ -agonists and hormone treatments, the major benefits are through improvements in carcass values, though savings in feed costs are not insubstantial. For hormone treatment, the costs of the treatment and labour for administration are likely to greatly offset these benefits.

The magnitude of these benefits may appear to be quite trivial. Two points should be considered. First, although the value of a UK pig will range between AUS \$110 and AUS \$220 depending on weight, quality and prevailing market conditions, the net margin per pig over the last five years has typically been between AUS \$0 and AUS \$9 per pig.

Second, even relatively small benefits per pig become important when aggregated across national production. A benefit of AUS \$3 per pig on annual UK production amounts to roughly AUS \$45 million per annum for this one item of technology. By comparison, the UK pigmeat trade deficit for 1988-1993 averaged about AUS \$420 million per annum.

From this incremental analysis it can be seen that the classical growth promoters are cost saving, with no effect on output value. Both  $\beta$ -agonists and hormones do have a significant effect on output values, but differ in their effects on costs. The  $\beta$ -agonists are relatively cheap to manufacture, and, once competition is established, may be expected to be a fairly low-cost ingredient. Administration to animals in feed would keep costs down. Thus slight increases in cost per kilogram of feed are more than covered by savings in total feed consumed. In contrast, hormones are likely to be more costly to produce and will involve significant costs of administration. These additional costs are likely to be greater than the reductions in cost attributable to increased throughput or improved feed efficiency. Thus, with  $\beta$ -agonist treatment there will be costs savings and output increases, whilst with hormone treatment, the net benefit is reliant on output value increases (because of premiums for improved carcass characteristics) being sufficient to cover increases in net costs. As discussed above, it is doubtful that these premiums would be maintained in the event of these techniques being available.

#### *Effects on production systems*

Whilst the effects of productivity enhancers on average production are of interest, particularly in identifying the financially important attributes, little insight is provided into the distribution of benefits among different systems of production or farm size or type. An insight into the distribution of benefits is critical in estimating potential markets and likely adoption patterns. This has proved to be an important stage in these studies, as segmentation of production usually identifies niches that would be willing to pay higher prices, or, with a given price, could be expected to be early adopters. It is possible to plot product price against potential market volume, and, given the manufacturers cost of production, estimate a price that would maximise returns to the manufacturer.

The UK pig finishing sector can be segmented in a number of ways. The principle division depends on finishing weight with porkers, cutters (used for institutional catering and processing) and baconers being recognised as distinct groups. Further sub-division is possible according to gender (male-entire, male-castrate or female), feeding system (restricted intake or *ad libitum*) and farm size. For the purpose of this paper, differential effects on porkers, cutters and baconers are given in Table 9.

The differences in effects of the classical growth promoters between systems is minimal. As most of the benefits occur in the earliest weeks of a pig's life, they are common to all three systems. Differences among systems are more pronounced with  $\beta$ -agonists and hormones. With both these enhancers, the changes in carcass characteristics are worth more as finishing weights increase. Effects on cost of treatment are variable and depend on the length of period of treatment. The results would seem to suggest that hormone treatment should commence at a higher weight for baconers than for cutters.

**Table 9** Effects of in-feed growth promoters,  $\beta$ -agonists and hormones on performance of three UK production systems (converted from £ sterling @ AUS \$2.1/£).

		In-feed growth promoter	In-feed $\beta$ -agonist	Implanted/ injected hormone
Porker (68kg)	Output	\$0.00	+ \$5.23	+ \$5.36
	Costs	- \$3.53	- \$1.64	+ \$3.76
	Net margin	+ \$3.53	+ \$6.89	+ \$1.60
	Throughput	+ 5.8%	+ 1.7%	+ 3.8%
Cutter (85kg)	Output	\$0.00	+ \$6.66	+ \$6.36
	Costs	- \$3.57	- \$1.53	+ \$2.88
	Net margin	+ \$3.57	+ \$8.17	+ \$3.48
	Throughput	+ 4.8%	+ 1.0%	+ 2.4%
Baconer (92kg)	Output	\$0.00	+ \$7.22	+ \$6.80
	Costs	- \$3.59	- \$2.02	+ \$5.73
	Net margin	+ \$3.59	+ \$9.24	+ \$1.07
	Throughput	+ 4.5%	+ 1.5%	+ 2.5%

### Other novel enhancers

In this section some of the issues that are likely to affect the development and uptake of a selection of the novel techniques presented in this symposium are discussed.

#### *Genetic engineering*

The use of genetic engineering described by McCauley *et al.* (1995) clearly has several technical problems to overcome. Other issues to be addressed include the ownership of genetic material, the persistence of traits between generations and the ethics of transgenics (particularly where human genetic material is involved).

#### *Immunomodulation*

In contrast, the techniques of immunomodulation are likely to be less controversial and relatively attractive because of the ease of treatment. Modulation of cholecystokinin appears to have advantages of improved feed efficiency and growth rate. In many countries, increased finishing weights have little economic benefit. Changes in carcass composition or in the range of carcass weights and grades are crucial. Thus the wide variation in response is of concern and is likely to be particularly important with castrates on restricted diets. Any adjustments in feed composition may negate the observed benefits.

Attempts to date at immunomodulation of somatostatin in growing pigs appear to have no commercial application. Immunisation of pregnant gilts, however, appears to offer scope for further economic evaluation. Frequently the benefits of changes in growth patterns in the earlier stages of a pig's life extend beyond the immediate treatment period.

#### *pST and $\beta$ -agonists*

Economic analysis of some of the applications of pST and  $\beta$ -agonists described by Dunshea and Walton (1995) are discussed in the previous section. These trials highlight the need for adequate levels of feed of appropriate quality.



### IGF-I

The use of insulin-like growth factor (IGFs) appears to have negative effects in adequately fed finisher pigs (Dunshea and Walton, 1995). Effects on sucking pigs of LR<sup>3</sup>IGF-I appear to have potential economic benefits. Changes in average daily gain are evident, but these are over a relatively short period and will have little direct value. An extra 250-300 g body-weight at four weeks of age is unlikely to allow a significant reduction in weaning age which could increase litters per sow per year. However the need to foster a proportion of piglets for a further week may be removed, allowing a small reduction in farrowing interval for the foster sow.

It is more likely that the average weaning weight would be increased with this technique. Stronger weaned piglets, with improved viscera and gut development are likely to demonstrate improved performance throughout the growing and finishing periods. This technique will have significant commercial potential if there is also a consequent decrease in the variability of pig growth.

Higher weaning weights and improved immunity are also likely to lead to significant reductions in piglet mortality both before and after weaning. The economic benefits of reduced mortality are dramatically illustrated in the previous section.

Although LR<sup>3</sup>IGF-I is orally active in these piglets, an appropriate method of administration will need to be devised. Incorporation into feed is the most cost-effective method of administration but is unlikely to be practicable with animals of this age

### Conclusions

There are several stages from the identification of a novel growth enhancer to its commercial adoption. Evaluation of the potential success of a new technology at each stage is important in determining the likely benefits and is vital to *ex-ante* cost: benefit analysis. The impacts of political decision making, user perceptions, consumer reactions and market adjustments significantly complicate economic evaluation.

The economic benefits of growth enhancers are varied. Whilst feed efficiency, daily gain and carcass composition are extremely important determinants of profitability, reductions in mortality and the variability of performance also appear to be crucial.

Techniques that improve piglet development and survival, such as immunomodulation of somatostatin in gilts and IGF-I treatment of sucking piglets, would appear to merit more detailed economic analysis.

## SYMPOSIUM CONCLUSIONS

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The most important factor that will change production strategies is the drive to remain viable in an increasingly unpredictable market. There is more pressure on the pig industry today than ever before to improve the efficiency of production. The use of methods that target specific metabolic processes in the pig that enhance growth may lead to significant and exciting changes in management practices.

Dunshea and Walton (1995) described the use of exogenous modifiers and indicated that, at least for pST and  $\beta$ -agonists, enough is known about their modes of action to determine the appropriate doses required and the nutritional regimes required for the greatest effects on growth to be seen. Daily injectable pST, an approved procedure in Australia, clearly increases protein gain in both grower and finisher pigs. The benefits of increased growth rate and feed conversion from using pST must be balanced against the costs of labour for treatment, changes in the diet composition and the pST itself. The net economic advantages of pST depend almost entirely on the premium paid for leaner carcasses. The  $\beta$ -agonists work by increasing protein deposition specifically in skeletal muscle and this may prove to be particularly useful in gilts to increase the mass of lean tissue at market weight or to increase the mass of lean tissue in preparation for pregnancy and lactation. The use of IGF-I in the young pig (neonate) may prove to be an exciting new development and, if the improvement in pig growth is mediated by an increase in visceral and gut development as suggested by Dunshea and Walton (1995), the most significant benefits may be in post-weaning performance. The mode of action of both betaine and chromium picolinate to promote growth is not clear but it appears that they may have potential to improve carcass composition under certain circumstances. An advantage of the in-feed growth metabolic modifiers is a lower cost of administration compared with injectable growth promoters. Where there is a change in carcass composition, it is essential to ensure that eating qualities are not compromised and that any changes in the premiums received for lean meat do not negate the apparent benefits of improving the carcass composition.

McCauley *et al.* (1995) described a number of alternatives to manipulate the endogenous hormone levels in animals. With recent advances in recombinant DNA technology, work has recently focussed on two specific areas where genetic manipulation may be useful to increase pig growth. The first is the introduction of genes for somatotropin, growth hormone releasing factor, or IGF-I, and the second is the interest in identifying genes that may improve milk yield or milk composition to improve piglet growth. Several technical problems need to be overcome before this transgenic technology can be adopted. The lack of ability to control the expression of specific genes poses the biggest problem at present, but other issues such as the ownership of genetic material, the persistence of traits between generations and the ethics of transgenics need to be addressed.

Another approach outlined by McCauley *et al.* (1995) to alter the activity of endogenous hormones is immunomodulation, in which specific immunity is developed against a particular hormone. The techniques of immunomodulation are likely to be less controversial than transgenics and should be relatively attractive because of the ease of treatment.

The two hormones that have received the most attention are cholecystokinin (CCK) and somatostatin (SST). Cholecystokinin has been identified as a satiety signal and, as feed intake is often a limiting factor in pig growth, it has been proposed that reducing the concentration of CCK by active immunization should enhance feed intake and growth. Somatostatin inhibits the release of somatotropin (growth hormone), so immunizing against SST presents a plausible means of increasing pig growth. While improvement in pig growth due to immunisation against either CCK or SST have been demonstrated, the responses have not been consistent. Further work is required to develop successful immunization procedures so that the titres to CCK and SST are adequate. One area of

particular promise, as was the case with IGF-I treatment (Dunshea and Walton, 1995), is with the sucking pig. McCauley *et al.* (1995) described a 20% increase in litter performance in response to immunizing sows against SST, although the mechanisms behind this response remain to be elucidated. Improvements in piglet performance are particularly attractive because changes in the growth patterns in the earlier stages of a pig's life extend beyond the immediate treatment period.

Bent (1995a) outlined the long and often complicated process for the translation of novel laboratory techniques into methods that are adopted and integrated into production systems. The basis of obtaining regulatory approval by legislature has traditionally been the scientific criteria of safety, quality and efficacy, but these are being supplemented and occasionally superseded in many regions by more subjective political considerations of ethics, animal welfare and socio-economic impacts. Once approval has been granted, it is necessary for well-designed scientific experiments to be coupled with on-farm trials to clearly demonstrate to producers that the adoption of new technology can increase profitability, reduce risk or effort, or increase job satisfaction. It is also necessary for processing and retailing sectors of the food industry to accept any changes in the end-products caused by the adoption of growth enhancers. The processing and retailing sector is also responsible for gauging and frequently prompting consumer responses to proposed growth enhancers but, interestingly, the stated responses of consumers are frequently inconsistent and at variance with their behaviour (Bent and Buckwell, 1993).

In evaluating the likely acceptance of novel growth enhancers in a particular country, it is important to recognize the markets that are being targeted, the current structure of the industry and the current limitations to productivity, and the particular political and social environment of that country. It may not be entirely valid to extrapolate from one animal industry to another, or from one country to another.

The pig industry has shown itself to be more willing to adopt new techniques and implement new management strategies than many other animal production enterprises. With the current pressure to improve production efficiency, there is a real need for continued research in 'novel' methods that are both acceptable to consumers and economically feasible. If both of these criteria are met, the pig industry would be expected to make use of new advances in improving the efficiency of growth.

## References

- ADEOLA, O., DARKO, E.A., HE, P. and YOUNG, L.G. (1990). Manipulation of porcine carcass composition by ractopamine. *Journal of Animal Science*. 68:3633-3641.
- ANDERSON, D.B., VEENHUIZEN, E.L., WIATT, W.P., PAXTON, R.E. and YOUNG, S.S. (1987). The effect of dietary protein on nitrogen metabolism, growth performance and carcass composition of finishing pigs fed ractopamine. *Federation Proceedings*. 46:1021
- ASTON, R., COWDEN, W.B. and ADA, G. (1989). Antibody mediated enhancement of hormone activity. *Molecular Immunology*. 25:435-446.
- BALDWIN, B.A., COOPER, T.R. and PARROTT, R.F. (1983). Intravenous cholecystokinin octapeptide in pigs reduces operant responding for food, water, sucrose solution or radiant heat. *Physiology and Behaviour*. 30:399-403.
- BENT, M.J.M. (1993a). Farm enterprise budgets and partial equilibrium models. In "Livestock Productivity Enhancers - an Economic Assessment", pp. 94-122, ed. M.J.M. Bent. (CAB International: Wallingford).
- BENT, M.J.M. (1993b). The European approval procedures for livestock productivity enhancers. In "Livestock Productivity Enhancers - an Economic Assessment", pp. 24-42, ed. M.J.M. Bent (CAB International: Wallingford).
- BENT, M.J.M. (1995a). Commercialisation of novel growth enhancers in pigs - Economic and political considerations. In "Manipulating Pig Production V", pp. 62-74, eds D.P. Hennessy and P.D. Cranwell. (Australasian Pig Science Association: Werribee).
- BENT, M.J.M. (1995b). The use of technical and economic models to assess the impacts of productivity enhancers in European livestock systems, Volumes 1 & 2. PhD Thesis. University of London.
- BENT, M.J.M. and BUCKWELL, A.E. (1993). "The socio-economic impact of bovine somatotropin (bST) - a European review", FBU Occasional Paper No. 20. (Wye College, University of London).
- BOLEMAN, S.L., BOLEMAN, S.J., BIDNER, T.D., WARD, T.L., SOUTHERN, L.L., PIKE, M.M. and PONTIF, J.E. (1994). Effect of chromium tripicolinate (CrPic) on growth, carcass composition, and sensory characteristics of growing-finishing pigs. *Journal of Animal Science*. 72 (Supplement 1):273.
- BOYD, D.R. and BAUMAN, D.E. (1989). Mechanisms of action for somatotropin in growth. In "Animal Growth Regulation", pp. 257-293, eds D.R. Campion, G.J. Hausman and R.J. Martin. (Plenum Publishing: New York).
- BOYD, R.D., BAUMAN, D.E., FOX, D.G. and SCANES, C.G. (1991). Impact of metabolic modifiers on protein accretion and protein and energy requirements of livestock. *Journal of Animal Science*. 69 (Supplement 2):56-75.

- BRACHER-JAKOB, A. and BLUM, J.W. (1990). Effects of a  $\beta$ -adrenergic agonist on growth performance, body composition and nutrient retention in finishing pigs fed normal or low amounts of protein. *Animal Production*. 51:601-611.
- BRAND, A. and ELLERTON, A. (1989). "Report on hormone-treated meat" (Club de Bruxelles, Brussels).
- BREM, G., BREINIG, B., MULLER, M., KRAUBLICH, H. and WINNACKER, E.L. (1988). Production of transgenic pigs and possible application to pig breeding. *Occasional Publications of British Society of Animal Production*. 12:15-19.
- BUCKWELL, A.E. (1993). Assessing economic impact - the optimisation approach. In "Livestock Productivity Enhancers - an Economic Assessment", pp. 78-93, ed. M.J.M. Bent (CAB International: Wallingford).
- BURRIN, D.G., WESTER, T.J., DAVIS, T.A., HEATH, J.P., McAVOY, S. and SKOTTNER, A. (1995). Oral insulin-like growth factor-I (IGF-I) increases small intestinal growth in formula-fed neonatal pigs. *Proceedings of the 5th International IGF-I Symposium*. p. 15.
- BURTON, P. (1993). The use of econometric models in the evaluation of livestock productivity enhancers. In "Livestock Productivity Enhancers - an Economic Assessment", pp. 51-77, ed. M.J.M. Bent (CAB International: Wallingford).
- CADOGAN, D.J., CAMPBELL, R.G., HARRISON, D. and EDWARDS, A.C. (1993). The effects of betaine on the growth performance and carcass characteristics of female pigs. In "Manipulating Pig Production IV", p. 219, ed. E.S. Batterham. (Australasian Pig Science Association: Attwood).
- CAMPBELL, R.G. (1995). Future directions and research needs of the Australian pig industry. In "Manipulating Pig Production V", pp. 1-6, eds D.P. Hennessy and P.D. Cranwell. (Australasian Pig Science Association: Werribee).
- CAMPBELL, R.G., JOHNSON, R.J., TAVERNER, M.R. and KING, R.H. (1991). Interrelationships between exogenous porcine somatotropin (pST) administration and dietary protein and energy intake on protein deposition capacity and energy metabolism of pigs. *Journal of Animal Science*. 69:1522-1531.
- CAMPBELL, R.G., JOHNSON, R.J., KING, R.H. and TAVERNER, M.R. (1990a). Effects of gender and genotype on the response of growing pigs to exogenous administration of porcine growth hormone. *Journal of Animal Science*. 68:2674-2681.
- CAMPBELL, R.G., JOHNSON, R.J., KING, R.H., TAVERNER, M.R. and MEISINGER, D.J. (1990b). Interaction of dietary protein content and exogenous porcine growth hormone administration on protein and lipid accretion rates in growing pigs. *Journal of Animal Science*. 68:3217-3225.
- CAMPBELL, R.G., STEELE, N.C., CAPERNA, T.J., McMURTRY, J.P., SOLOMON, M.B. and MITCHELL, A.D. (1988). Interrelationships between energy intake and exogenous growth hormone administration on the performance, body composition and protein and energy metabolism of growing pigs weighing 25 to 55 kilograms body-weight. *Journal of Animal Science*. 66:1643-1655.
- CAMPBELL, R.G., STEELE, N.C., CAPERNA, T.J., McMURTRY, J.P., SOLOMON, M.B. and MITCHELL, A.D. (1989a). Interrelationships between sex and exogenous porcine growth hormone administration and performance, body composition and protein and fat accretion of growing pigs. *Journal of Animal Science*. 67:177-186.
- CAMPBELL, R.G., STEELE, N.C., CAPERNA, T.J., McMURTRY, J.P., SOLOMON, M.B. and MITCHELL, A.D. (1989b). Effects of exogenous porcine growth hormone administration between 30 and 60 kilograms on the subsequent and overall performance of pigs grown to 90 kilograms. *Journal of Animal Science*. 67:1265-1271.
- CAPERNA, T.J., STEELE, N.C., KOMAREK, D.R., McMURTRY, J.P., ROSEBROUGH, R.W., SOLOMON, M.B. and MITCHELL, A.D. (1990). Influence of dietary protein and recombinant porcine somatotropin administration in young pigs: growth, body consumption and hormone status. *Journal of Animal Science*. 68:4243-4252.
- DUBREUIL, P., BRAZEAU, P. and MORISSET, J. (1994). Effects of GRF with or without a SRIF antiserum on GH, IGF-I, thyroxin, cholecystokinin, gastrin and metabolite concentrations in growing rats. *Growth Regulation*. 4:56-62.
- DUBREUIL, P., PELLETIER, G., PETITCLERC, D., LAPIERRE, H., GAUDREAU, P. and BRAZEAU, P. (1989). Effects of active immunization against somatostatin and serum growth hormone concentration in growing pigs: influence of fasting and repetitive somatotropin injections. *Endocrinology*. 125:1378-1384.
- DUNAISKI, V., DUNSHEA, F.R., CLARKE, I.C., OWENS, P.C. and WALTON, P.E. (1994). Endocrine regulation of growth hormone secretion by IGF-I and IGF-I analogues in pigs". *Proceedings of the Endocrine Society of Australia*. 37:116.
- DUNSHEA, F.R. (1991). Factors affecting efficacy of  $\beta$ -agonists for pigs. *Pig News and Information*. 12:227-231.
- DUNSHEA, F.R. (1993). Effect of metabolism modifiers on lipid metabolism in the pig. *Journal of Animal Science*. 71:1966-1977.
- DUNSHEA, F.R. (1994). Nutrient requirements of pigs treated with metabolic modifiers. *Proceedings of the Nutrition Society of Australia*. 18:103-114.
- DUNSHEA, F.R. and GANNON, N.J. (1995). Nutritional and other factors affecting efficacy of  $\beta$ -agonists in pigs. In "Recent Advances in Animal Nutrition in Australia", pp. 46-52, eds J.B. Rowe and J.V. Nolan. (University of New England: Armidale).
- 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 WALTON, P.E. (1995). Potential of exogenous metabolic modifiers for the pig industry. In "Manipulating Pig Production V", pp. 42-51, eds D.P. Hennessy and P.D. Cranwell. (Australasian Pig Science Association: Werribee).
- DUNSHEA, F.R., EASON, P.J., KING, R.H. and CAMPBELL, R.G. (1993a). Effects of ractopamine, dietary energy and sex on protein and fat deposition in growing swine. *Journal of Animal Science*. 71 (Supplement 1):133.
- DUNSHEA, F.R., KING, R.H. and CAMPBELL, R.G. (1993b). Interrelationships between dietary protein and ractopamine on protein and lipid deposition in finishing gilts. *Journal of Animal Science*. 71:2931-2941.

- DUNSCHEA, F.R., KING, R.H., CAMPBELL, R.G., SAINZ, R.D. and KIM, Y.S. (1993c). Interrelationships between sex and ractopamine on protein and lipid deposition in rapidly growing pigs. *Journal of Animal Science*. 71:2919-2930.
- EBERT, K.M., LOW, M.J., OVERSTROM, E.W., BUONOMO, F.C., BAILE, C.A., ROBERTS, A.L. and GOODMAN, R.H. (1988). A Moloney MLV-rat somatotrophin fusion gene produces biologically active somatotrophin in a transgenic pig. *Molecular Endocrinology*. 2:277-283.
- EBERT, K.M., SMITH, T.E., BUONOMO, F.C., OVERSTROM, E.W. and LOW, M.J. (1990). Porcine growth hormone gene expression from viral promoters in transgenic swine. *Animal Biotechnology*. 1:145-159.
- ETHERTON, T.D., WIGGINS, J.P., EVOCK C.M., CHUNG, C.S., REBHUN, J.F., WALTON, P.E. and STEELE, N.C. (1987). Stimulation of pig growth and performance by porcine growth hormone: Determination of the dose-response relationship. *Journal of Animal Science*. 64:433-443.
- EVANS, G.W. and JOHNSON, P.E. (1980). Growth stimulating effect of picolinic acid added to rat diets. *Proceedings of the Society for Experimental Biology and Medicine*. 165:457-461.
- EVOCK, C.M., ETHERTON, T.D., CHUNG, C.S. and IVY, R.E. (1988). Pituitary porcine growth hormone (pGH) and a recombinant pGH analog stimulate pig growth performance in a similar manner. *Journal of Animal Science*. 66:1928-1941.
- EVOCK-CLOVER, C.M. and STEELE, N.C. (1994). Effects of dietary chromium picolinate (CrP) with or without recombinant porcine somatotropin (rpST) on growth performance and carcass composition of growing/finishing pigs. *Journal of Animal Science*. 72 (Supplement 1):159.
- FADLALLA, A.M., SPENCER, G.S.G. and LISTER, D. (1985). The effect of passive immunization against somatostatin on marker retention in lambs. *Journal of Animal Science*. 61:234-239.
- FALLERT, R.F. and HALLBERG, M.C. (1992). BST and the price of milk and dairy products. In "Bovine Somatotropin and Emerging Issues - An Assessment", ed. M. C. Hallberg (Westview Press: Boulder).
- FARMER, C., DUBREUIL, P., PELLETIER, G., PETITCLERC, D. and BRAZEAU, P. (1990). Active immunization against somatostatin in gestating gilts and the effect of transferred immunity on piglets. *Canadian Journal of Animal Science*. 70:211-218.
- FLORIOT, J.-L., LABLANCHE, P., FAUCHER, H., BENT, M.J.M., COURBOIN, V., GARCIA, A.M. and BUCKWELL, A.E. (1995). "Perspective de développement en France de la somatotropine bovine (bST)". (Institut de Gestion Agro-Alimentaire: Cergy-Pontoise).
- GROPP, J. (1991). The efficacy and mode of action of growth promoters. In "The impact on animal husbandry in the European Community of the use of growth promoters - a report to European Commission", Vol 1, pp. 41-130, eds N.J. Young, M.J.M. Bent and J. Gropp. (CEAS Consultants: Ashford).
- GU, Y., SCHINCKEL, A.P., FORREST, J.C., KUEI, C.H. and WATKINS, L.E. (1991). Effects of ractopamine, genotype and growth phase on finishing performance and carcass value in swine. I. Growth performance and carcass merit. *Journal of Animal Science*. 69:2685-2693.
- HARRELL, R.J., THOMAS, M.J., BOYD, R.D., BAUMAN, D.E., CZERWINSKI, S. and STEELE, N.C. (1994). Ontogenic dependent response to exogenous porcine somatotropin in growing pigs. *Journal of Animal Science*. 72 (Supplement 1):253.
- HENMAN, D. (1995). Use of betaine in pig production. In "Recent Advances in Animal Nutrition in Australia", pp. 43-52, eds J.B. Rowe and J.V. Nolan. (University of New England: Armidale).
- HOLDER, A.T., BLOWS, J.A., ASTON, R. and BATES, P.C. (1988). Monoclonal antibody enhancement of the effects of human growth hormone on growth and body composition in mice. *Journal of Endocrinology*. 117:85-90.
- INGVARTSEN, K.L. and SEJRSEN, K. (1995). Effect of immunization against somatostatin (SS) in cattle: a review of performance, carcass composition and possible mode of action. *Acta Agriculturae Scandinavica Section A - Animal Science*. 45:124-131.
- JAMES, S. and PELL, J.M. (1991). Immunomodulation of human growth hormone-releasing factor. *Journal of Endocrinology*. 129:217.
- JARDIEU, P., CLARK, R., MORTENSEN, D. and DORSHKIND, K. (1994). *In vivo* administration of insulin-like growth factor-I stimulates primary B lymphopoiesis and enhances lymphocyte recovery after bone marrow transplantation. *Journal of Immunology*. 152:4320-4327.
- KANIS, E. (1990). Effect of food intake capacity on production traits in growing pigs with restricted feeding. *Animal Production*. 50:333-341.
- KANIS, E. and DE VRIES, A.G. (1992). Optimisation for feed intake capacity in pigs. *Animal Production*. 55:247-255.
- KRAFT, L.A. and WANG, B.S. (1994). Evaluation of porcine somatotrophin enhancement activity by monoclonal antibodies using insulin tolerance measurements in pigs. *Journal of Animal Science*. 72 (Supplement 1):161.
- KRICK, B.J., BOYD, R.D., RONEKER, K.R., BEERMANN, D.H., BAUMAN, D.E., ROSS, D.A. and MEISINGER, D.J. (1993). Porcine somatotropin affects the dietary lysine requirement and net lysine utilization for growing pigs. *Journal of Nutrition*. 123:1913-1922.
- KRICK, B.J., RONEKER, K.R., BOYD, R.D., BEERMANN, D.H., DAVID, P.J. and MEISINGER, D.J. (1992). Influence of genotype and sex on the response of growing pigs to recombinant porcine somatotropin. *Journal of Animal Science*. 70:3024-3034.
- LEMIEUX, C.M. and WOHLGENANT, M.K. (1989). Ex-ante evaluation of the economic impact of agricultural bio-technology: the case of porcine somatotropin. *American Journal of Agricultural Economics*. 71:903-914.
- LIPKIN, W.J., SCHWIMBECK, P.L. and OLDSTONE, M.B.A. (1988). Antibody to synthetic somatostatin-28 (1-12): immunoreactivity with somatostatin in brain is dependent on orientation of immunizing peptide. *Journal of Histochemistry and Cytochemistry*. 36:447-451.
- MALMLOF, K., CORTOVA, Z., SAXERHALT, H., KARLSSON, E. and SKOTTNER, A. (1994). IGF-I and GH: Metabolic effects during experimentally induced catabolism. *Growth Regulation*. 4 (Supplement 1):51.
- MCCAULEY, I., BILLINGHURST, A., MORGAN, P.O. and WESTBROOK, S.L. (1995). Manipulation of endogenous hormones to increase growth. In "Manipulating Pig Production V", pp. 52-61, eds D.P. Hennessy and P.D. Cranwell. (Australasian Pig Science Association: Werribee).
- McLAUGHLIN, C.L., BAILE, C.A. and BUONOMO, F.C. (1985). Effect of CCK antibodies on food intake and weight gain in Zucker rats. *Physiology and Behaviour*. 34:277-282.
- MERTZ, W. and ROGINSKI, E.E. (1969). Effects of chromium (III) supplementation on growth and survival under stress in rats fed low protein diets. *Journal of Nutrition*. 97:531-536.

- MIHARA, M., KOISHIHARA, Y., FUKUI, K., YASUKAWA, K. and OHSUGI, Y. (1991). Murine anti-human IL-6 monoclonal antibody prolongs the bioactivity of human IL-6 in mice. *Immunology*. 74:55-59.
- 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.
- MOONEY, K.W. and CROMWELL, G.L. (1993). Effects of chromium picolinate on performance, carcass composition and tissue accretion in growing-finishing pigs. *Journal of Animal Science*. 71 (Supplement 1):167.
- MOONEY, K.W. and CROMWELL, G.L. (1994a). Effects of Cr picolinate or Cr chloride on performance, ham composition and tissue accretion rates in growing-finishing pigs. *Journal of Animal Science*. 72 (Supplement 1):273.
- MOONEY, K.W. and CROMWELL, G.L. (1994b). Effects of Cr chloride or Cr picolinate on performance, blood parameters, and ham accretion rates in growing pigs. *Journal of Animal Science*. 72 (Supplement 2):62.
- MORAN, T.H., SAWYER, T.K., SEEB, D.H., AMEGLIO, P.J., LOMBARD, M.A. and MCHUGH, P.R. (1992). Potent and sustained satiety actions of a cholecystokinin octapeptide analogue. *American Journal of Nutrition*. 55:286-290.
- NATIONAL RESEARCH COUNCIL (1994). "Metabolic modifiers: Effects on the nutrient requirements of food-producing animals" (National Academy Press: Washington).
- OFFICIAL JOURNAL of the EUROPEAN COMMUNITY (1981). Opinion of the European Parliament on hormonal substances, O.J. C50 p. 87.
- OFFICIAL JOURNAL of the EUROPEAN COMMUNITY (1985). Directive 85/649/EEC, O.J. L382 p. 228.
- OFFICIAL JOURNAL of the EUROPEAN COMMUNITY (1985). Opinion of the Economic and Social Committee on the prohibition of hormonal substances, O.J. C44 p.14.
- OFFICIAL JOURNAL of the EUROPEAN COMMUNITY (1985). Opinion of the European Parliament on the prohibition of hormonal substances, O.J. C288 p.158.
- PAGE, T.G., SOUTHERN, L.L., WARD, T.L. and THOMPSON, J.N.R., D.L. (1993). Effect of chromium picolinate on growth and serum and carcass traits of growing-finishing pigs. *Journal of Animal Science*. 71:656-662.
- PEKAS, J.C. (1985). Animal growth during liberation from appetite suppression. *Growth*. 49:19-27.
- PEKAS, J.C. (1991). Effect of cholecystokinin immunization, enhanced food intake and growth of swine on lean yield and carcass composition. *Journal of Nutrition*. 121:563-567.
- PEKAS, J.C. (1993). Cholecystokinin octapeptide immunization: effect on growth of barrows and gilts. *Journal of Animal Science*. 71:2499-2505.
- PEKAS, J.C. and TROUT, W.E. (1990). Stimulation of food intake and growth of swine by cholecystokinin immunisation. *Growth, Development and Aging*. 54:51-56.
- PELL J.M., JOHNSSON, I.D., PULLAR, R.A., MORRELL, D.J., HART, I.C., HOLDER, A.T. and ASTON, R. (1989). Potentiation of growth hormone activity in sheep using monoclonal antibodies. *Journal of Endocrinology*. 120:R15-R18.
- PEETERS, J.M., HAZENDONK, T.G., BEUVERY, E.C. and TESSER, G.I. (1989). Comparison of four bifunctional reagents for coupling peptides to proteins and the effect of the three moieties on the immunogenicity of the conjugates. *Journal of Immunological Methods*. 120:133-143.
- PETICLERC, D., PELLETIER, G., DUBREUIL, P., LAPIERRE, H., FARMER, C. and BRAZEAU, P. (1988). Effects of active immunization against somatostatin and growth hormone-releasing factor infusion on growth hormone secretion in dairy heifers. *Journal of Animal Science*. 66 (Supplement 1):389.
- PURSEL, V.G., HAMMER, R.E., BOLT, D.J., PALMITER, R.D. and BRINSTER, R.L. (1990). Integration, expression and germline transmission of growth-related genes. *Journal of Reproduction and Fertility Supplement*. 41:77-87.
- PURSEL, V.G., MILLER, K.F., BOLT, D.J., PINKERT, C.A., HAMMER, R.E., PALMITER, R.D. and BRINSTER, R.L. (1989a). Insertion of growth hormone genes into pig embryos. In "Biotechnology of Growth Regulation", pp. 181-188, eds R.B. Heap, C.G. Prosser, G.E. Lamming. (Butterworths: London).
- PURSEL, V.G., PINKERT, C.A., MILLER, K.F., BOLT, D.J., CAMPBELL, R.G., PALMITER, R.D., BRINSTER, R.L. and HAMMER, R.E. (1989b). Genetic engineering of livestock. *Science*. 244:1281-1288.
- PURSEL, V.G., REXROAD, C.E., BOLT, I.D.J., MILLER, K.F., WALL, R.J., HAMMER, R.E., PINKERT, C.A., PALMITER, R.D., PALMITER, R.L. and BRINSTER, R.L. (1987). Progress on gene transfer in farm animals. *Veterinary Immunology and Immunopathology*. 17:303-312.
- REEDS, P.J. and MERSMANN, H.J. (1991). Protein and energy requirements of animals treated with beta adrenergic agonists: A discussion. *Journal of Animal Science*. 69:1532-1550.
- SAUNDERSON, C.L. and MACKINLAY, J.H. (1990). Changes in body-weight, composition and hepatic enzyme activities in response to dietary methionine, betaine and choline levels in growing chicks. *British Journal of Nutrition*. 63:339-349.
- SCHOKNECHT, P.A., EBNER, S., SKOTTNER, A., BURRIN, D.G., DAVIS, T.A. and POND, W.G. (1993). Exogenous IGF-I increased early neonatal weight gain in progeny of protein-restricted sows. *Journal of Animal Science*. 71 (Supplement 1):134.
- SHANAHAN, C.M., RIGBY, N.B., MURRAY, J.D., MARSHALL, J.T., TOWNROW, C.A., NANCARROW, C.D. and WARD, K.A. (1989). Regulation of expression of a sheep metallothionein 1a-sheep growth hormone fusion gene in transgenic mice. *Molecular and Cellular Biology*. 9:5473-5479.
- SMITH, II, J.W., OWEN, K.Q., NELSEN, J.L., GOODBAND, M.D., TOKACH, M.D., LOHRMANN, T.L. and BLUM, S.A. (1994). The effects of dietary carnitine, betaine and chromium nicotinate supplementation on growth and carcass characteristics in growing-finishing pigs. *Journal of Animal Science*. 72 (Supplement 1):274.
- SPENCER, G.S.G. (1992). Immunization against cholecystokinin decreases appetite in lambs. *Journal of Animal Science*. 70:3820-3824.
- SPENCER, G.S.G. and GARSSEN, G. J. (1983). A novel approach to growth promotion using auto-immunisation against somatostatin. I. Effects on growth and hormone levels in lambs. *Livestock Production Science*. 10:25-37.
- STEELE, N.C. and ROSEBROUGH, R.W. (1979). Trivalent chromium and nicotinic acid supplementation for the turkey poul. *Poultry Science*. 58:983-984.
- STEELE, N.C., ALTHEN, T.G. and FROBISH, L.T. (1977). Biological activity of glucose tolerance factor in swine. *Journal of Animal Science*. 45:1341-1345.

- STEWART, C.E.H., BATES, P.C., CALDER, T.A., WOODALL, S.M. and PELL, J.M. (1993). Potentiation of Insulin-like growth factor-I (IGF-I) activity by an antibody: supportive evidence for enhancement of IGF-I bioavailability *in vivo* by IGF binding proteins. *Endocrinology*. **133**:1462-1465.
- SUN, Y.X., DRANE, G.L., CURREY, S.D., LEHNER, N.D., GOODEN, J.M., HOSKINSON, R.M., WYNN P.C. and McDOWELL, G.H. (1990a). Immunization against somatotrophin release inhibiting factor improves digestibility of food, growth and wool production of crossbred lambs. *Australian Journal of Agricultural Research*. **41**:401-411.
- SUN, Y.X., SINCLAIR S.E., WYNN, P.C. and McDOWELL G.H. (1990b). Immunization against somatotrophin release inhibiting factor increases milk yield in ewes. *Australian Journal of Agricultural Research*. **41**:393-400.
- TERRY, L.C. and MARTIN, J.B. (1981). The effects of lateral hypothalamic-medial forebrain stimulation and somatostatin antiserum on pulsatile growth hormone secretion in freely behaving rats: evidence for a dual regulatory mechanism. *Endocrinology*. **109**:622-627.
- TROUT, W.E., PEKAS, J.C. and SCHANBACHER, B.D. (1989). Immune, growth and carcass responses of ram lambs to active immunisation against desulfated cholecystokinin (CCK-8). *Journal of Animal Science*. **67**:2709-2714.
- TURMAN, E.J. and ANDREWS, F.N. (1955). Some effects of purified anterior pituitary growth hormone on swine. *Journal of Animal Science*. **14**:7-18.
- van der HEL, W., PARMENTIER, H.K., HOLE, N.J.K., JAMES, S., BRANDSMA, H.A., FENTENER van VLISSINGEN, J.M., NIEUWLAND, M.G.B. and JOLING, P. (1994). Effect of recombinant porcine somatotrophin and monoclonal antibody directed to ovine somatotrophic hormone on nitrogen retention and immune parameters in pigs. *Journal of Animal Science*. **72**:2820-2827.
- van HEUGTEN, E. and SPEARS, J.W. (1994). Immune response and growth of stressed weanling pigs supplemented with organic or inorganic forms of chromium. *Journal of Animal Science*. **72** (Supplement 1):274.
- van OERS, J.W.A.M. and TILDERS, F.J.H. (1991). Antibodies in passive immunization studies: characteristics and consequences. *Endocrinology*. **128**:496-503.
- VERSTEGEN, M.W.A., van der HEL, W., HENKEN, A.M., HUISMAN, J., KANIS, E., van der WAL, P. and van WEERDEN, E.J. (1990). Effect of exogenous porcine somatotropin administration on nitrogen and energy metabolism in three genotypes of pigs. *Journal of Animal Science*. **68**:1008-1016.
- WALL, R.J., PURSEL, V.G., SHAMAY, A., McKNIGHT, R.A., PITTIUS, C.W. and HENNINGHAUSEN, L. (1991). High-level synthesis of a heterologous milk protein in the mammary glands of transgenic swine. *Proceedings of the National Academy of Sciences USA*. **88**:1696-1700.
- WALTON, P.E., OWENS, P.C., KNOWLES, S.E., DUNAISKI, V. and DUNSHEA, F.R. (1994). Metabolic actions and roles of IGF-I analogues in growth regulation in pigs. *Journal of Animal Science*. **72** (Supplement 1):253.
- WANG, B.S., SZEWCZYK, E., SHIEH, H.M. and HART, I.C. (1990). Potentiation of the growth-promoting activity of porcine growth hormone (pGH) with an antibody generated in rabbits to the peptide sequence pGH110-118. *Journal of Endocrinology*. **127**:481-485.
- WANGSNESS, P.E., MARTIN, R.J. and GAHAGAN, J.H. (1977). Insulin and growth hormone in lean and obese pigs. *American Journal of Physiology*. **233**:E104-E108.
- 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.
- WESTBROOK, S.L., ALI, A.M. and McDOWELL, G.H. (1994). Passively-acquired antibodies to somatotropin release inhibiting factor (SRIF) increase appetite and growth of milk-fed lambs. *Australian Journal of Agricultural Research*. **45**:293-302.
- WESTBROOK, S.L., CHANDLER, K.D. and McDOWELL, G.H. (1993). Immunization of pregnant ewes against somatotropin release inhibiting factor increases growth of twin lambs. *Australian Journal of Agricultural Research*. **44**:229-238.
- WIDDOWSON, E.M., COLOMBO, V.E. and ARTAVAVIS, C.A. (1976). Changes in the organs of pigs in response to feeding for the first 24 hours after birth. II. The digestive tract. *Biology of the Neonate*. **28**:272-281.
- WILLIAMS, I.H. (1995). Sows' milk as a major nutrient source before weaning. In "Manipulating Pig Production V", pp. 107-113, eds D.P. Hennessy and P.D. Cranwell. (Australasian Pig Science Association: Werribee).
- WRAY-CAHEN, D., ROSS, D.A., BAUMAN, D.E. and BOYD, R.D. (1991). Metabolic effects of porcine somatotropin: nitrogen and energy balance and characterization of the temporal pattern of blood metabolites and hormones. *Journal of Animal Science*. **69**:1503-1514.
- XU, R.-J., MELLOR, D.J., BIRTLES, M.J., BREIER, B.H. and GLUCKMAN, P.D. (1994). Effects of oral IGF-I or IGF-II on digestive organ growth in newborn pigs. *Biology of the Neonate*. **66**:280-287.
- YEN, J. T., NIENABER, J.A., KLINDT, J. and CROUSE, J.D. (1991). Effect of ractopamine on growth, carcass traits, and fasting heat production of U.S. contemporary crossbred and Chinese Meishan pure- and crossbred pigs. *Journal of Animal Science*. **69**:4810-4822.

## RELATIVE AVERSIVENESS OF A NEW INJECTION PROCEDURE FOR PIGS

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The aversiveness of a new injection procedure, developed for the daily administration of somatotropin to pigs, was examined over a 3-week period in finisher pigs. The injection treatment, "Inj" involved daily intra-muscular (im) injections of saline via a 13 mm, 16 g needle and a gas injection gun. The aversiveness was assessed on the basis of: i) the behavioural responses following imposition of the treatment by the experimenter on day 22 as assessed from video records; ii) the behavioural response to an unfamiliar experimenter in a standardized arena test on day 23; and iii) the cortisol concentrations in samples collected 10 min post-treatment and 60 min after a challenge of adrenocorticotrophic hormone (ACTH; Synacthen, Ciba-Geigy, 50 IU im) as indicators of acute and chronic stress responses. The relative magnitude of these responses were compared with a positive control ("Pos" treatment, involving rewarding components such as patting and stroking of pigs that approached), a negative control ("Neg" treatment, involving aversive components such as approach by humans and an electric shock with a commercial battery-operated goad if individual pigs failed to avoid the approaching human) and a neutral control ("Con" treatment, with human contact similar to that which occurs during routine husbandry). The treatments were imposed daily on a total of 120 individual pigs housed in 24 groups of 5 male pigs (average live-weight of 63.1 kg).

**Table 1. Effects of treatment on the behavioural responses of pigs in treatment and in a standardized human test and the cortisol concentrations (nmol/L of pigs to treatment and to an ACTH challenge (n=30).**

Parameter	Inj	Con	Neg	Pos	SEM
Withdrawal distance (cm) <sup>1</sup>	2.84 <sup>y</sup>	1.24 <sup>x</sup>	5.27 <sup>z</sup>	0.81 <sup>x</sup>	0.425
Proportion facing human	0.57 <sup>y</sup>	0.88 <sup>z</sup>	0.06 <sup>x</sup>	0.80 <sup>z</sup>	0.073
<b>In arena:</b>					
Time to 0.5 m of human (s)	90.0 <sup>bcy</sup>	68.7 <sup>by</sup>	103.6 <sup>cy</sup>	33.1 <sup>ax</sup>	14.31
Time within 0.5 m of human (s)	43.8 <sup>y</sup>	49.1 <sup>y</sup>	17.5 <sup>x</sup>	50.4 <sup>y</sup>	7.60
Time to interact (s)	111.5 <sup>c</sup>	79.6 <sup>bxy</sup>	145.3 <sup>dz</sup>	52.3 <sup>ax</sup>	14.51
Number of interactions <sup>1</sup>	3.2 <sup>y</sup>	3.2 <sup>y</sup>	1.8 <sup>x</sup>	3.8 <sup>y</sup>	0.35
Cortisol response	33.5 <sup>x</sup>	24.9 <sup>x</sup>	76.7 <sup>y</sup>	33.8 <sup>x</sup>	6.86
Cortisol response to ACTH	235.9	222.3	249.2	238.9	14.15

<sup>1</sup>log<sub>e</sub>(x+1) transformed data; <sup>abcd,xyz</sup> denote significant differences P<0.05 and P<0.01.

The behavioural responses in the Inj treatment were intermediate to those of pigs in the Neg treatment and in the Con and Pos treatments and the cortisol response of pigs in the Inj treatment was similar to that of pigs in the Con and Pos treatments but lower (P<0.05) than in the Neg treatment. In the arena test the Inj treatment was similar to the Con treatment for three of the four approach parameters and different (P<0.05) than those in the Neg treatment for three of the four variables. There were no treatment effects of ACTH (P>0.05). Although, there was evidence that the pigs in the Negative treatment may have experienced a chronic physiological stress response with a significant (P<0.05) depression of growth rate, there was neither physiological or production evidence that the "Inj" treatment adversely affected the long-term stress physiology of the pigs. In conclusion, the daily imposition of this new injection procedure over a 3-week period was, at the worst, moderately aversive to pigs and the welfare of these pigs was similar to that of pigs receiving minimal human contact as occurs in the routine husbandry of growing pigs.



## ABSORPTION OF INSULIN-LIKE GROWTH FACTOR I IN NEONATAL PIGS IS INDEPENDENT OF GUT CLOSURE

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High levels of insulin-like growth factor I (IGF-I) have been found in porcine milk and may play a role in regulating postnatal development in neonatal pigs (Xu, 1995). The objective of this study was to determine if milk-borne IGF-I can be absorbed intact by newborn pigs, and if so whether absorption is independent of gut closure.

Three newborn unsuckled pigs (one male and two females) and three 3-day-old naturally suckled pigs (two males and one female) were obtained from two litters of Landrace  $\times$  Large White pigs. Each animal was given, via an orogastric tube, 10 ml porcine colostrum per kg body-weight containing 0.3  $\mu\text{g/ml}$   $^{125}\text{I}$  labelled IGF-I (specific activity 0.06 mCi/ $\mu\text{g}$ ) and 6.5 mg/ml fluorescent isothiocyanate dextran (FITC-D, a macromolecular marker of about 71 kd) as an index of gut closure. Blood samples were taken at 0, 1, 2, 3 and 4 h after orogastric delivery of the mixture for the measurement of plasma levels of total radioactivity, acid precipitable radioactivity and FITC-D concentration. Intact  $^{125}\text{I}$ -IGF-I in the plasma samples were identified by chromatography.

Total plasma and acid precipitable radioactivity in both newborn and 3-day-old pigs increased significantly ( $P < 0.05$ ) within the first hour. In contrast significant absorption of FITC-D was only observed in newborn pigs (Figure 1). Chromatographic analysis revealed that up to 20% of plasma total radioactivity is intact  $^{125}\text{I}$ -IGF-I in newborn pigs and up to 10% in 3-day-old pigs. Most of the  $^{125}\text{I}$ -IGF-I found in the plasma was associated with IGF binding proteins.

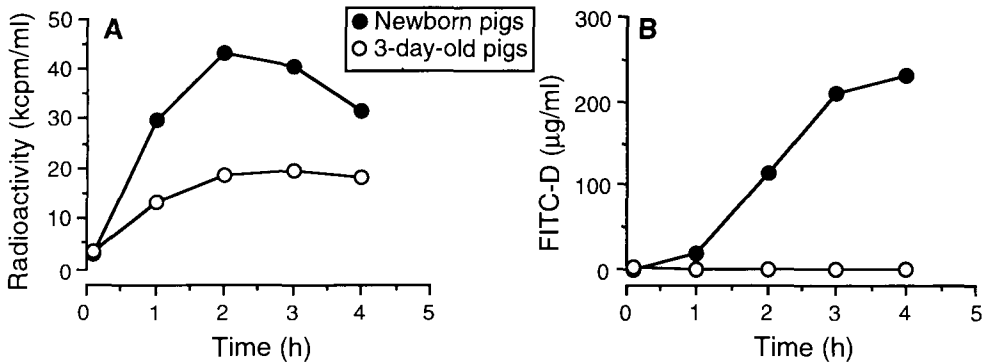


Figure 1. Plasma levels of acid precipitable radioactivity (A) and FITC-D (B) in newborn and 3-day-old pigs following orogastric administration of iodinated IGF-I and FITC-D.

The results show that milk-borne IGF-I can be absorbed by newborn pigs, which is in accordance with our previous finding that orally administered IGFs can stimulate intestinal and pancreatic tissue growth in newborn pigs (Xu *et al.*, 1994). Significant amounts of  $^{125}\text{I}$ -IGF-I but not FITC-D were detected in the plasma of the 3-day-old pigs. Since cessation of FITC-D absorption is an indicator of gut closure, these data suggest that absorption of IGF-I can occur after gut closure in the neonatal pigs.

### References

- XU, R.J. (1995). *Reproduction, Fertility and Development*. (In press).  
 XU, R.J., MELLOR, D.J., BIRTLES, M.J., BREIER, H.B. and GLÜCKMAN, P.D. (1994). *Biology of the Neonate*. 66:280-287.

## EFFECTS OF INTRA-VENOUS ADMINISTRATION OF INSULIN-LIKE GROWTH FACTOR II ON ENERGY METABOLISM

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Tissue synthesis and secretion into blood of insulin-like growth factors, IGF-I and -II, are regulated and influenced by growth hormone, age, nutrition and gender. Blood plasma levels of IGF-I correlate positively with indicators of lean growth and protein deposition in pigs, such as live-weight gain. The same is observed for IGF-II in young pigs, but in older animals the concentration of endogenous IGF-II in blood correlates positively with indicators of fat deposition, such as backfat thickness measurements eg., P2 (Owens *et al.*, 1994). The aim of this study was to compare the effects of IGF-II and IGF-I on energy metabolism.

Recombinant IGF-II polypeptide was produced by expression in *Escherichia coli* of a plasmid containing a gene construct encoding a synthetic precursor of IGF-II. The precursor was purified and cleaved to yield several hundred milligrams of IGF-II. Recombinant IGF-I polypeptide was obtained from GroPep Pty Ltd, Adelaide. Under general anaesthesia, silastic catheters were surgically implanted into each cephalic vein of twelve, well fed, Large White finisher gilts. A minimum of 3 d recovery was permitted before experimentation commenced. Of the 12 gilts, six were fed and six were fasted overnight prior to the treatment with IGFs. One half of each group of six were given a bolus intra-venous injection of IGF-I the other half were given IGF-II.

Fasting reduced the concentrations in blood plasma of glucose by 45% ( $P < 0.001$ ), urea by 41% ( $P < 0.001$ ), insulin by 97% ( $P < 0.001$ ), IGF-I by 30% ( $P < 0.01$ ) and IGF-II by 15% ( $P < 0.02$ ), consistent with a catabolic state. Fasting also increased plasma free fatty acids ten-fold ( $P < 0.001$ ), indicating a significant rate of lipolysis. The IGFs were injected at 46  $\mu\text{g}/\text{kg}$  in fed and 48  $\mu\text{g}/\text{kg}$  in fasted gilts ( $n=3$  per polypeptide growth factor and nutrition group). Both IGFs markedly reduced plasma insulin in fasted gilts ( $P < 0.001$ ) and showed similar, but weaker, hypoinsulinemic actions in fed animals ( $P < 0.025$ ). At the same dose IGF-I, but not IGF-II, reduced blood glucose in both fed and fasted gilts ( $P < 0.001$ ). In fasted, but not in fed animals, both IGF-I and IGF-II altered blood plasma concentrations of free fatty acids ( $P < 0.001$ ). Free fatty acids abruptly declined in the first 15 min after injection of IGFs into fasted gilts, after which they progressively rose to reach levels that were higher than those observed before treatment. Both the initial decrease and subsequent rise in fatty acid levels were greater for IGF-I than for IGF-II ( $P < 0.001$ ).

This study shows that the pharmacological actions of IGF-II are similar, but not identical, to those of IGF-I, and that both have acute metabolic actions similar to those of insulin. The greater potency of IGF-I on indicators of energy metabolism suggest that these effects occur by binding to type-I IGF receptors, for which IGF-I has a slightly greater affinity. The initial fall in plasma free fatty acids in fasted pigs following injection of either IGF-I or IGF-II indicate that both IGFs can pharmacologically inhibit lipolysis. A discrepancy between physiological correlations of endogenous IGFs with growth of lean and fat and the effects of exogenous IGFs on fat metabolism requires further investigation. In summary, intra-venous injection of insulin-like growth factors I and II suppresses insulin and alters metabolism of glucose and lipids. These effects are sensitive to nutrition and metabolic status. This is the first report of actions of IGF-II in pigs.

### References

OWENS, P.C., CAMPBELL, R.G., FRANCIS, G.L. and QUINN, K.J. (1994) *Journal of Animal Science*. 72 (Supplement 1):253.

## $\beta$ -ADRENERGIC RECEPTORS ON PORCINE IMMUNE CELLS

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Pigs reared under commercial conditions perform 15 to 25% below their potential (Black *et al.*, 1994). When exposed to the disease, climate, and social stressors experienced in a commercial environment the porcine immune system can show an impaired function manifesting in increased disease susceptibility and reduced performance. Epinephrine (EP) increases following exposure to stress which subsequently influences cell-mediated immunity via specific  $\beta$ -adrenergic receptors on immune cells. Consequently, this study was designed to elucidate the gene and deduce the amino acid sequence of the porcine  $\beta$ -adrenergic receptor in order to characterise its activation pathway in lymphocytes.

Porcine genomic DNA was obtained from lymphocytes and adrenal cortical cells. This DNA served as a template in a polymerase chain reaction (PCR), using 20-mer oligonucleotide primers (forward: 5'-AAAGCCGGTGCCTCACCTG; reverse: 5'-TGCAGGCTTCTGCTTTTAA) analogous to the published sequence of the 5-prime and 3 prime ends of the hamster  $\beta$ -adrenergic receptor gene (Dohlman *et al.*, 1987). Murine adrenal and lymphocyte DNA was used as a positive control.

Following PCR of pig lymphocyte and adrenal DNA, a single 1200 base pair (bp) band was observed on agarose gels. Employing the same primers and conditions, PCR of murine DNA resulted in a single band at 1300 bp. Both porcine and murine PCR bands were consistent with the  $\beta$ -adrenergic receptor gene sizes for other mammalian species and thus were sequenced. The presence of serine and tyrosine residues in the cytoplasmic domain of each amino acid sequence, deduced from the respective genes for the  $\beta$ -adrenergic receptor, confirms the presence of intra-cellular phosphorylation sites observed in other species (Dohlman *et al.*, 1987). These sites serve as initial activation signals in the cascade to alter cellular activity, which may result in immune cell dysfunction.

Preliminary *in vitro* phosphorylation experiments were conducted on cultured prepubertal female porcine and murine lymphocytes to ascertain the primary activation pathway of the  $\beta$ -adrenergic receptor. These cells ( $2 \times 10^7$ /400 mL) were stimulated with EP (10-100 mM for 0.5-10 min at 23°C). Cell proteins were separated electrophoretically and transferred to a PVDF membrane. Incubation with antibodies specific for phosphoserine and phosphotyrosine identified a major phosphorylated protein at 64 kD, indicative of the  $\beta$ -adrenergic receptor. Epinephrine stimulation of lymphocytes induced a time and dose dependent increase in the phosphorylation state of the 64 kD protein.

As a result of this investigation we have been able to identify the sequence of the porcine immune cell  $\beta$ -adrenergic receptor, and have begun to establish an *in vitro* bioassay to measure the activation state of these receptors. This information will contribute to the development of technologies aimed at preventing the deleterious effects of stress hormones on porcine immune cells.

### References

- BLACK, J.L., DAVIES, G.T. and BRADLEY, L.R. (1994). In "Livestock Production for the 21st Century: Priorities and Research Needs", pp. 229-249, ed. P.A. Thacker. (Annual Meeting of Canadian Society of Animal Science: Saskatchewan).
- DOHLMAN, H.G., BOUVIER, M., BENOVIC, J.L., CARON, M.G. and LEFKOWITZ, R.J. (1987). *The Journal of Biological Chemistry*. 262:14282-14288.

## PRODUCTION AND ENDOCRINE RESPONSES IN PIGS TO ACUTE PRE-NATAL TESTOSTERONE TREATMENTS

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The growth characteristics of female animals are sensitive to pre-natal androgen treatment (Clarke, 1982). Chronic androgen exposure leads to the development of endocrine profiles that are associated with increased rates of body-weight gain and reduced carcass fatness (De Haan *et al.*, 1990). This experiment examined the responses of a number of production and endocrine parameters to single injections of testosterone in late gestation. Two forms of testosterone were investigated to determine the effects of chemical form of the hormone on the duration and elevation of testosterone concentrations in the maternal plasma and the subsequent performance of the progeny.

Twelve Camborough (Pig Improvement Company, PIC) pregnant sows (parity 2+), which had been mated with boars from the PIC terminal sire line, were selected on the basis of weight and mating date and allocated at random into three groups. The treatments consisted of single intra-muscular injections of testosterone (3 mg/kg live-weight) at 98 d post-mating given either as testosterone propionate (TP) or testosterone enanthate (TE). The sows in the third, untreated group acted as controls (C). Blood samples were obtained via jugular veni-puncture at 8, 24, 48, 96 and 168 h after injection for the determination of plasma testosterone and oestradiol concentrations. Additional samples were taken from all sows at intervals from 112 d post-mating to 2 d after farrowing for determination of plasma oestradiol and progesterone concentrations. The progeny were weighed at intervals from birth they were slaughtered at approximately 18 weeks-of-age. Carcass yield and P2 fat depth were obtained at slaughter. Hormone assays were performed at the Victorian Institute of Animal Science, Attwood. Least square analysis of main effects for gender, treatment and their interaction were performed using age at slaughter as a covariate.

**Table 1. Performance of progeny from sows treated with single intra-muscular injections of testosterone (3 mg/kg live-weight) at 98 d post-mating.**

	TE		TP		C		SED
	F <sup>a</sup>	M <sup>b</sup>	F	M	F	M	
Slaughter weight (kg)	66.1	68.2	72.0	70.0	61.0	63.5	2.5
Live-weight gain (g/day)	553	563	618	613	534	544	22.4
Fat depth (mm)	11.2	9.2	9.7	7.9	10.9	10.2	0.69
Carcass yield (%)	80.6	79.1	79.1	78.2	81.0	81.9	1.20

<sup>a</sup>Female; <sup>b</sup> Male

Treatment with TP produced peak concentrations of testosterone in the maternal circulation (46 nmol/L at 15 h) more rapidly ( $P < 0.05$ ) than did treatment with TE (32 nmol/L at 66 h). Androgen treatment in late gestation did not affect the length of gestation or plasma oestradiol or progesterone concentrations. Rates of gain (Table 1) were greater ( $P < 0.05$ ) and dressing percentage lower ( $P < 0.01$ ) in animals exposed to TP relative to C and TE treatments.

The two chemical forms of testosterone, administered as single injections, produced different patterns of testosterone concentration in the maternal circulation which were associated with differences in the growth of the progeny. Testosterone levels in late gestation were elevated by treatment with TP and this appeared to have a greater effect on carcass yield and composition than did TE.

### References

- CLARKE, I.J. (1982). *Oxford Reviews of Reproductive Biology*. 4:101-147.  
 DeHAAN, K.C., BERGER, L.L., BETCHEL, P.J., KESLER, D.J., McKIETH, F.K. and THOMAS, D.L. (1990). *Journal of Animal Science*. 68:4100-4108.

## ENDOCRINE REGULATION OF SOMATOTROPIN SECRETION BY IGF-I AND IGF-I ANALOGUES IN PIGS

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Insulin-like Growth Factor-I (IGF-I) promotes protein and DNA synthesis, inhibits protein breakdown and enhances growth in normal rats (Tomas *et al.*, 1993). A variant of IGF-I, Long R<sup>3</sup>IGF-I (LR<sup>3</sup>IGF-I), that binds poorly to IGF binding proteins (IGFBP) is 10-fold more potent in its anabolic effects than IGF-I *in vitro* (Francis *et al.*, 1992). However, it has recently been found that these analogues act to inhibit growth in finisher pigs (Walton *et al.*, 1994). The objective of the current study is to determine if the negative effects of IGF analogues on growth are associated with a feedback inhibition of somatotropin (pST) secretion or synthesis.

Twenty-four male Large White × Landrace pigs (55 kg body-weight) were fitted with indwelling jugular catheters, allowed 8 d to recover from surgery and then grouped into one of the following six treatments: 1. Vehicle (100 mM acetic acid, 150 mM NaCl); 2. IGF-I; 3. LR<sup>3</sup>IGF-I; 4. pST; 5. IGF-I + pST; 6. LR<sup>3</sup>IGF-I + pST. The IGFs were dissolved in 100 mM acetic acid and infused via Alzet mini-osmotic pumps for 90 h at a rate of 180 µg/kg/d, and pST was dissolved in 150 mM NaCl and administered 4 times daily by sub-cutaneous injections at 30 µg/kg/d. Controls infusions or injections were 100 mM acetic acid or 150 mM NaCl for IGFs and pST respectively. Blood samples were taken at 10 min intervals for 6 h prior to treatment and for 6 h after 4 d of treatment. After 94 h of treatment, all animals were challenged with an intra-venous dose of 1.8 µg/kg growth hormone releasing factor (GHRF), and blood samples were taken for a further 60 min. Plasma pST, insulin, IGFBP-3, IGF-I and LR<sup>3</sup>IGF-I were measured by radioimmunoassay (Walton and Etherton, 1989; Owens *et al.*, 1990), and plasma IGF-II by radioreceptor assay (Owens *et al.*, 1990). The pST profiles were analysed using PULSAR analysis (Merriam and Wachter, 1982). Data were analysed using repeated measures one-way or two-way analyses of variance.

Treatment with LR<sup>3</sup>IGF-I reduced plasma IGFBP-3, IGF-I and insulin concentrations by 50%. Average plasma pST concentration was decreased by 23% while peak areas were reduced by 60% ( $P < 0.05$ ). Co-administration of LR<sup>3</sup>IGF-I with pST decreased plasma IGF-I concentrations by nearly 50% ( $P < 0.05$ ) but had no effect on plasma insulin and IGFBP-3 concentrations. Treatment with IGF-I alone, and in combination with pST affected only plasma IGFBP-3 concentrations, which were increased by 22% and 45% respectively ( $P < 0.05$ ). A non-significant trend was observed for IGF-I and LR<sup>3</sup>IGF-I infusion to produce a greater pST peak in response to a GRF challenge.

It is apparent that the IGF-I analogue, LR<sup>3</sup>IGF-I, suppresses plasma pST levels in finisher pigs, and that this may contribute to the reduced concentrations of plasma IGF-I, IGFBP-3 and insulin as well as reduced growth rates in pigs treated with this peptide. The effect of LR<sup>3</sup>IGF-I treatment on plasma pST concentrations could be due to an inhibition of pST secretion from the pituitary.

### References

- FRANCIS, G.L., ROSS, M., BALLARD, F.J., MILNER, S.J., SENN, C., McNEIL, K.A., WALLACE, J.C., KING, R. and WELLS, J.R.E. (1992). *Journal of Molecular Endocrinology*. 8:213-223.
- MERRIAM, C. and WACHTER, K. (1982). *American Journal of Physiology*. 243:E310-E318.
- OWENS, P.C., JOHNSON, R.J., CAMPBELL, R.G. and BALLARD, F.J. (1990). *Journal of Endocrinology*. 124:269-275.
- TOMAS, F.M., KNOWLES, S.E., CHANDLERS, C.S., FRANCIS, G.L., OWENS, P.C. and BALLARD, F.J. (1993). *Journal of Endocrinology*. 317:413-421.
- WALTON, P.E. and ETHERTON, T.D. (1989). *Journal of Endocrinology*. 120:153-160.
- WALTON, P.E., OWENS, P.C., KNOWLES, S.E., DUNAISKI, V. and DUNSHEA, F.R. (1994). *Journal of Animal Science*. 72 (Supplement 1):253.

## EFFECT OF CONCENTRATION OF SPERMATOZOA ON *IN VITRO* FERTILIZATION OF PIG OOCYTES MATURED *IN VITRO*

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Two major problems with porcine *in vitro* fertilization (IVF) are the high incidence of polyspermic fertilization and the failure of formation of the male pronucleus (PN; Behalova *et al.*, 1993). This study examined the effect of concentration of spermatozoa on the fertilization of *in vitro* matured pig oocytes with ejaculated boar spermatozoa.

Immature oocytes (N=890) collected on 5 d were matured at 39°C (5% CO<sub>2</sub> in air) in a modified tissue culture medium 199 (TCM199) plus 25% fresh porcine follicular fluid, 0.5 mM cysteamine, LH, FSH and oestradiol. After 46 h of culture, the cumulus-expanded (presumed mature) oocytes were washed twice in fertilization medium (modified PVA-TALP plus 25mM bicarbonate) to remove excess cumulus cells and placed in 100 µl droplets of IVF medium under oil (25 oocytes/drop).

The sperm rich fraction of a freshly collected boar ejaculate was extended five-fold in Beltsville thawing solution (Johnson *et al.*, 1988), cooled and stored overnight at 18°C. On the day of IVF, the semen was washed three times (centrifugation at 800 g) and resuspended each time in sperm pre-incubation medium (modified TCM199 + 8.3 mM calcium lactate, 12% fetal bovine serum). The spermatozoa were then pre-incubated (39°C, 5% CO<sub>2</sub> in air) at  $2.4 \times 10^8$ /ml in the same medium for 15 min prior to insemination. Sperm suspension was then added to each fertilization drop so that the final concentration was 1.25, 2.5, 5.0 or  $10.0 \times 10^5$  spermatozoa/ml. The spermatozoa and oocytes were then coincubated at 39°C in 5% CO<sub>2</sub> in air. Eighteen h after insemination, presumptive zygotes were fixed in ethanol:acetic acid:chloroform (6:3:1), stained with 1% aceto-orcein and examined for fertilization (Table 1; all data were analysed by the  $\chi^2$  test).

**Table 1. Effect of concentration of spermatozoa on fertilization of oocytes. The percentage of oocytes penetrated or polyspermic is shown in parentheses.**

Sperm/ml ( $\times 10^5$ )	No. Oocytes			% Oocytes with male PN <sup>1</sup>	% Oocytes Monospermic <sup>2</sup>
	Matured	Penetrated	Polyspermic		
1.25	182	69 (38) <sup>a</sup>	6 (9) <sup>a1</sup>	77 <sup>ab</sup>	35 <sup>a</sup>
2.5	196	128 (65) <sup>b</sup>	26 (13) <sup>a</sup>	85 <sup>b</sup>	57 <sup>c</sup>
5.0	171	142 (83) <sup>c</sup>	42 (30) <sup>b</sup>	87 <sup>b</sup>	59 <sup>c</sup>
10.0	237	204 (86) <sup>c</sup>	94 (46) <sup>c</sup>	68 <sup>a</sup>	46 <sup>b</sup>

<sup>1</sup>As a % of oocytes penetrated. <sup>2</sup>As a % of mature oocytes. Percentages in columns with different superscripts differ ( $P < 0.01$ ).

The proportion of oocytes penetrated and the proportion of polyspermic oocytes increased with increasing concentration of spermatozoa ( $P < 0.001$ ). However, the greatest number of monospermic oocytes was obtained after insemination with  $5.0 \times 10^5$  spermatozoa/ml. The proportion of oocytes with at least one male PN was lower for oocytes inseminated with  $10 \times 10^5$  spermatozoa/ml than for other concentrations of spermatozoa. It appears that the ability of oocytes to decondense penetrated spermatozoa, and develop the male PN, is compromised when more than one spermatozoon is present in the cytoplasm.

This study suggests that the optimal concentration of spermatozoa for *in vitro* fertilization of *in vitro* matured pig oocytes using this system is  $2.5 - 5 \times 10^5$ /ml.

### References

- BEHALOVA, E., PAVLOK, A., MOTLIK, J. and FULKA, J. (1993). *Animal Reproduction Science*. 32:127-133.  
 JOHNSON, L.A., AALBARS, J.G. and GROOTEN, H.J.G. (1988). *Zuchthygiene*. 23: 49-55.

## MORPHOLOGY AND *IN VITRO* FERTILIZING CAPACITY OF BOAR SPERMATOOZOA FOLLOWING SEX SELECTION BY FLOW CYTOMETRY

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Sex selection by flow cytometry involves incubating spermatozoa with a fluorescent DNA binding stain (H33342) for 1 h followed by exposure to a laser, allowing DNA measurement and subsequent sorting of the spermatozoa into 2 populations. This procedure has led to the production of normal piglets of predetermined sex after fertilization *in vivo* (Johnson, 1991). Lower conception rates and litter sizes associated with the sex-sorting technique are possibly as a result of exposure of spermatozoa to detrimental conditions during the sex-sorting process. To date fertilization *in vitro* of sorted spermatozoa has only been performed on *in vivo* matured oocytes. The aim of this study was to (1) investigate the morphology and membrane integrity of spermatozoa by transmission electron microscopy (TEM) following sex-sorting, and (2) examine the ability of putative sex-sorted spermatozoa to fertilize *in vitro* matured pig oocytes.

Semen was collected by the gloved-hand method from 1 boar. The sperm-rich fraction was diluted to a concentration of  $90 \times 10^6$  spermatozoa/ml, stored overnight at 18°C, resuspended to a concentration of  $10 \times 10^6$ /ml, incubated with H33342, at 35°C for 1 h, and held at 30°C until flow cytometric assessment. Unsorted (control) spermatozoa were further diluted to give a concentration of  $1 \times 10^6$ /ml, the approximate concentration after sorting. All dilutions were made with Beltsville thawing solution (Johnson *et al.*, 1988). Sorted and control spermatozoa were processed for TEM according to Simpson *et al.* (1986) and examined for general morphology and membrane integrity using a Zeiss 902 electron microscope. For assessment of the fertilizing capacity of spermatozoa, oocytes were collected from abattoir-sourced ovaries and matured *in vitro*. All culture conditions for *in vitro* maturation and subsequent fertilization were at 39°C in 5% CO<sub>2</sub> in air. Semen was collected and sorted as previously described into microcentrifuge tubes containing 100 µl test yolk buffer (Johnson *et al.*, 1989). One ml of the sorted spermatozoa and buffer mixture was removed, placed on a mini-percoll column and centrifuged at 2000g for 3 min. The sperm pellet was resuspended in equilibrated *in vitro* fertilization medium and centrifuged at 50g for 6 min. The supernatant was removed and the remaining pellet aliquoted into 50 µl drops and placed under paraffin oil with 10-15 *in vitro* matured oocytes per drop (n=64; final concentration  $2 \times 10^5$  spermatozoa/ml; 80% motile). For comparison, fresh spermatozoa from the same boar were washed three times with Medium 199 containing 0.9 mM sodium pyruvate, 8.3 mM calcium lactate and 12% foetal bovine serum, and incubated with *in vitro* matured oocytes (n=66; final concentration of  $5 \times 10^5$  spermatozoa/ml; 90% motile). Oocytes were fixed and stained 17 h later for evaluation of fertilization.

Forty-seven percent of mature oocytes contained two pronuclei following fertilization with unsorted spermatozoa whilst a significantly lower fertilization rate (22%;  $P < 0.01$ ) was obtained with sorted spermatozoa. This discrepancy may be a result from the different concentrations of spermatozoa used at fertilization. It does not appear to be associated with ultrastructure as there was no difference in the proportion of spermatozoa exhibiting normal morphology, including intact plasma and acrosome membranes, between control (49%; n=200) and sorted (57%; n=200) groups.

These results demonstrate that (1) ultrastructure of spermatozoa is not adversely affected by the sorting process and (2) that sorted sperm can fertilize *in vitro* matured pig oocytes. Work is now focussed on improving sorted spermatozoa concentrations and viability, which may lead to improved *in vitro* fertilizing capacity of sorted spermatozoa.

### References

- JOHNSON, L.A., AALBARS, J.G. and GROOTEN, H.J. G. (1988). *Zuchthygiene*. 23:49-55.  
JOHNSON, L.A., FLOOK, J.P. and HAWK, H.W. (1989). *Biology of Reproduction*. 41:199-203.  
JOHNSON, L.A. (1991). *Reproduction in Domestic Animals*. 266:309-314.  
SIMPSON, A.M., SWAN, M.A. and WHITE, I.G. (1986). *Gamete Research*. 15:43-56.

## INTRODUCTION OF GILTS TO BOARS ELEVATES PLASMA CORTISOL BUT DOES NOT AFFECT REPRODUCTION

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It is well accepted that stressors cause elevation of plasma concentrations of cortisol and that this may reduce reproductive performance in female pigs (Clarke *et al.*, 1992). Females are particularly susceptible to stress during the endocrine events leading up to ovulation. A routine practice used by farmers is to introduce female pigs to a boar for the detection of oestrus. If the female is not sexually receptive to the boar, his active courtship may be stressful, resulting in an elevation of plasma cortisol (Jongman, 1993) which may result in a reduction of reproductive performance. The purpose of this study was to compare the cortisol response and reproductive performance of gilts detected for oestrus by introduction to boars and by a more recently developed method which is considered to be less stressful. The latter method involves putting gilts in an arena which is surrounded by pens containing boars (the Detection, Mating Arena, DMA) and testing each gilt's standing response to pressure on the back (back pressure test, BPT). The hypotheses tested were that the introduction of sexually unreceptive gilts to boars for detection of oestrus results in elevation of plasma cortisol, and that this elevation of cortisol reduces reproductive performance.

Sixty-four gilts were allocated to two treatments (32 per treatment). Gilts in the "INTRO" treatment were introduced to a boar in his own accommodation pen for 5 min. Gilts in the "BPT" treatment were put in the DMA for 5 min and their standing response to the BPT was tested. Treatment was imposed twice daily for 14 d prior to expected oestrus. Sixteen gilts in each treatment had cannulae inserted into the cephalic vein 4 d prior to the commencement of treatment. Samples of blood were taken 15 min after the start of treatment to measure the acute response of cortisol to treatment. Twelve hours after oestrus was first detected, all gilts were artificially inseminated once only. Gilts were slaughtered  $21 \pm 1$  d after insemination and the numbers of corpora lutea and embryos were recorded.

Table 1. Mean( $\pm$  SEM) response of cortisol and reproductive parameters of gilts.

	Cortisol (ng/ml)	Ovulation rate	No. of embryos	Embryos/ CL	Pregnant (%)	Oestrus (days)
INTRO	30.0 $\pm$ 1.6 <sup>a</sup>	13.5 $\pm$ 0.3 <sup>a</sup>	8.2 $\pm$ 0.9	0.63 $\pm$ 0.07	57	2.6 $\pm$ 0.2
BPT	21.0 $\pm$ 1.0 <sup>b</sup>	12.1 $\pm$ 0.4 <sup>b</sup>	7.2 $\pm$ 0.8	0.63 $\pm$ 0.06	64	2.6 $\pm$ 0.2

<sup>ab</sup>In each column values with different superscripts were significantly different,  $P < 0.05$ .

There was a significantly ( $P < 0.05$ ) greater acute response of plasma cortisol to INTRO than to BPT (Table 1), suggesting that INTRO may be the more stressful of the two procedures for detecting oestrus. Nevertheless, there was a greater ( $P < 0.05$ ) ovulation rate in gilts in the INTRO than the BPT treatment and neither treatment affected other parameters of reproduction.

### References

- CLARKE, I.J., HEMSWORTH, P.H., BARNETT, J.L. and TILBROOK, A.J. (1992). In "Stress and Reproduction", 86th edn, pp. 239-252, eds K.E. Sheppard, J.H. Boublik and J.W. Funder. (Serono Symposia Publications: New York).
- JONGMAN, E.C. (1993). The effects of conditions around the time of mating on reproductive efficiency in pigs. PhD Thesis. University of Melbourne.



## THE EFFECTS OF CONTACT FREQUENCY AND SEASON ON THE EFFICACY OF THE BOAR EFFECT

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Paterson *et al.* (1989) have conclusively shown that the boar effect (stimulation of early puberty in gilts through regular boar contact) is an additive stimulus and that regular reinforcement of the stimulus is necessary to maximise the stimulation of the attainment of early puberty in the gilt (pubertal response). Indeed, Hughes (1994a) reported that the age of gilts at puberty can be significantly reduced by increasing the frequency of daily boar contact. However, several studies have suggested that the gilt's responsiveness to puberty stimulation varied with season (Hughes, 1994a). The present experiment was therefore conducted to investigate the interaction between the frequency of boar contact and season on the timing of puberty in young gilts.

One hundred and twenty eight Large White  $\times$  Landrace gilts were used in this study. The study was conducted in four replicates (Spring, Summer, Autumn and Winter), eight gilts being allocated to each of four treatments in each replicate. The treatments involved either daily (D), twice daily (2D) or three times daily (3D) boar contact. Puberty attainment by gilts in the boar exposure regimens was compared with puberty attainment in a non-boar exposed control group of gilts (C). Boar exposure began at a mean gilt age of 160 d and continued for 60 d. The duration of boar contact was for 60, 30 and 20 min/exposure period respectively for treatments D, 2D and 3D (i.e., all boar-exposed gilts received a total of 60 min of boar contact/day). Four commercially-used boars were used in this study.

There was no significant effect of season on the timing of puberty attainment in these young gilts. The mean days to puberty  $\pm$  SEM for the Spring, Summer, Autumn and Winter replicates were  $32.2 \pm 3.70$  d,  $22.8 \pm 2.98$  d,  $33.7 \pm 3.75$  d and  $34.5 \pm 3.41$  d respectively. Boar exposure significantly increased the proportion of gilts attaining puberty within 60 d of the commencement of treatments ( $P < 0.001$ ) compared with control gilts (0.71 vs 0.13 respectively). Boar exposure three times daily significantly reduced mean gilt age at puberty relative to once-daily boar exposure ( $183.2 \pm 2.71$  vs  $196.0 \pm 3.00$  days of age respectively,  $P < 0.01$ ). Twice-daily boar contact resulted in an intermediate mean gilt age at puberty ( $190.3 \pm 3.01$  d). There was also a trend towards a higher proportion of gilts reaching puberty earlier with increasing frequency of boar contact. However, this difference was only significant ( $P < 0.05$ ) between once- and three times-daily boar contact at day 20 of treatment.

It is clear that the pubertal response of the gilt to the boar effect is enhanced when the frequency of daily boar contact is increased. However, while other reports have indicated that the pubertal response of the gilt to boar contact is modulated by season, this effect was not evident in the present results. As considerable between-boar differences exist in their ability to stimulate precocious puberty in gilts (Hughes, 1994b), it is tentatively suggested that the commercially-used boars in this study may have released sufficient stimuli to overcome any seasonal depression which may have occurred.

### References

- HUGHES, P.E. (1994a). *Animal Reproduction Science*. 35:273-280.  
HUGHES, P.E. (1994b). *Animal Reproduction Science*. 35:111-118.  
PATERSON, A.M., HUGHES, P.E. and PEARCE, G.P. (1989). *Animal Reproduction Science*. 21:115-124.

## EFFECT OF PRE-PUBERTAL BODY FAT ON THE DEVELOPMENT OF THE REPRODUCTIVE TRACT IN GILTS

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Selection for leanness may lead to lower reproductive efficiency in the sow (Young *et al.*, 1991; Gaughan *et al.*, 1995).

The effect of body fat on reproductive development was examined in 54 pre-pubertal gilts. The gilts ( $145 \pm 3$  days of age) were housed in groups of four and were fed a diet containing 14.4 MJ DE/kg, 0.57 g available lysine MJ DE and 200 g crude protein at a rate of 2.8 kg/pig/day. The gilts were grouped according to depth of backfat at P2 (Bd). The groups were lean (L) 10 to 12 mm (84 kg live-weight), medium (M) 13 to 15 mm (88 kg) and fat (F)  $\geq 16$  mm (93 kg). The gilts were monitored daily for signs of oestrus. Blood samples were collected every 3 d until first oestrus and serum samples were analysed for progesterone (P<sub>4</sub>) concentration. Elevation in P<sub>4</sub>, visual observation and boar behaviour were used to confirm oestrus. At puberty, weight and Bd were measured. The gilts were slaughtered at 202 days of age, the reproductive tracts were removed and examined for the number of corpora lutea (CL) to determine ovulation rate and the number of follicles ( $>3$  mm) to determine ovarian activity.

All the F gilts, 92% of M gilts and 67% of L gilts reached puberty by slaughter. The L gilts were 12 days older (184.5 days) at puberty than either M ( $P < 0.05$ ) or F ( $P < 0.01$ ) gilts. Age at puberty for M and F gilts were similar to the herd average. There were no significant differences for Bd (L 17.4; M 15.9 and F 17.9 mm) or weight (L 97.7; M 100.5 and F 109.1 kg) at puberty. The number of oestrous cycles for L gilts was significantly lower than for either M ( $P < 0.01$ ) or F ( $P < 0.001$ ) (Table 1). The L gilts also had significantly fewer follicles than either the M ( $P < 0.01$ ) or F ( $P < 0.05$ ) gilts. There were no differences in the number of CL. At slaughter, mean Bd for L gilts at  $18.0 \pm 0.95$  mm was significantly lower ( $P < 0.05$ ) than the F gilts ( $21.6 \pm 1.16$  mm) but similar to the M gilts ( $19.4 \pm 0.82$  mm). Slaughter weights ( $124 \pm 4$  kg) did not differ significantly.

**Table 1. Reproductive parameters (least squares mean) for gilts which reached puberty.**

	Lean	Medium	Fat	SEM
Number of gilts	8	26	16	
No. of oestrous cycles	1.16 <sup>a</sup>	1.96 <sup>b</sup>	2.25 <sup>b</sup>	0.10
No. of follicles	13.14 <sup>a</sup>	19.08 <sup>b</sup>	18.25 <sup>b</sup>	0.78
No. of corpora lutea	11.08 <sup>a</sup>	11.90 <sup>a</sup>	12.75 <sup>a</sup>	0.38

Means within a row with different superscripts differ significantly  $P < 0.05$

The rate of fat deposition and weight gain of the L group after selection is evidence of later physiological development. The delay in puberty was because the gilts were physically less mature and not as a result of an underlying reproductive problem. The greater ovarian activity of M and F gilts may be more a function of the number of heat periods rather than body composition. Selection for leanness may lead to delayed reproductive development. However, the factors which restrict growth and development may be more important than the effect of leanness *per se* on reproduction.

### References

- GAUGHAN, J.B., CAMERON, R.D.A., DRYDEN, G.McL. and JOSEY, M.J. (1995). *Animal Science*, 61: (In press).
- YOUNG, L.G., KING, G.J., SHAW, J., QUINTON, M., WALTON, J.S. and McMILLAN, I. (1991). *Canadian Journal of Animal Science*, 71:567-575.

## THE EFFICACY OF THE BOAR EFFECT WHEN CONDUCTED IN A DETECTION-MATING AREA

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The Pig Research and Development Corporation (1994) recommend that early puberty stimulation in gilts can be achieved by either full boar contact or the use of a Detection-Mating Area (DMA) as described by Hemsworth *et al.* (1991). While this recommendation is supported by the results of a small study reported by Turner *et al.* (1994), many other reports (see Hughes *et al.*, 1990 for review) suggest that the fenceline contact permitted in a DMA would not maximise puberty stimulation in gilts. This study therefore compared the effects of boar contact in, either a DMA or a conventional pen allowing full physical contact, on the attainment of puberty in the gilt.

Fifty six Large White  $\times$  Landrace gilts from 14 litters were allocated to four treatments by litter and live-weight in a 2  $\times$  2 factorial experiment. The four treatments were (1) no full boar contact, no exposure to the DMA, (2) no full boar contact, exposure to the DMA, (3) full boar contact, no exposure to the DMA, and (4) full boar contact, exposure to the DMA. Five mature boars were used for 20 min/d as stimulus animals in treatments 2-4, these being rotated for use in the DMA stalls, the DMA exposure pen and the isolated exposure pen. Each gilt thus received 20 min/d of boar contact either in the DMA or in an isolated pen. All treatments began at a mean gilt age of 160 d and continued on a daily basis for 60 d. Daily boar exposure significantly reduced mean gilt age at puberty (36.1 vs 56.8 d, SEM = 5.09, LSD = 7.19) and increased the proportion of gilts attaining puberty within 60 d of commencement of treatment compared with gilts which received no boar exposure (Table 1). In contrast, daily exposure to a DMA failed to significantly reduce mean gilt age at puberty or to increase the proportion of gilts attaining puberty within 60 d of commencement of treatment compared with gilts which received no DMA exposure (Table 1).

**Table 1. The effects of treatment on gilt puberty attainment.**

	Proportion pubertal <sup>1</sup>	Days to puberty	SEM
No full boar contact	0.41 <sup>a</sup>	56.8 <sup>x</sup>	3.26
Full boar contact	0.79 <sup>b</sup>	36.1 <sup>y</sup>	3.88
No DMA exposure	0.59	49.7	3.35
DMA exposure	0.50	42.9	4.63

<sup>1</sup> Gilts reaching puberty by day 60 of the study. Within columns a,b and x,y superscripts denote differences at P<0.05 and P<0.01 respectively.

It is concluded that (1) daily boar exposure is a potent stimulus for early puberty attainment in gilts, (2) full physical contact with the boar must occur in order to achieve the boar effect, and (3) the current recommendation that gilt puberty stimulation can be adequately achieved by exposing them to a DMA on a daily basis requires re-assessment.

### References

- HEMSWORTH, P.H., HANSEN, C., COLEMAN, G.J. and JONGMAN, E. (1991). *Applied Animal Behaviour Science*. 30:273-285.
- HUGHES, P.E., PEARCE, G.P. and PATERSON, A.M. (1990). *Journal of Reproduction and Fertility*. 40 (Supplement):323-341.
- PIG RESEARCH and DEVELOPMENT CORPORATION (1994). "Mating and Reproduction", 2nd edn. (PRDC: Canberra, Australia).
- TURNER, A.I., HEMSWORTH, P.H., LOHUIS, H. and TILBROOK, A.J. (1994). *Proceedings of the Australian Society for Reproductive Biology*. 26:11.

## A SYMPOSIUM - CONSTRAINTS TO PRE-WEANING GROWTH

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### Introduction

There is clear evidence that increasing the growth rate of piglets prior to weaning has a marked effect on their lifetime performance and on the profitability of the pig enterprise (Mahan and Lepine, 1991). The average growth rate of piglets prior to weaning at 3 to 4 weeks of age rarely exceeds 230 g/d (Whittemore, 1993). The results of recent experiments conducted in a commercial herd in Australia have revealed that pre-weaning growth rate of pigs in average litter sizes of 9 - 10 piglets is about 215 g/d (Tritton *et al.*, 1993). However, post-natal live-weight gains of over 500 g/d are possible during the first weeks of life through the supply of sufficient nutrients to artificially-reared piglets (Hodge, 1974). Commercial pig production has yet been unable to exploit fully the potential of early growth that can be offered by the young piglet.

Sow lactation was extensively reviewed by Hartmann and Holmes (1989) who concluded that the important role of sows' milk in promoting piglet growth, development and protection against pathogenic micro-organisms had been largely ignored. In addition they considered that more information was required about the factors affecting the relationship between the piglets' demand for milk and the sow's capacity to produce milk before real progress could be made in developing management strategies to maximise the benefits of sows' milk.

Since the review by Hartmann and Holmes (1989), the Pig Research and Development Corporation (PRDC) have developed and funded a sow lactation program which has been directed towards identifying key factors which influence the production of milk by sows, and to develop strategies to improve piglet weaning weight without compromising sow performance. The profitability of commercial pig production could be increased substantially if piglets could achieve growth rates before weaning that are much closer to their potential. An AUSPIG simulation conducted by B.P. Mullan (personal communication) showed that if piglet pre-weaning growth could be increased from 200 to 250 g/d, slaughter weight would increase by over 5 kg and net revenue per sow would almost double.

The aims of this symposium are to examine the current knowledge about the major factors that may influence weaning weight, to identify the limitations to sows' milk production and piglet growth, and to discuss opportunities to manipulate the production of milk by sows and the nutrient intake of piglets to improve pre-weaning growth performance.

## METABOLIC REGULATION OF SOW LACTATION

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### Introduction

Milk is a very complex secretion consisting of cells (leucocytes, macrophages and epithelial cells), lipids (triacylglycerols, free fatty acids, phospholipids, sterols, hydrocarbons and fat-soluble vitamins), carbohydrates (lactose, oligosaccharides, galactose, glucose and glycoproteins), proteins (caseins,  $\alpha$ -lactalbumin,  $\beta$ -lactoglobulin, lactoferrin, secretory IgA and other immunoglobulins, lysozyme, enzymes, hormones and growth factors), non-protein nitrogenous compounds (urea, creatine, creatinine, uric acid, amino acids including glutamine, nucleic acids, nucleotides and polyamines), water-soluble vitamins, macronutrient elements and trace elements (Renner, 1989). Furthermore, it is now clear that the proportions of these constituents in milk of a particular species are uniquely appropriate for their young at a time when growth and development are occurring at near-maximal rates, yet when many of the neonate's systems (such as, digestive, hepatic, immune, neural, vascular, renal and skeletal) are functionally immature. Milk must be considered, not only in the narrow role of supplying an appropriate mix of classical nutrients to the young mammal, but also in the context of providing metabolites, enzymes, growth factors and hormones to counter deficiencies in the neonate's immature metabolism and thereby promote optimum rates of growth and development. These considerations are of particular importance during lactation as weaning weight is an important determinant of subsequent growth. Furthermore, an understanding of the factors in sows' milk which lead to the optimal metabolic development and growth of piglets is essential for the formulation of diets for very-early weaning, i.e., within the first two weeks of life.

Successful lactation requires the development of fully-functional mammary glands. Whereas other major organs are relatively mature at birth, both morphologically and functionally, the mammary gland undergoes very limited structural development *in utero* with the most dramatic changes occurring in the sow during the lactation cycle - pregnancy, lactation and weaning.

### Udder development in the non-pregnant sow

The glands of the newborn piglet have a poorly developed ductal system and consist largely of connective tissue (Hughes and Varley, 1980). The glands remain quiescent until approximately 6 months of age when the pig reaches puberty (Turner, 1952). With the onset of ovarian activity there is a rapid development of the ductal system in response to hormonal stimuli (Hughes and Varley, 1980) and, by the time of mating at approximately 8 months of age, the mammary glands consist of an extensive duct system and numerous "lateral sprouts or bud-like outgrowths" (Turner, 1952). In general, there is little or no development of the lobulo-alveolar system before the first pregnancy in the sow as in other mammals (Knight and Peaker, 1982).

### Lactation cycle

Conception marks the beginning of the lactation cycle which includes further development of the udder (mammogenesis), the initiation of milk synthesis (lactogenesis 1) and secretion (lactogenesis 2), lactation (galactopoiesis), and regression of the udder after weaning (involution).

### Mammogenesis

By 45 days post-conception development of the lobulo-alveolar system (the milk secretory apparatus) is present (Cross *et al.*, 1958). However, the major development occurs between 75 and 90 days of gestation and is characterized by rapid tissue growth, large increases in total DNA and RNA content associated with the lactocytes (mammary secretory epithelial cells), a reduction in adipose and connective tissue and an increase in the number of lobules and alveoli (Kensinger *et al.*, 1982). The total DNA content associated with the lactocytes is maximal by day 90 of gestation, indicating the completion of mammogenesis.

Weldon *et al.* (1991) attempted to stimulate mammogenesis by altering the diet of the pig in the last third of pregnancy (i.e. when there is a rapid increase in DNA in the gland) but with no positive result. However, Head and Williams (1991) and Head *et al.* (1991) have shown that mammogenesis can be influenced by nutrition. In a study on fat and lean gilts it was shown that the lean animals showed a four-fold increase in DNA concentration and twice the number of lactocytes in the mammary tissue at day 112 of gestation. In this connection it is of interest that failure of lactation has been associated with over nutrition of beef heifers (Owens *et al.*, 1993). Thus it appears possible to influence the potential number of secretory cells in the mammary gland during pregnancy but this may relate more to a reduction from normal cell numbers than to stimulation of a greater secretory capacity.

### Lactogenesis 1

The term lactogenesis I refers to the development of the capacity of the mammary gland to synthesize unique milk components (eg. lactose, casein,  $\alpha$ -lactalbumin).

Recent investigations have shown that the concentration of specific milk proteins (eg.,  $\beta$ -lactoglobulin,  $\alpha$ -lactalbumin) in the blood during late pregnancy provide a good predictor of milk production during the subsequent lactation in dairy cows (Mao and Bremel, 1995). Dodd *et al.* (1994) observed an increase in the plasma concentration of  $\alpha$ -lactalbumin one week prior to farrowing, with a marked rise 3 d pre-partum, which correlated to declining concentrations of progesterone. In contrast,  $\beta$ -lactoglobulin concentrations increased from as early as 5 weeks pre-partum. Thus, the concentration of these two whey proteins in the plasma reflects mammary gland development. Despite the difference in the timing of lactogenesis 1 between cows and sows, the possibility that these or other unique products of milk may be useful predictors of lactation performance in sows should be investigated.

### Lactogenesis 2

Lactogenesis 2 is used to refer to the initiation of copious milk secretion (Cowie *et al.*, 1980). Histological analysis of mammary tissue on the day of parturition has shown structural arrangements characteristic of lactocytes (Saake and Heald, 1974; Kensinger *et al.*, 1986). Consequently Kensinger *et al.* (1982, 1986) concluded that lactogenesis 2 in the sow occurs just prior to parturition and is characterized by morphological changes in the mammary tissue. However, other workers have assessed lactogenesis 2 in a variety of ways and achieved a variety of results. The increase in the concentration of lactose in the mammary secretion (Martin *et al.*, 1978; Gooneratne *et al.*, 1979) and blood (Hartmann *et al.*, 1984) following farrowing has been used as an indicator of lactogenesis 2. Other workers (Nara and First, 1981; Nara *et al.*, 1982) have assessed lactogenesis 2 as the time when milk can first be expressed manually from the gland. However, Willcox *et al.* (1983) observed that the onset of lactation was extremely rapid and varied greatly among sows and that the expression of mammary secretion may not be an accurate measure of lactogenesis 2. Although lactogenesis 2 is defined as the onset of copious milk synthesis and secretion, there is inconclusive physiological evidence for the exact timing of lactogenesis 2 in the sow.

### *Control of lactogenesis 2*

The ability of the mammary gland to synthesise milk is thought to be controlled by both endocrine (hormonal) and autocrine (local) factors, while milk yield is determined by both these factors and the number of secretory cells. There is a decline in progesterone during farrowing (Robertson and King, 1974) which is closely associated with increased lactose synthesis by the gland (Hartmann *et al.*, 1984), and hence progesterone withdrawal is considered the hormonal signal for the initiation of lactogenesis 2 in a number of species (Kuhn and Lowenstein 1967; Hartmann *et al.*, 1973; Kulski *et al.*, 1977). Martin *et al.* (1978) found a negative correlation between the concentration of lactose in colostrum and the concentration of progesterone in sows' blood, while Gooneratne *et al.* (1979) and Whitely *et al.* (1990) showed that the administration of progesterone during late pregnancy delayed lactogenesis 2 in the sow. Therefore progesterone withdrawal also appears to be the trigger for lactogenesis 2 in the sow.

Atwood and Hartmann (1993) observed an increase in the concentration of fat in colostrum prior to the increase in lactose during farrowing. In addition, they observed that the amount of colostrum removed from the glands by the newborn piglets had a major influence on the increase in the concentration of fat during the farrowing period. It is of interest that in the rat both the withdrawal of progesterone (Murphy *et al.*, 1973) and removal of colostrum (Martyn and Hansen, 1980) was necessary to initiate fatty acid synthesis in the gland. In addition to fat, Atwood *et al.* (1995) showed that the increase in lactose and its metabolites in colostrum was promoted by the removal of colostrum by the newborn piglets. Their findings suggest that lactogenesis 2 in the sow is under an additional level of control. The withdrawal of progesterone primes the gland for lactogenesis 2 but withdrawal of colostrum triggers the initiation of copious milk secretion, presumably through an autocrine mechanism (see review by Hartmann *et al.*, 1995). Thus milk synthesis in the udder of the sow is controlled to ensure maternal energy is conserved by not 'switching-on' lactogenesis 2 in unsuckled glands. Furthermore, the dependence of the initiation of lactogenesis 2 on the withdrawal of colostrum, together with the early development of teat order, ensures that the last born piglet obtains colostrum of the same composition and protective capacity as the first born.

Throughout pregnancy relaxin is synthesised and accumulated in the corpora lutea of the sow and is released into the circulation just prior to parturition. In the absence of circulating relaxin there is prolongation of parturition and a decrease in the frequency of live births (Nara *et al.*, 1982). Furthermore, a relationship between poor piglet performance and high post-partum concentrations of progesterone in sows' blood was found by de Passillé *et al.* (1993) who hypothesised that high mortality and poor piglet growth rates resulted from an inhibition of the onset of lactogenesis 2, leading to reduced availability of milk for the piglet. In a preliminary study, a positive relationship between lactose concentration in the plasma of sows and the litter growth rate was observed by Glencross *et al.* (1994). This finding supports the hypothesis of de Passillé *et al.* (1993) that suppression of lactogenesis 2 resulting from elevated progesterone concentrations causes poor postnatal growth. In this connection, it is of interest that early investigations of the synchronization of farrowing in sows used prostaglandin administration to induce parturition in sows before their earliest expected date of farrowing, i.e., at about day 111 of gestation (Ash and Heap, 1973; Diehl *et al.*, 1974; Walker, 1977). Despite the fact that this resulted in the premature birth of piglets their viability and growth was surprisingly good. Subsequent investigations by Gooneratne *et al.* (1979) found that progesterone could be used to prevent premature farrowing. The subsequent withdrawal of progesterone administration coupled with prostaglandin administration provided good synchronization of delivery on day 116 of gestation. Fetal mortality, birth weights and growth rates of piglets remained unaffected by this treatment. In view of the findings of de Passillé *et al.* (1993) it is possible that the administration of prostaglandin rapidly decreased progesterone to low levels facilitating a favourable development of lactogenesis 2, subsequent lactation and good piglet growth.

Hormonal interventions should not only control the time and duration of farrowing, but also facilitate the rapid withdrawal of progesterone to permit optimal development of lactogenesis 2. Such procedures have important practical applications in commercial piggeries. However, some caution must be exercised in the development of these protocols as the early initiation of lactogenesis 2 has been associated with the development of mastitis, metritis and agalactia (MMA) in sows (Gooneratne *et al.*, 1982).

Milk ejection during lactogenesis 2 and established lactation is under the control of oxytocin. With the difficulties associated in measuring acute changes in the circulating concentration of oxytocin, measurement of intramammary pressure provides a viable, indirect means of determining the release of oxytocin into the blood. Chard (1974) wrote that "the measurement of circulating oxytocin is not, and may never be, a simple routine procedure." Whittlestone (1954a,b) characterized the response of the myoepithelium of the mammary gland to various doses of oxytocin or oxytocin equivalents. He concluded that the mammary myoepithelium showed a delay in its response to oxytocin. Furthermore, it would appear that the myoepithelium has a period during which it is insensitive to oxytocin. Moreover, Smith (1994) noted multiple intramammary pressure peaks at farrowing in the sow, and concluded that there was a cyclical response to oxytocin, where the myoepithelial cells undergo a period of relaxation before again responding to stimulation. This prevents a continuous response under conditions, eg., at parturition, where oxytocin concentrations in the blood remain elevated for long periods of time (Ellendorff *et al.*, 1979; Forsling *et al.*, 1979; Fuchs, 1985; Whitely, 1989).

In addition to the role of oxytocin in milk ejection, Dale (1909) discovered that extracts from the posterior pituitary caused uterine contractions. The elevation in the concentration of oxytocin at parturition was believed to facilitate the birthing process by enhancing uterine activity (Fuchs, 1985). The first recordings of changes in intramammary pressure during the pre-farrowing and farrowing periods in the sow have recently been reported by Smith (1994). Pressure peaks occurred in 51 clear traces (not confounded by movements of the sow, piglets or experimenter) in the minute after the delivery of a piglet; less than 12% of the clear traces showed pressure peaks just prior to, or concurrent with, delivery. In contrast to the accepted dogma of the Ferguson reflex (Folley, 1969), Smith (1994) concluded that the stimulus for the release of oxytocin was the relaxation of the distended birth canal rather than the act of distending the canal (Ferguson, 1941; Roberts and Share, 1968; Kendrick *et al.*, 1991). The conclusions of Smith (1994) have recently been confirmed by Gilbert *et al.* (1994). It has been shown that infusion of oxytocin into the cerebrospinal fluid induces maternal behaviour in virgin rats (Pedersen and Prange, 1979) and the results of Smith (1994) and Gilbert *et al.* (1994) would appear to direct the role of oxytocin away from an aid to the birthing process and toward post-delivery maternal behaviour (Keverne, 1988). The facilitation of the release of oxytocin in the brain of the sow may provide a management strategy for the improvement of maternal behaviour in sows and thereby increase piglet postnatal survival.

#### Established lactation (galactopoiesis)

During the first few days of lactation there is a rapid change in the proportions of the major milk nutrients such as protein, lactose and fat (Fahmy, 1972; Willcox *et al.*, 1983) which then stabilise within a few days of lactation. The literature pertaining to the concentrations and functions of these major components has been reviewed recently (Hartmann and Holmes, 1989). To date it has not been possible to manipulate these major milk nutrients to benefit productivity in any mammal of economic importance. However, this may not apply to important minor milk components. One minor component studied within our laboratory is the non-protein nitrogen compound creatine. Creatine has been identified in human, bovine and porcine milk (Atkinson *et al.*, 1989) and recently, creatine phosphate has been identified as a minor component of sows' milk (Comber and Hartmann, 1993). Both creatine and creatine phosphate are involved in many biological processes within the body and also are associated with the growth of muscle fibres and brain development. The concentration of creatine increases rapidly from farrowing to about day 5 of lactation and then remains at the higher concentrations throughout established lactation. In a preliminary study in this laboratory involving five sows, the concentration of creatine in pre-farrowing colostrum showed little variation



among glands but varied significantly among sows. However, during established lactation, the creatine concentration varied significantly among glands (Figure 1) but not among sows. These compounds may serve as an indicator of glandular function and milk synthesis during lactogenesis, as well as being of benefit to the growth and development of the piglet.

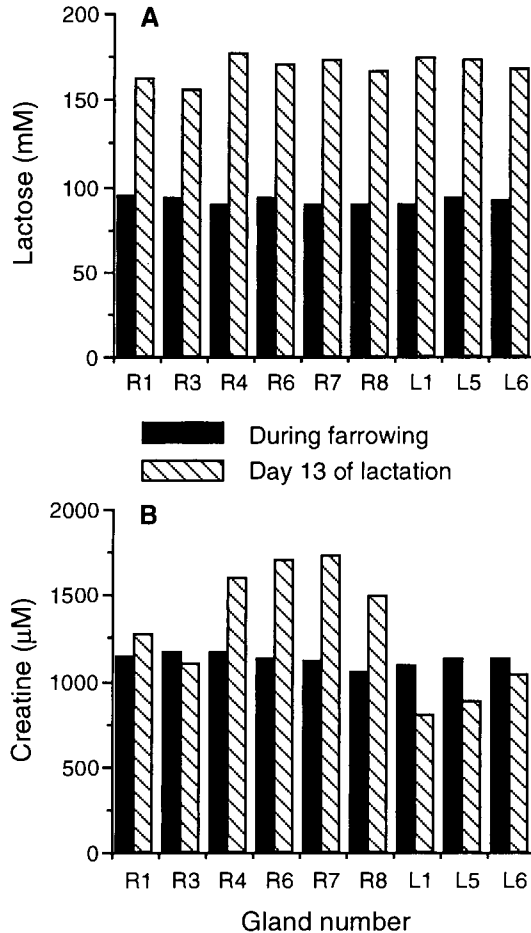


Figure 1. The concentration of lactose (A) and creatine (B) in milk from glands of one sow during farrowing (day 0) and during established lactation (day 13).

#### Control of established lactation.

Galactopoiesis (maintenance of established lactation) appears to be under a combination of endocrine, autocrine and metabolic control which varies according to the species and the stage of lactation. The relative importance of these mechanisms depends on whether or not the species has been selected for dairy production. Nevertheless, the removal of milk from the mammary gland is of the utmost importance for the maintenance of milk secretion in all mammals.

Prolactin plays a major role in the maintenance of milk secretion (Forsyth, 1986; Vernon, 1989) and is released upon tactile stimulation of the nipple/teat. In addition, it has been shown by Plaut *et al.* (1989) that the binding of prolactin to its receptor is a major effector of milk production in the sow. Prolactin remains elevated in the blood of sows throughout early lactation as a result of frequent sucking by the piglets (Threlfall *et al.*, 1974; van Landeghem and van de Weil, 1978; Holmes, 1991), and then declines after approximately 3 weeks (van Landeghem and van de Weil, 1978). In addition, it has been shown that insulin is important for the maintenance of milk secretion (Vernon, 1989) and may be required for the survival of lactocytes during lactation (Baldwin and Louis, 1975). However, insulin does not appear to influence the uptake of glucose by the mammary gland of the sow (Holmes, 1991).

The milk ejection reflex of the sow is under tight control, with milk only available to the piglets during a 'let-down' which lasts for about 15 seconds (Smith, 1994). This control, primarily as a result of oxytocin release, ensures that a dominant piglet does not deprive others of milk. However, this strategy limits the amount of milk that can be removed by the piglets during a suckling. Peaker and Wilde (1987) proposed the autocrine control theory of milk synthesis during established lactation for dairy goats. They isolated a protein called feedback inhibitor of lactation (FIL) which appears to suppress milk synthesis as milk accumulates in the mammary gland between sucklings by reversibly inhibiting the transfer of newly synthesized protein from the endoplasmic reticulum to the Golgi vesicles. If an autocrine mechanism is involved in the control of milk synthesis in the sow, then the tightly controlled duration of milk ejection may prevent the piglet from removing all of the available milk from the gland. Hence the duration of milk letdown, rather than the piglet's appetite, may regulate the removal of the inhibitory compound(s) and thereby the subsequent rate of milk synthesis.

The presence of lipases in milk suggests that an autocrine mechanism of control of milk synthesis which involves free fatty acids is possible in lactating sows. It has been shown that there is a difference in the fat content of sows' milk between the beginning and end of a suckling (Atwood and Hartmann, 1992), and Heesom *et al.* (1992) have demonstrated that medium-chain free fatty acids inhibit glucose metabolism and lipid synthesis in isolated mammary acini of rats.

Molenaar *et al.* (1992) used <sup>35</sup>S-labelled cRNA probes to localize the sites of mRNA synthesis for  $\alpha$ -lactalbumin,  $\alpha$ -S1-casein, and lactoferrin in sheep. In early lactation expression of  $\alpha$ -lactalbumin and  $\alpha$ -S1-casein was high in some alveoli but not in others. Furthermore, there were more fat globules in the cells and lumen of alveoli which had low expression of these proteins. However, these alveoli almost exclusively expressed lactoferrin. This suggests that milk secretion is either heterogeneous across lobules or occurs sequentially with time in the lactocytes of the alveolus as newly-secreted milk accumulates between sucklings. The latter concept seems more plausible.

It is suggested that FIL and free fatty acids may act locally to sequentially regulate the short-term rates of synthesis of milk constituents either within the udder or, more probably, within the lobules in the udder depending on the degree of emptying of the lobules. However, ultimately the maximum volume of milk that can be removed by the piglet will be limited by the metabolic capacity of the sow to synthesize milk.

## Involution

In intensive, commercial piggeries lactation is abruptly terminated by weaning the piglets at 3 to 4 weeks post-partum, even though milk production is maximal at this time (Hartmann and Holmes, 1989). A study by Atwood (1993) on partial (selected glands) weaning in the sow showed that the composition of the milk was unchanged over the following 5 h. In contrast, total weaning of the litter resulted in a significant decrease in the concentration of glucose and significant increases in the concentrations of glucose 6-phosphate and UDP-galactose in the milk, over the same time period.

### Summary

The composition and production of milk, which evolved to maximise the survival of feral piglets, may not permit the optimal growth and development of piglets in the controlled environment of an intensive piggery. Recent research into the initiation and maintenance of lactation in the sow has revealed that the factors controlling milk synthesis are more complex than those described for other species. Whereas in other species progesterone withdrawal triggers the initiation of copious milk production at birth (lactogenesis 2), in sows both a fall in progesterone to very low concentrations and the withdrawal of colostrum are required before lactogenesis 2 will occur in a particular gland. Thus lactogenesis 2 is under both endocrine and autocrine control in the sow. During established lactation (galactopoiesis) milk production appears to be under autocrine control in sows as in other species. However, the very short period of milk flow in the sow (~15 seconds) at each suckling (a strategy consistent with restricting the influence of dominant piglets) limits the ability of the piglet to empty the gland. Thus the gland is not fully released from inhibitory autocrine factors which inhibit milk production. A better understanding of these processes is required before milk production can be effectively manipulated to help reduce the number of neonatal deaths and to improve the growth rate of piglets in large litters, which are now a feature of modern, intensive pig production.

### Acknowledgments

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## THE INFLUENCE OF SUBSTRATE SUPPLY ON MILK PRODUCTION IN THE SOW

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### Introduction

The lactating sow is one of the marvels of modern agriculture. Genetic selection and sophisticated management technology have produced modern sows with a massive capacity to convert substrates into milk, in order to support the rapid growth rate of large litters. Production of this milk requires an immense supply of substrates, the raw material for the manufacture of milk by the sow's mammary glands. To provide that quantity of substrates is a daunting task. The substrates come from two sources, namely the diet consumed during lactation, and the sow's body reserves. Both are quantitatively important. This discussion focuses on the supply of energy and amino acids, but the reader should recognize that supplies of water, minerals and vitamins are also important.

A concept of how nutrition affects milk production, incorporating the contribution of substrates from both sources, is offered in Figure 2. This concept emphasizes the key role of the sow's metabolic state in bringing together substrates from the diet and from body reserves to support milk production. The metabolic state is a nebulous concept that includes, but is not limited to:

- metabolite (substrate) concentrations
- metabolic hormone concentrations
- tissue sensitivity to these hormones (receptor number and affinity)
- tissue metabolic capacity ( $V_{max}$ )
- affinity constants ( $K_m$ ) for substrates
- homeostasis
- homeorhesis

The metabolic state is largely a function of nutrient intake, but during lactation is also affected by the amount of body reserves (protein and fat) available for mobilization to supply substrates to the mammary glands. Of course, the metabolic state also affects the rate of net deposition or mobilization of these reserves, and cumulatively over time affects their quantity. Non-nutritional effects, including genetic strain, parity, and stage of lactation, also affect the metabolic state. For example, the tissue metabolic capacity is presumed to increase with advancing parity during the early parities.

There are other non-nutritional factors that affect the amount of milk produced, including the number of sucking piglets and the aggressiveness with which they nurse. As litter size increases, milk yield increases even when nutrient intake and body reserves are not changed. This increased milk yield increases the outflow of nutrients, and thus affects the metabolic state.

Thus, neither substrate supply (metabolic state, including nutrient intake and body reserves) nor the milk demand of the litter is the sole determinant of milk yield. Often, changing either nutrient intake or milk demand by the litter can alter milk yield. Milk yield is regulated by complex interactions between metabolic state and milk demand.

For total substrate supply, the relative importance of current nutrient intake and body reserves seems to change as lactation progresses. It appears that during the first few days of lactation, milk yield is relatively insensitive to current nutrient intake. However, as lactation progresses, current nutrient intake becomes increasingly important.

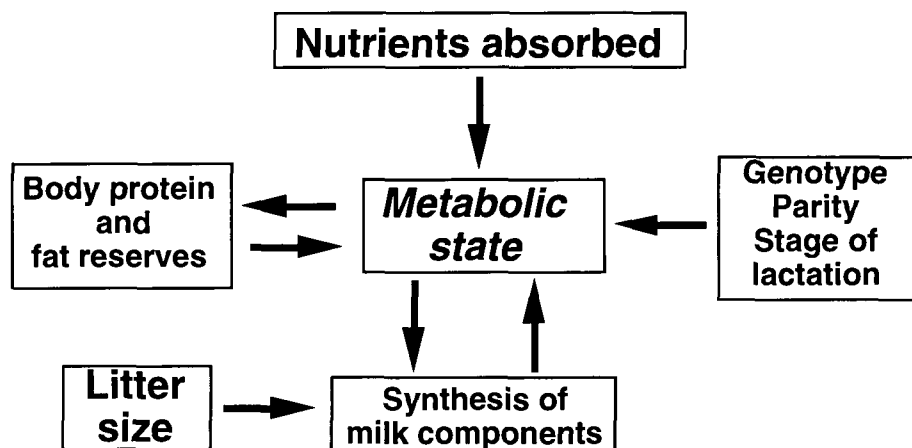


Figure 2. Schematic representation of a concept of the role of metabolic state in bringing together substrates from the diet and from body reserves to support milk production. Modified from Pettigrew et al. (1993).

#### Changing relative importance of body reserves and the diet during lactation

Protein and fat contained in the sow's body can contribute to the difficult task of providing ample substrates to the lactating mammary glands, especially during the first week of lactation. The changing importance of these protein and fat reserves as lactation progresses has been shown in several experiments.

For example, Mullan and Williams (1989) provided pregnant gilts with different amounts of feed in order to vary the amount of their body reserves at farrowing. Specifically, they provided three treatments during gestation: High - 3.0 kg/d, reduced to 2.0 kg/d during the last 30 d; Medium - 2.0 kg/d; Low - 1.5 kg/d. After farrowing, sows on each gestation treatment were divided into two groups and offered two levels of feeding during lactation: High - *ad libitum*, and Low - 2.0 kg/d. Results (Table 1) showed that during the first week of lactation, litter growth was not affected by the current feeding level, but was reduced by the low feeding level during gestation. During the fourth week of lactation there was a large effect of feeding level, and an interaction between the treatments during gestation and lactation. Litter growth was most rapid in sows allowed *ad libitum* access to feed during lactation, and in these sows was not affected by the amount of body reserves in the sow at farrowing. Severe restriction of feed intake during lactation caused a marked reduction in litter growth during the fourth week, and the size of this reduction depended on the amount of reserves at farrowing. Feed restriction was most detrimental in sows with the least reserves.

Table 1. Effect of level of feed intake during gestation and lactation on piglet growth (Mullan and Williams, 1989).

	Level of feed intake						SED
	High	High	Medium	Medium	Low	Low	
Gestation	High	Low	High	Low	High	Low	
No. of litters	42	36	62	46	53	36	
Piglet growth (g/d)							
Week 1	194 <sup>a</sup>	190 <sup>a</sup>	199 <sup>a</sup>	185 <sup>a</sup>	174 <sup>b</sup>	172 <sup>b</sup>	9
Week 4	210 <sup>a</sup>	186 <sup>b</sup>	210 <sup>a</sup>	159 <sup>c</sup>	219 <sup>a</sup>	152 <sup>c</sup>	10
No. of pigs weaned	8.5	8.3	8.9	8.7	8.5	8.4	1.3

<sup>abc</sup>Means within a row with different superscripts are significantly different ( $P < 0.05$ ).

Kusina *et al.* (1995) have recently reported a similar response to amino acid intake. Gilts were fed three different levels of amino acids during pregnancy. All amino acids were from intact proteins, and the levels provided were 4, 8, or 16 g lysine/d. During lactation the gilts were provided with high or low levels of amino acids. Litter weaning weights were affected by the treatments during gestation and lactation independently. However, milk yield measurements suggested that the amino acid intake during gestation affected milk yield during early lactation whereas milk yield in later lactation was influenced by amino acid intake during lactation. The intermediate level of amino acid intake during pregnancy (8 g lysine/d), which is near the NRC (1988) lysine requirement, did not adequately support maximal milk yield even at the higher level of amino acid intake during lactation. This is consistent with a factorial estimate of amino acid requirements during pregnancy (Pettigrew, 1993), which suggests that pregnant gilts need 11 to 14 g lysine/d to gain 30 to 40 kg of lean tissue during pregnancy. Probably most gilts used in commercial pork production consume this much lysine.

The initial hypothesis was that any detrimental effect of low amino acid intake during gestation on subsequent milk production would be mediated through effects on the degree of mammary development, as might be inferred from the data of Head and Williams (1991) and Head *et al.* (1991). They showed impaired mammary development in gilts given a low-protein, high-energy diet during pregnancy. However, the gilts fed the same diets during gestation as described above and slaughtered near term showed no treatment effects on mammary development, as measured by total amounts or concentrations of DNA, RNA, RNA/DNA ratio, or protein in the mammary parenchyma (Kusina *et al.*, unpublished data). Perhaps the effects on mammary development reported by Head and Williams (1991) and Head *et al.* (1991) were caused by high energy intake rather than by low amino acid intake. It is suggested that the effects on milk yield observed in the experiment by Kusina *et al.* (1995) were mediated through effects on body protein stores, although specific effects on the developing fetuses cannot be ruled out.

Finally, Koketsu (1994) has surveyed daily feed intake of about 25,000 lactating sows on commercial farms in the Midwestern US. There were significant relationships of litter weaning weight to feed intake of the sows during the three individual weeks of lactation. However, the regression coefficients showed that the effect became larger as lactation progressed. Increasing average daily feed intake during week 1 by 1 kg increased the litter weaning weight by 0.33 kg; corresponding values for weeks 2 and 3 were 0.51 and 0.74 kg, respectively. This provides additional evidence that the relative importance of substrate supply for milk production gradually shifts from body reserves at farrowing toward current nutrient intake as lactation progresses.

## Energy and amino acid intake during lactation

### *Variation in lysine requirement*

The lactating sow will respond to an increasing supply of a given substrate by producing more milk only to the level at which some other factor limits milk production. This is shown clearly in estimates of the daily lysine requirement of lactating sows reported in the literature over a span of two decades. The estimates spanned a bewildering range from less than 20 to more than 50 g/d. However, 77% of this variation can be explained by variation among experiments in litter growth rate (Pettigrew, 1993). Results derived from regression analysis showed an increase in lysine requirement of 2.6 g/d (2.3 g ileal apparently digestible lysine/d) for each increment of 100 g/d in litter growth rate. Presumably, the milk production of sows with low lysine requirement was limited by some factor other than lysine intake, such as genetic capacity, litter size, health, or energy intake. This is entirely consistent with the observation by Tokach *et al.* (1992) that the lactating sow's response to daily lysine intake varies dramatically as metabolizable energy (ME) intake varies (Figure 3). The intake of ME and amino acids was varied separately over wide ranges. The responses shown in Figure 3 are derived from multiple regression equations that included linear and quadratic effects of ME intake and of lysine intake, and their linear interaction. Note that at the highest ME intake (69.0 MJ/d), the sow appears to respond to at least 45 g lysine/d. At the intermediate ME intake (48.1 MJ/d), milk yield was maximized at about 37 g lysine/d. At the low level of

ME intake (27.2 MJ/d), milk yield was maximized by only about 25 g lysine/d. When milk yield was limited by the energy supply, the sow was unable to respond to higher lysine intakes. Similarly, the sows increased milk production in response to increasing ME intake only when they consumed adequate lysine to support that increase.

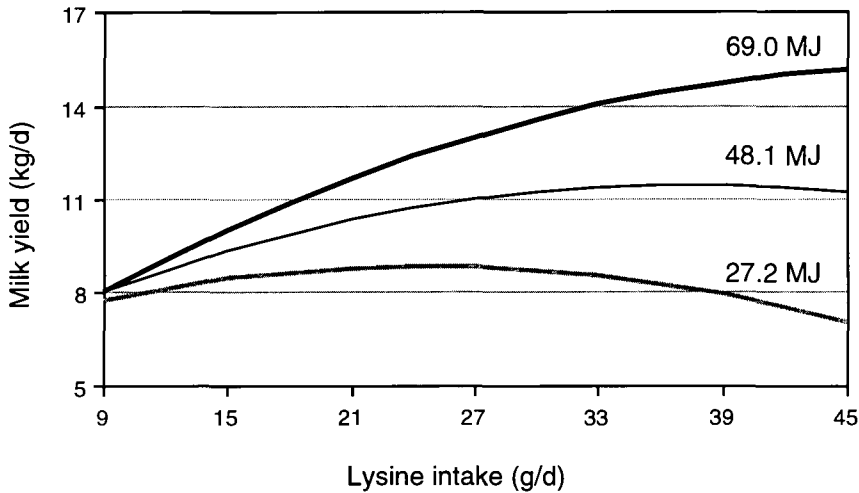


Figure 3. Influence of daily lysine and ME (MJ) intakes on milk yield on day 22 of lactation. Curves derived from multiple regression equation ( $r^2 = 0.74$ ). Adapted from Tokach *et al.* (1992) with permission.

#### Variation in milk fat concentration

There is a general connection between energy and amino acid requirements, because there is a general connection between their output in milk. However, fat concentration in milk can vary considerably, and independently from protein and lactose concentrations, in response to the diet. For example, addition of fat to the lactating sow's diet increases the fat concentration of the milk (Pettigrew, 1981). Sows restricted to a low level of energy intake had a higher fat concentration in milk, and actually produced more milk fat, than did sows on a higher energy intake (Noblet and Etienne, 1986). Increasing the dietary lysine concentration has also been reported to increase fat concentration of milk (Thaler *et al.*, 1992; King *et al.*, 1993). These three dietary manipulations all increase fat concentration of milk but appear so different, probably have a common metabolic mechanism. All three manipulations increase fat concentrations in serum, and it appears that the mammary glands are especially effective at taking up fatty acids from serum and putting them into milk fat. Sows consuming a high level of dietary fat should be expected to have high serum triglyceride levels. Those restricted to a low level of energy intake are in a strongly negative energy balance, which causes them to mobilize body fat. Fat mobilization is accompanied by an increase in circulating non-esterified fatty acid (NEFA) concentrations, and these NEFA are taken up by the mammary glands. When increasing the dietary lysine concentration increases milk yield, it forces the sow into a more strongly negative energy balance, again triggering an increase in serum NEFA concentrations.

## RELATIONSHIPS

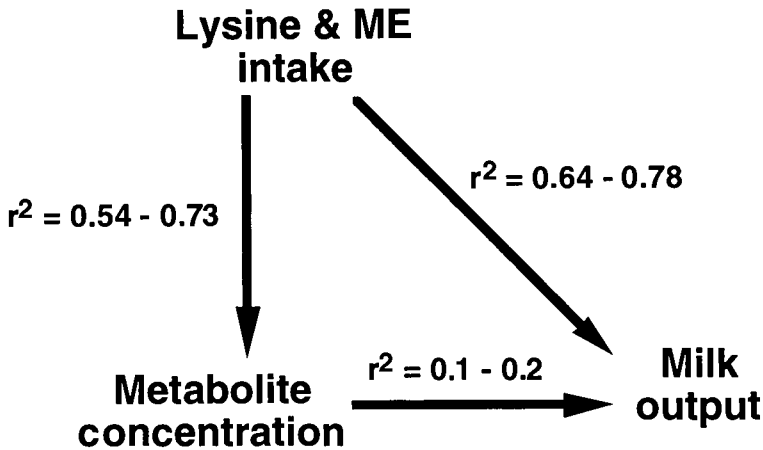


Figure 4. Coefficients of determination ( $r^2$ ) of multiple regressions among daily lysine and ME intake, daily production of milk components (fat, protein and lactose) and metabolite concentrations (glucose, nonesterified fatty acids, triglycerides and lysine). Reprinted from Pettigrew *et al.* (1993) with permission.

### Substrate concentrations

With these relationships in mind, Tokach *et al.* (1992) attempted to define quantitatively the relationships between production of milk components and either nutrient intake or circulating concentrations of substrates. They found strong relationships of intake of ME and lysine with production of milk fat, protein and lactose (Figure 4). In an attempt to define the mechanism through which nutrient intake affects milk production, strong relationships were found between both ME and lysine intake with circulating concentrations of substrates for milk components. However, there were essentially no relationships of substrate concentrations with the amounts of their products (milk fat, protein and lactose). This observation suggests that substrate concentrations are not the components of metabolic state that connect nutrient intake to milk production. However, Tokach *et al.* (1992) did find strong correlations of yield of milk components with serum insulin.

### Branched-chain amino acids

Recently, much attention has been directed to an apparent special role of the branched-chain amino acids, especially valine, but perhaps also isoleucine and leucine, in milk production. A thorough review by Boyd *et al.* (1995) suggested that these amino acids, unlike most others, are taken up by the mammary gland in larger quantities than they are secreted in milk proteins. Similar observations have been made in ruminants (Boyd *et al.*, 1995). The observation that the mammary gland does something with valine besides put it into milk protein is consistent with the fact that the lactating sow's dietary requirement for valine is higher relative to other amino acids when estimated empirically (NRC, 1988) than when estimated by a factorial approach that assumes the same efficiency of use of all amino acids by the mammary glands (ARC, 1981; Pettigrew, 1993).

Recent results of Trottier and Easter (1995) confirm the excess mammary uptake of some branched-chain amino acids, although they do not show it clearly in the case of valine. Their further work with the powerful technique of measuring uptake of substrates by the mammary gland using arterio-venous difference will likely contribute much to our understanding of nutrient needs of the lactating sow.



Three recent experiments have confirmed that the valine requirement of the high-producing lactating sow is higher than previously thought. First, litter weaning weights improved in response to increasing the dietary valine concentration from 0.60% to 0.90%, in a sow lactation diet containing 0.90% lysine (Tokach *et al.*, 1993). The valine effect was bigger in litters of more than 10 pigs weaned (64.1 vs 60.5 kg) than in smaller litters (57.1 vs 55.4 kg), emphasizing that the primary concern with valine is in sows producing a very high amount of milk. Second, a collaborative study between two universities evaluated graded levels of valine in diets containing 0.90% lysine (Richert *et al.*, 1994; Table 2). Litter weaning weight was maximized at 65.5 kg, at a dietary valine concentration of 1.15% (128% of the lysine concentration). Third, a collaborative study between two continents used a factorial arrangement of treatments, two lysine concentrations by three valine/lysine ratios (Richert *et al.*, 1995). Results (Table 3) show that high dietary levels of both lysine and valine are necessary to maximize litter growth when there is a high level of milk production, but not when milk production is lower.

**Table 2. Effect of valine concentration in sow diets on litter growth [(Richert *et al.*, 1994); total 203 litters (40 or 41 per treatment)].**

	Dietary valine (%)					CV (%)
	0.75	0.85	0.95	1.05	1.15	
Litter growth to 21 d (kg) <sup>1</sup>	46.9	47.1	48.3	49.5	49.6	13.9
Litter weaning weight at 21 d (kg) <sup>1</sup>	62.4	62.6	64.0	65.0	65.5	11.9

<sup>1</sup>Linear effect of valine,  $P < 0.05$ .

**Table 3. Effect of lysine and valine concentrations in sow diets on litter growth (kg) between days 2 and 24 of lactation (Richert *et al.*, 1995).**

Lysine (%)	0.8			1.2			CV (%)
	80	100	120	80	100	120	
Valine/lysine (%)							
≥ 10 pigs/litter <sup>1</sup>	47.5	46.0	49.7	51.3	49.8	55.7	14.5
< 10 pigs/litter <sup>2</sup>	41.1	42.3	41.4	40.5	45.3	39.8	17.5

<sup>1</sup>Lysine effect ( $P < 0.001$ ); valine effect (linear  $P < 0.05$ ; quadratic  $P < 0.05$ ). Range of 19 to 25 litters per treatment. <sup>2</sup>Range of 10 to 14 litters per treatment.

### Summary

Both the diet consumed during lactation and the sow's body reserves contribute to the enormous substrate supply required to support a high level of milk production. It is proposed that they interact through their effects on the sow's metabolic state. Body reserves are most important early in lactation, and the importance shifts to nutrient intake during lactation in late lactation. Nutrient requirements during lactation depend on the level of milk production. Milk fat concentration is increased by any nutritional manipulation that increases circulating concentrations of triglycerides or NEFA. Milk production is associated with levels of metabolic hormones, but not clearly to substrate concentrations. The mammary gland uses branched-chain amino acids, especially valine, for purposes other than to incorporate them into milk protein. Therefore, the dietary requirement for valine is higher than previously predicted.

## SOWS' MILK AS A MAJOR NUTRIENT SOURCE BEFORE WEANING

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### Optimum nutrient requirements for the piglet

There are two reasons for the renewed interest in the nutrient requirements of piglets in the first three to four weeks of life. First, faster growth during lactation means that piglets adapt better to solid diets and suffer less of a growth check at weaning and, subsequently, grow faster and reach slaughter weight sooner. Second, early weaning is again becoming popular, particularly in North America. Segregated early weaning systems involve weaning piglets at 10 to 14 days of age and rearing them in isolation from the sow with major benefits in the control of disease. Such benefits can only be realised if rapid growth rates are obtained and this depends on a precise knowledge of nutrient requirements for growth at that early stage.

A comprehensive study of nutrient requirements of piglets for protein (amino acids) and energy during the first three weeks of life was conducted twenty years ago (Williams, 1976). Cows' milk protein was fed to piglets from 1.8 to 6.4 kg at two levels of energy intake, 3.2 and 5.2 MJ GE/d or approximately 2.5 and 4.0 times maintenance (Figure 5). The response of nitrogen retention to nitrogen intake was similar at both levels of energy intake which suggests that nitrogen (protein/amino acids) requirements are a constant of energy intake. Calculating from the lower level of energy intake, the piglets required 12.0 g cows' milk protein per MJ DE assuming apparent digestibility of milk energy to be 0.96. This value can be adjusted for both digestibility and quality of protein so that the requirement becomes 10.0 g of "ideal" protein/MJ of DE. Taking the calculation a stage further and assuming that amino acids in milk are fully available after absorption then the piglet's tissue requirement for lysine was 0.7 g/MJ of DE.

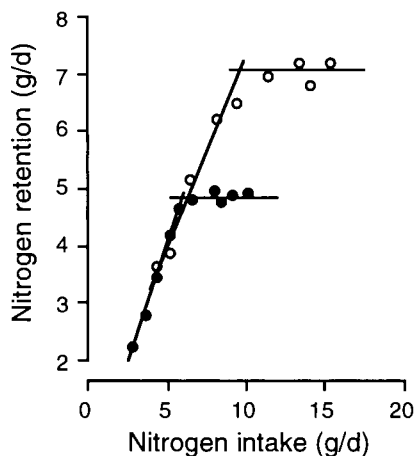


Figure 5. Two-phase response of nitrogen retention (NR) to nitrogen intake (NI) for piglets fed low (3.2 MJ GE/d; ●) or high (5.2 MJ GE/d; ○) levels of energy from 1.8 to 6.4 kg live-weight. Maximum NR was 7.03 g for the piglets fed high energy and 4.86 g for the piglets fed low energy. When NI was limiting NR the relationship was described by  $NR = 0.83NI - 0.17$  for the low levels of energy and  $NR = 0.72NI + 0.33$  for the high levels of energy (Williams, 1976).

The piglets in the study by Williams (1976) were representative of the genotypes in Australia at that time but they do not represent the fast-growing genotypes used today and, as Campbell and Taverner (1988) have shown, genotypes have changed substantially in the last 20 years. Modern strains are capable of much higher rates of maximum protein deposition, they retain proportionally more water in the fat-free empty body and they have higher maintenance requirements than conventional strains of twenty years ago. However, most if not all of these changes can be explained with the simple assumption that modern genotypes differ from traditional ones only in their body size. It follows that if comparisons are made at the same proportion of body size (similar degree of physiological maturity) then differences between genotypes disappear. If this simple explanation is correct it follows that the nutrient requirements of these modern genotypes if expressed relative to each other should be the same as that of the older genotypes. For example, the amount of ideal protein per MJ of DE should be the same for a large or small genotype but the absolute amounts of either energy or protein needed will be different; they will increase for the bigger genotype. D.E. Auldism and R.H. King (personal communication) have re-examined the protein (amino acid) requirements of baby pigs but have used a modern genotype which is very different to that of twenty years ago. Their estimate of lysine requirements for piglets between 2 and 7 kg live-weight was 0.74 g available lysine/MJ of DE and, in keeping with the hypothesis above, this suggests there has been little change in requirements despite substantial changes to genotype.

### Sows' milk and optimal nutrition for piglets

Sucking piglets usually grow at an average of 180 to 240 g/d between birth and weaning. If they are removed from the sow shortly after birth and fed cows' milk *ad libitum* their growth rate can double. The fastest growth rates ever recorded were those of Hodge (1974) who fed cows' whole milk to piglets *ad libitum* and measured an average daily gain of 576 g from 10 to 30 days of age.

Why do sow-suckled pigs grow so far below their potential? There are at least two reasons. First, the amount of milk produced by the sow limits the growth of the pig. Harrell *et al.* (1993) calculated that milk production becomes limiting to the sucking piglet at around 8 to 10 days of age and that the differences between need and supply progressively increases as lactation proceeds. These authors calculated that, at day 21 of lactation, the sow needs to produce in excess of 18 kg milk/d in order to supply piglets with enough energy to grow at rates comparable to artificially-reared piglets of the same age. Currently this rate of milk production far exceeds that of even the highest-producing sows in most herds.

Second, the potential for lean tissue growth in the baby pig is most likely limited by the composition of sows' milk *per se*. From an evolutionary viewpoint sows' milk is likely to have evolved to enhance the survival of the baby pig and hence perpetuation of the species. The piglet at birth is small in body size and has a large surface area to body-weight ratio relative to other farm animals. It has very small amounts of body lipid, generally between 1 and 2% of total body weight (Mellor and Cockburn, 1986), and so the glucose stores in its liver and the protein in its skeletal muscle represent the main energy stores that can be used to maintain body temperature. These energy stores are small which means that unless the piglet has frequent access to large amounts of dietary energy it will rapidly succumb to cold and become hypothermic and die. Sows' milk is well designed for the survival of piglets because it is high in fat and it is delivered at frequent intervals (once each hour) by the sow. It is not only rich in fat but it is also low in protein and, because of this relatively low protein to energy ratio, it encourages the piglet to deposit body fat. Much of this fat is deposited subcutaneously and can serve both as an energy store and an insulation layer. So sows' milk is designed to promote both the deposition of fat and lean rather than just lean tissue and this seems to be the case for at least the first 3 weeks of life. Apart from encouraging survival by building the energy stores of the piglet, diets which have less protein may also be an advantage in certain disease states. This is the case when piglets with diarrhoea, caused either directly by microorganisms or indirectly by indigestible nutrients reaching the large intestine, become dehydrated. In these circumstances high-protein diets can be lethal because excess protein is deaminated to urea, a diuretic, which will dehydrate the animal further.

How deficient in protein is sows' milk? Williams (1976) allowed piglets to be suckled by the sow or removed them at 2 days of age and artificially reared them on liquid milk diets. The diets, which ranged in protein concentration from 113 to 460 g crude protein/kg dietary dry matter, were offered at two levels of energy intake (3.2 or 5.2 MJ GE/d) from 1.8 to 6.4 kg live-weight. Both sow-suckled and artificially-reared piglets were slaughtered at 6.4 kg live-weight to measure body composition (Fig. 6).

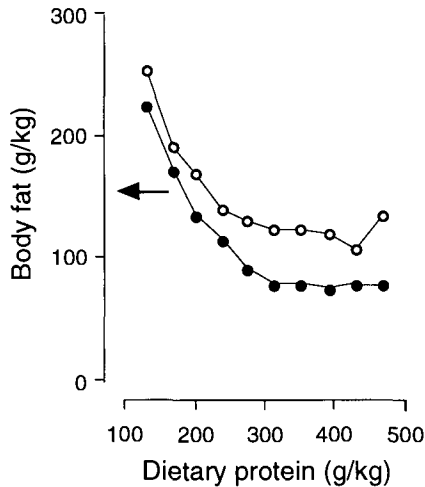


Figure 6. Body fat content at 6 kg empty body-weight in piglets fed various concentrations of protein at either 3.2 (●) or 5.2 (○) MJ GE/d from 1.8 to 6.4 kg live-weight. The arrow represents sow-suckled piglets slaughtered at the same empty body-weight (Williams, 1976).

At both energy intakes an increase in dietary protein reduced the amount of body fat in artificially-reared piglets and minimal body fat was induced with a diet containing about 300 g cows' milk protein/kg dry matter. The high body fat content (153 g fat/kg body-weight) of the sow-suckled pigs indicates that they had been consuming a diet with approximately 200 g protein/kg dry matter, much lower than that needed to promote minimum body fat and hence maximum lean gain. Although fat deposition probably aids the survival of piglets outdoors and may be a substantial reserve of energy at weaning, it is doubtful whether pigs need to deposit fat so early in life if raised in modern commercial facilities where the physical and, perhaps, the microbiological environment can be more closely controlled.

Although sows' milk may be deficient in protein relative to its energy content, the protein which it does contain is almost certainly used with very high efficiency. Because of the difficulty in obtaining sufficient quantities of milk from sows there have been very few direct measurements made on the value of sows' milk to piglets. Lucas and Lodge (1961) cite work from the Rowett where enough milk was collected to determine digestibility with two piglets. Both dry matter and nitrogen were digested completely: true digestibility was 1.0 and apparent digestibility was 0.97. The efficiency of use of sows' milk has only been measured indirectly because of the difficulty of obtaining sows' milk. Noblet and Etienne (1987) used gas exchange and comparative slaughter to determine the efficiency of use of sows' milk by piglets in the first 3 weeks of life. As anticipated milk was used with very high efficiency. The gross energy was retained with an efficiency of 0.55 and the nitrogen with a gross efficiency of 0.89. If endogenous loss and digestibility of nitrogen are taken into account the biological value of protein in sows' milk must be very close to 1.0. This is not surprising as the amino acid balance in sows' milk is very similar to that in the lean tissue of pigs (King *et al.*, 1993).

### Supplementary creep feeding and piglet weight at weaning

To supplement milk production, producers often provide piglets at 10 to 14 days of age with a 'creep' or 'starter' diet based predominately on cereals (eg., wheat, barley, oat groats) and various sources of animal and plant protein (eg., dried skim-milk powder, soyabean and spray-dried plasma protein). The suggestion is that piglets that consume more dry feed during lactation will be heavier at weaning, have a more advanced gastrointestinal tract, and hence be better able to handle the extreme dietary changes at weaning. Evidence to support this notion that supplying piglets with creep food during lactation will increase weaning weight is equivocal. Pluske *et al.* (1995a) have reviewed a number of experiments and calculated the contribution of creep food to daily energy intake (Table 4). This clearly shows that the intake of creep is generally small and very variable. Direct evidence for its value in stimulating voluntary food intake and live-weight gain after weaning is also limited.

**Table 4. Contribution of creep food to daily energy intake of the piglet during lactation (Pluske *et al.*, 1995a).**

	Duration of feeding (d)	Lactation length (d)	Creep food (g/piglet/d)	Piglet growth (g/d)	Contribution of creep to daily energy (%)
Okai <i>et al.</i> (1976)	11	28	3	167	1
	21	35	43	168	16
Aherne <i>et al.</i> (1982)	21	35	36	244	10
Barnett <i>et al.</i> (1989)	18	21	4	190	1
de Passillé <i>et al.</i> (1989)	11	21	17	180	5
Gatel and Guion (1990)	24	28	18	209	5
Pajor <i>et al.</i> (1991)	18	28	27	218	7
Appleby <i>et al.</i> (1992)	7	28	51	248	12
	7	28	77	261	17
Pluske (1993)	15	29	10	251	3

Supplementation of piglets during lactation with liquid diets offers the potential to provide a massive boost to piglet growth but, surprisingly, there have been very few attempts made to quantify the response. Reale (1987) offered cows' whole milk to piglets from 1000 h each day and added fresh milk every 2 h until 2300 h from 7 to 28 days of age. He stimulated growth by 151 g/d (70%) in the fourth week of lactation and, overall from 7 to 28 d, by 87 g/d which amounted to an extra 1.8 kg of live-weight at weaning; a response far in excess of that from dry diets (Table 5). It is worth pondering on why the piglets kept sucking from the sow when there was a better source of milk nearby. It suggests that the cues that arise from sow-piglet interactions still override the potential nutritional response.

**Table 5. Dry matter intakes of the supplement and growth of piglets offered dry creep food (control) or cows' whole milk while sucking the sow from 7 to 28 days of age (Reale, 1987).**

	Age (days)			
	7-14	14-21	21-28	7-28
Dry matter intake (g/d)				
Control	2	2	2	2
Cows' milk	18	57	149	74
Daily gain of piglets (g/d)				
Control	255	247	210	237
Cows' milk	277	335	361	324

## Manipulating the composition and yield of sows' milk

Piglet growth can be increased either by increasing the quantity of sows' milk produced or by increasing its concentration of protein. Can either of these be achieved?

Several attempts have been made to manipulate the protein content of sows' milk. Lodge (1959) fed sows 11, 15 and 19% crude protein during lactation and found no changes in protein concentration in their milk. Holden *et al.* (1968) fed sows 8, 12, 16 and 20% crude protein and found a slight increase in protein from 5.0 to 5.7%. A small increase from 5.4 to 5.7% was observed by Elliott *et al.* (1971) as they increased dietary crude protein from 5 to 15%. More recently, King *et al.* (1993) compared diets containing either 6 or 24% and, again, found a small, non significant increase from 5.3 to 5.9%. Several studies have been extended to the level of amino acids (Elliott *et al.*, 1971; Duee and Jung 1973; Dourmad *et al.*, 1991; King *et al.*, 1993) and it is noteworthy how constant the amino acid profile of sows' milk is and how small the variation is between various estimates.

The sow is well known for her ability to buffer milk production during lactation. If there is a deficiency in the diet she will mobilise her body reserves to cover the shortfall so perhaps it is not surprising that the composition of milk is largely immutable. Ranford *et al.* (1994) hypothesised that diet might become an important determinant of milk composition in situations when sows had low body reserves, in particular, low reserves of body protein. They tested this by manipulating gilts during gestation so that they started lactation with either high or low reserves of body protein. By offering a high- or low-protein diet during lactation the extremes were investigated, that is, either high or low endogenous supply of amino acids from body reserves and a high or low exogenous supply of amino acids from the diet. Although milk production was reduced in those sows with low body protein fed a low-protein diet during lactation the hypothesis was rejected because the changes in milk composition were small. Sows with the smallest mass of body protein and fed the low-protein diet (7.8% crude protein) during lactation produced milk with 4.1% protein while, at the other extreme, sows with high body protein and fed the high-protein (19.0% crude protein) diet produced milk with only slightly higher protein content (4.9%). The conclusion is that the proportion of protein in milk as well as its amino acid composition remains remarkably stable despite large differences in either the dietary or endogenous supply of precursors.

Although the composition of milk, particularly its protein content, remains fairly constant the total output of milk can vary substantially depending on a number of factors; the protein and energy supply in the diet and the endogenous supply of precursors or the body reserves of animals. The effect of body reserves on milk production is well illustrated by the data of Mullan and Williams (1989) who manipulated gilts during gestation so that body reserves (fat or lean or both) at the beginning of lactation were either high, medium, or low. Half the gilts were then restricted in energy intake (to about 24 MJ DE/d) during lactation and forced to use their body reserves to produce milk. As expected the gilts with higher body reserves produced more milk as measured by the growth of their piglets. As maternal energy intake was increased during lactation to *ad libitum* intake, the partition of dietary energy between maternal needs and those of milk production remained the same irrespective of the level of body reserves (Figure 7). This was contrary to expectation because it was thought that the lower the body reserves the greater the impetus would be to replace them when nutrition allowed. The strategy adopted by the gilts with low body reserves was to increase their voluntary food intake above that of the gilts with high reserves. The increase in intake was sufficient to bring their milk output to the same level as the gilts with the higher body reserves.

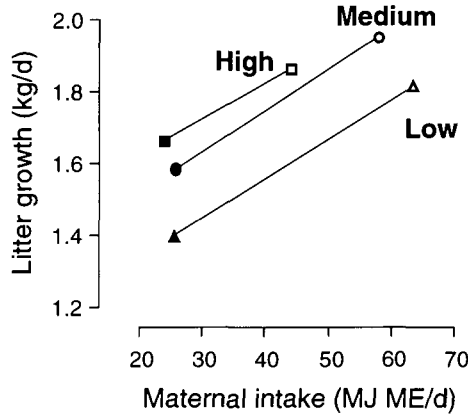


Figure 7. Litter growth and maternal energy intake of gilts with high, medium or low body reserves at the start of lactation. Closed symbols are restricted energy intake and open symbols represent ad libitum energy intake (Mullan and Williams, 1989).

Milk production is well buffered by body reserves as already discussed, but it still responds to dietary energy during lactation. There have been few studies designed specifically to determine the relationship between milk production and dietary energy and the studies that are available often have treatments spread over a narrow range of dietary intakes. Shown in Figure 8 are the results of a number of studies and they show that the response of milk output to energy intake is variable reflecting indirect measures of milk output (measured by piglet growth) and factors such as the weight of sows, their parity and body reserves, and the protein content of diets fed during lactation. Nevertheless, these data indicate that for maternal energy intakes between 20 and 70 MJ ME/d during lactation litter growth responds by about 9 g/MJ ME and, since most of these estimates are from gilts with 8 to 9 piglets/litter it suggests that each sucking piglet grows an extra 1 g/d for each MJ of ME consumed by the mother.

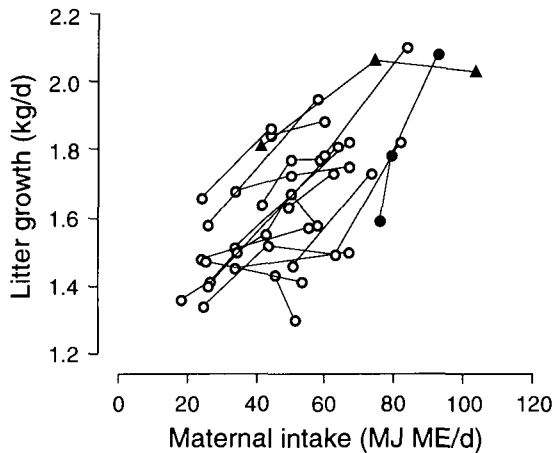


Figure 8. Litter growth and maternal energy intake for sows fed conventionally (open circles) (Reese et al., 1982; Nelssen et al., 1985; Johnston et al., 1986; Noblet and Etienne, 1987; Eastham et al., 1988; King and Dunkin, 1986; King and Williams, 1984ab; Mullan and Williams, 1989; Ranford et al., 1994) or via a stomach cannula to achieve a higher energy intake than ad libitum (Matzat et al., 1990 (●); Pluske et al., 1995b (▲)).

Most of these estimates have been obtained from gilts or sows fed in a conventional way from a trough and, in these situations, *ad libitum* intake, at least for gilts, is generally below 70 MJ of ME/d (open circles, Figure 8). There are two recent studies (closed symbols) in which animals were fed through a stomach cannula which allowed the normal mechanisms that limit food intake to be overridden and much higher intake than normal to be achieved. In one of these studies Pluske *et al.* (1995b) fed gilts 42, 75 or 104 MJ ME/d and demonstrated a ceiling to milk production because piglet growth did not respond beyond 75 MJ ME. The extra 29 MJ ME/d that these super-alimented gilts received went not into milk but into body-weight gain; they gained 12 kg of body-weight and 1.7 mm backfat while the gilts receiving 75 MJ ME/d lost 14 kg of body-weight and 3.7 mm backfat. The second study was conducted by Matzat *et al.* (1990) who super-alimented sows through stomach cannulae and showed a linear relationship between milk output and maternal energy intake. This suggests that gilts and sows might partition energy differently during lactation. There might be hormonal mechanisms in actively growing gilts that are designed to protect the maternal growth processes. It is more likely that the ceiling in milk output reflects nothing more complex than a shortage of secretory cells in the mammary tissue of gilts.

The composition of milk is particularly stable and it seems most unlikely that it can be manipulated through altering the body reserves of gilts or sows or changing the supply of either protein and energy in the diet. Milk output is responsive to dietary manipulation either directly during lactation or indirectly through the influence of previous nutritional history on body reserves of animals commencing lactation. The circumstantial evidence is that gilts have a ceiling to milk output and, if reached, do not respond further to nutrition whereas sows show a linear response of milk output to feed intake during lactation.



## PIGLETS' ROLE IN DETERMINING MILK PRODUCTION IN THE SOW

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Sow milk yield responds to stimuli such as litter size, piglet size and nursing demand. This paper discusses the attributes of the litter which influence the level of milk production in sows.

### The influence of litter size on milk yield

It is well established that total milk yield increases linearly with litter size although authors differ as to the magnitude of the response. The response to litter size is mainly as a result of an increased number of functional mammary glands, although milk yield can differ per gland. Elsley (1971) compiled four earlier studies which investigated the response of milk yield to litter sizes ranging from 4 to 12 piglets (Figure 9) and found a strong linear relationship between litter size and milk yield;  $Y = 0.58 X + 1.81$ , where  $Y$  = milk yield (kg/d) and  $X$  = litter size.

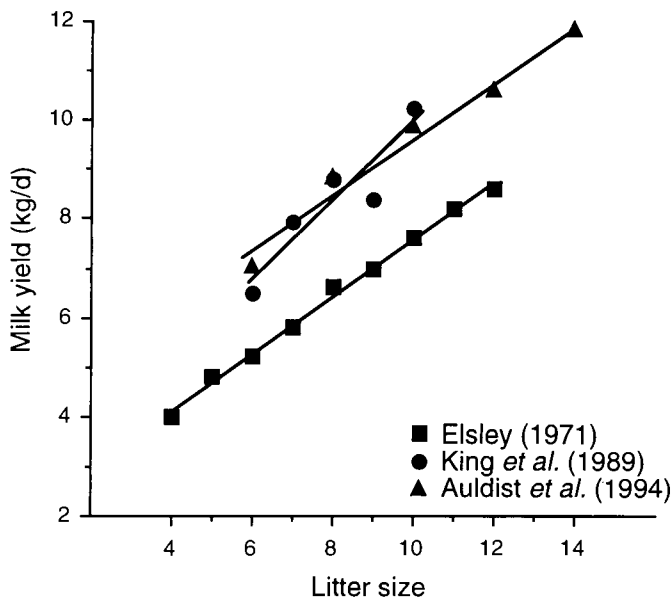


Figure 9. The effect of litter size on milk production of sows.

Later, King *et al.* (1989) and Auldust *et al.* (1994) reported similar slopes for the relationship between milk yield and litter size (Figure 9) but the relationships predicted approximately 2 kg/d greater milk yield than that reported by Elsley (1971). Improved milk yield over the past two decades may have resulted from genetic improvement through indirect selection for milk production by selection for growth rate and/or weaning weights of litters. Auldust *et al.* (1994) measured average litter daily gains of 2.84 kg/d for litters of 14 piglets. Using the relationship between milk consumption and piglet growth rate defined by King *et al.* (1989), this is equivalent to over 12 L milk/d. This

level of milk production is much greater than that generally assumed to be the average milk yield of sows (ARC, 1981) and is also likely to be nearing the biological limit as sows lost 23 kg live-weight and 8.8 mm backfat over 28 days of lactation despite consuming over 5 kg feed each day. The sows mobilized considerable amounts of body tissues in order to satisfy the requirements for high milk production. Sauber *et al.* (1994) imposed a high nursing demand on lactating sows in an attempt to attain maximum milk production during lactation. The size of each litter was twice the number of functional mammary glands of the sow. Litters were divided into two groups with each group allowed access to the sow at 40 min intervals for 18 h daily. For the remaining 6 h each day only one of the litter groups was allowed continuous access to the sow. Sows had an average group size of between 14 and 17 pigs which were suckled every 40 mins. Based upon average milk composition (Prawirodigdo, 1989) and energy content of the milk components (McDonald *et al.*, 1966), the sows produced between 10.4 and 12.1 kg/d. From these results it appears that, although sucking demand was markedly increased, Sauber *et al.* (1994) may still not have achieved maximum lactation capacity of the sows. Other factors such as dietary nutrient intake and maternal body reserves may have limited milk yield.

The strong relationship between milk yield and litter size results in an inverse relationship between litter size and individual milk consumption and consequently individual piglet growth rates. Elsley (1971) reported that milk consumption by piglets declined from 1 kg/d in litters of four to 0.7 kg/d in litters of 12, whereas Auldist *et al.* (1994) found that individual milk consumption decreased from 1.3 kg/d in a litter of six to 0.9 kg/d in a litter of 14. Milk consumption per piglet increases with decreasing litter size because of increased gland size (Auldist *et al.*, 1995, Carlson, 1995) and decreased competition from litter mates; individual piglets from small litters often stimulate and have access to more than one gland (Fraser, 1990).

### The influence of piglet size on milk yield

It is clear that the number of piglets is a major factor which determines milk yield but the size or weight of the piglets may also play a role. Hartman *et al.* (1962) were first to report a positive association between birth weight and milk intake of the piglet. They suggested that heavier piglets may be more efficient at draining the teats than lighter piglets and therefore may stimulate a greater subsequent milk flow. The strong positive relationship between birth weight and subsequent milk intake and growth rate of the sucking piglet has also been reported more recently by Fraser and Morley Jones (1975) and Hemsworth *et al.* (1976). Fraser (1984) suggested that a larger pig may massage the teat before ejection, more vigorously, thus achieving a greater blood flow to the gland and thereby altering the flow of hormones and metabolites to the gland. Measurements by Algiers and Jensen (1991) showed that the duration and intensity of teat stimulation influenced the milk production of that teat during the first days of lactation.

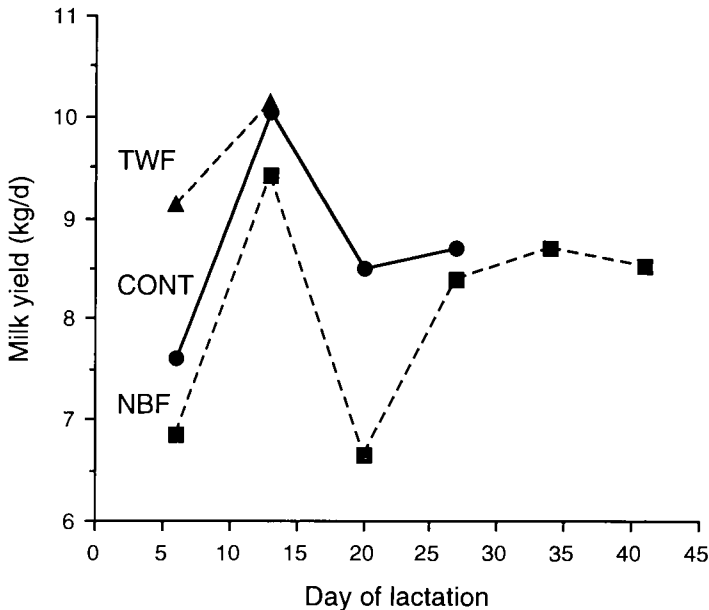
Whatever the mechanism of increased milk production, the most convincing evidence of the relationship between piglet body-weight and milk intake is provided by the experiment of Van der Steen and de Groot (1992). Half of the litters of Chinese Meishan (CM) and Large White x Landrace (LWL) sows were cross fostered within 1-2 days of farrowing to produce litters of approximately half CM piglets and half LWL piglets, each suckled by both types of sows. The experimental design resulted in four groups of piglets (Table 6). The heavier LWL piglets consumed similar amounts of milk and grew at similar rates irrespective of the type of sow they were suckled by. The lighter CM piglets failed to consume similar amounts of milk as the LWL piglets despite being suckled by the potentially high-producing LWL sows. In addition, light LWL piglets and heavy CM piglets, which had similar birth weights, had the same growth rate prior to weaning. Thus body-weight at birth has a substantial effect on milk intake and subsequent growth rate during the pre-weaning period. The experiment of Van der Steen and de Groot (1992) provides sufficient evidence to hypothesise that the demand of piglets for milk during lactation has a direct effect on milk yield.

**Table 6. Performance of piglets suckled by Chinese Meishan (CM) and Large White  $\times$  Landrace (LWL) sows (Van der Steen and de Groot, 1992).**

Sows	CM		LWL	
	CM	LWL	CM	LWL
Piglet birthweight (kg)	0.89	1.38	0.89	1.38
Piglet growth rate (g/d)	164	191	154	205
Average milk intake (g/d)	684	852	612	924

In the sow lactation program at the Victorian Institute of Animal Science, Werribee, a cross-fostering experiment was conducted to determine the effect of piglet weight on sows' milk production. Thirty sows were allocated at their first farrowing to three experimental treatments; control foster (CF), newborn foster (NBF) or two week foster (TWF). Within each experimental block, CF and NBF sows farrowed on the same day and the TWF sows farrowed  $15.0 \pm 0.3$  d later. Litter size was standardised to nine piglets by fostering within 1 d of farrowing. Piglets from TWF sows were allowed to suck colostrum from their natural mothers. At  $1.7 \pm 0.3$  d after TWF sows farrowed, there was a complete swap made of litters between each of the three treatments; litters suckled by NBF sows were transferred to CF sows, litters suckled by CF sows were transferred to newly farrowed TWF sows and the relatively newborn litters suckled by TWF sows were transferred to NBF sows.

All piglets were weaned at about 4 weeks of age resulting in lactation lengths for CF, NBF and TWF sows of  $28.9 \pm 0.4$ ,  $42.5 \pm 0.8$  and  $15.0 \pm 0.4$  d respectively. Milk yield for each sow was estimated by the deuterium oxide dilution method (King *et al.*, 1993) over 4-day periods on up to six occasions during lactation. These periods commenced at approximately day 4, 11, 18, 25, 32 and 39 of lactation.



**Figure 10.** The effect of fostering treatment on milk production of sows. The TWF sows received two week old piglets at parturition, NBF sows received newborn piglets at two weeks into lactation, and CONT sows received contemporary litters at two weeks into lactation.

Fostering two-week old piglets on to newly farrowed sows increased milk production of those sows by 26% during the first week of lactation (Figure 10). Conversely fostering newborn piglets on to sows in their third week of lactation reduced subsequent milk production by 22% during the third week of lactation (Figure 10). The results of this cross-fostering experiment support the view that piglet weight is an important controlling factor in determining milk intake and consequently sows' milk production. The results support the general hypothesis that sows' milk yield is primarily affected by the demand for milk by piglets during early lactation. As lactation proceeds the supply of substrates, governed by nutrient intake of the sow and the availability of body reserves, becomes increasingly important in determining milk production.

### The influence of suckling interval and duration of letdown on milk yield

Suckling frequency can be a major constraint to milk consumption by piglets and therefore limit pre-weaning growth. Suckling frequency is influenced by factors such as litter size and stage of lactation, but it may also be manipulated so that intervals are shorter thereby providing more opportunities for piglets to obtain milk.

The effect of litter size on suckling frequency was examined in an experiment described by Auldist *et al.* (1994). Litters of 6, 8, 10, 12 or 14 piglets were established by fostering within 36 h post-partum. Suckling frequency of sows was monitored by video recording for 24 h in early (day 10) and late (day 24) lactation. The video recordings were later examined and successful sucklings were recorded by observing the behaviour and auditory stimuli of the sow and her litter. Suckling interval was shorter in early lactation than in late lactation (48.2 min v 52.2 min,  $P < 0.05$ ) which is similar to the findings of Han and Park (1984). The average observed intervals were longer than the 44 min reported by Ellendorf and Poulain (1984) but within their reported range of 21 - 92 min.

The period of milk flow during milk ejection in the sow is very short and lasts for only 10 to 20 seconds (Ellendorf *et al.*, 1982). It can be detected by a rise and fall in intra-mammary pressure (Smith 1994). It is possible that the milk intake of piglets at a suckling could be limited by their ability to withdraw milk over this short period rather than by the amount of milk available in the gland. It may be expected that sows with longer periods of milk flow at milk ejection would produce more milk per gland than those with shorter periods of milk flow. The duration of the rise in intra-mammary pressure was measured by Auldist *et al.* (1994) and found to be shorter in late lactation than in early lactation (12.8 sec vs 14.6 sec,  $P < 0.05$ ). The period that milk is being withdrawn from the teat is less in late lactation because of the shorter duration of letdown and the less frequent numbers of sucklings. However, estimated milk yield was similar in early and late lactation, which suggests that the larger pigs in late lactation have a greater sucking strength.

The influence of suckling interval was examined in the study by Auldist *et al.* (1995) in which the effect of increased nursing demand on suckling frequency was measured. Litters of six or 12 piglets were established soon after parturition. Excess teats on the sows were taped until they regressed so that only one functional teat was available per piglet. A third treatment called "cross-suckle" was also established by day 6 of lactation. In the "cross-suckle" treatment each sow had six functional teats but two groups of six piglets. From day 6 to 27 of lactation, these groups were alternated every 30 min to be with the sow. The "cross-suckle" treatment therefore imposed a higher sucking demand per teat than the other two treatments because the two groups of piglets allowed the sow the opportunity to letdown every 30 min. The suckling interval was shorter for sows allocated to the "cross suckle" treatment (Table 7). Preliminary results also show that gland weight and estimated milk yield/gland of sows with the cross suckled litters were twice that of sows with a litter of 12. The average daily gain of piglets from day 10 to day 27 did not differ between those piglets in a litter of 12 and those in the "cross suckle" litters (208 and 181 g/d respectively) whereas piglets in the litter of six grew much faster (308 g/d).

**Table 7. Suckling interval (min) in early (day 10) and late (day 24) lactation for sows with litters of six, twelve and cross-suckle piglets (Auldust *et al.*, 1995).**

Litter size	Stage of lactation		Mean
	Early	Late	
Six	44.1	47.0	45.5 <sup>b</sup>
Twelve	43.5	52.3	47.9 <sup>b</sup>
Cross suckle	34.8	42.3	38.6 <sup>a</sup>
Mean	40.8 <sup>a</sup>	47.2 <sup>b</sup>	-

<sup>ab</sup>Means with different superscripts are significantly different ( $P < 0.05$ ).

From the above results, it is apparent that suckling interval is an important determinant of milk production. The suckling interval of lactating sows may be altered by environmental factors such as photoperiod and auditory stimulus. Mabry *et al.* (1983) studied the effects of photoperiod on the suckling frequency of sows during a lactation period of 28 days. The mean suckling interval of sows exposed to an 8 h/d photoperiod was 60 min whereas those sows exposed to an extended photoperiod of 16 h/d suckled every 50 min. These results are in agreement with those of Niwa *et al.* (1951) who reported shorter suckling intervals during daytime compared to suckling intervals during the night (64.8 vs 70.7 min). The increased suckling frequency observed during exposure to extended photoperiod resulted in greater milk production and litter growth rate (Mabry *et al.*, 1983).

Suckling frequency may also be manipulated by auditory stimuli. Stone *et al.* (1974) reported an increase in suckling frequency in response to playing recorded nursing sounds of sows suckling their litters during lactation. The response of treated sows was less evident in late lactation but nevertheless milk production of the treated sows was higher and their litters were heavier at weaning. However not all noises in the farrowing shed can be regarded as stimulatory to suckling frequency and milk yield. Algers and Jensen (1985) reported that loud continuous noise from fans can mask the grunts of the sow prior to suckling. If the litter doesn't respond to the sow's grunts sucking and letdown is disrupted, resulting in lower milk yields.

### Conclusions

From the results of studies reviewed here it is evident that milk production in the sow is responsive to stimuli such as litter size, piglet live-weight and sucking demand. It is through manipulation of these and other stimuli that the sow's lactation performance may be improved and piglet growth rates increased.

## GENERAL SUMMARY - CONSTRAINTS TO PRE-WEANING GROWTH

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### Introduction

The preceding papers display the richness of our current understanding of the factors affecting milk production by sows and pre-weaning growth of their piglets, but they also reveal frustrating gaps in our knowledge and our understanding of these factors, as well as an apparent incongruity.

Data were cited showing that piglets are capable of growing much faster when artificially reared than they do when nursing a sow, leading to the suggestion that the amount of milk produced by the sow is the chief factor limiting piglet growth. However, it was also emphasized that the sow responds to greater demands for milk by piglets (more pigs or larger pigs) by increasing milk production. This suggests that the piglets themselves put a limit on their growth rate. A clearer understanding of the interaction between the sow and the litter is required. The sow responds to greater nursing stimulus and/or greater milk removal by increasing milk synthesis. Perhaps it is the magnitude of her response that ultimately limits piglet growth. It would be useful to know if her response is limited by neural, endocrine or nutritional/metabolic factors.

It appears that significant improvements in litter weaning weights have been achieved during the past one to two decades. It is suggested that factors contributing to this apparent increase in weaning weights (pre-weaning growth rates) include genetic selection, improved health from greater attention to farm quarantine procedures and sanitation, more appropriate thermal environments, and more adequate nutrient intake. Further attention to these factors is likely to produce further increases in pre-weaning growth.

### Importance of increasing pre-weaning growth

Increasing pre-weaning growth rates of pigs is a worthwhile objective. But what is the benefit to the pork production enterprise of increasing pre-weaning growth rates? Quantitative estimates are needed of the effect of interventions to increase pre-weaning growth on post-weaning performance, including rates of protein and fat accretion all the way to market weight. Some pigs grow faster during the pre-weaning period than others, including their littermates, for unknown reasons. Perhaps those same reasons contribute to their greater subsequent growth rate. A clear demonstration that intervention to increase pre-weaning growth will also increase subsequent growth rate is required.

In all stages of production, the direction and magnitude of subsequent (carry-over) effects of nutritional interventions are important but not well understood. There has been some attention given to the impact of the quality of starter diets on performance during the growing-finishing period (Pettigrew and Stairs, 1991). It is clear that a moderate restriction of energy and/or amino acids results in a classical compensatory response, in which slow-growing pigs grow faster than controls when restored to an adequate diet. However, when pigs grow slowly because of inappropriate dietary ingredients (maize and soya bean meal with no milk, fish or blood products), they do not compensate and often continue to grow more slowly during later stages. Pettigrew and Stairs (1991) have proposed that the important factor is not early growth rate, but the physiological conditions that accompany that growth rate.

What are the physiological conditions that accompany differences in growth rate of pre-weaning pigs? If pre-weaning growth is limited by intake of energy and/or amino acids, would it not be reasonable to expect classical compensatory growth after weaning, when the nutrient intake restriction is released? On the other hand, if greater pre-weaning growth is accompanied by greater pancreatic weight and pancreatic enzyme secretion, it may prepare the pigs for greater post-weaning growth (Mahan and Lepine, 1991). What

other physiological conditions may be involved? Could greater pre-weaning growth rate be accompanied by a sustained anabolic condition (eg., higher IGF-I levels in serum) that persists after weaning?

Again, increasing pre-weaning growth rates of pigs is worthwhile. However, there is a need for quantitative estimates of the effect of interventions to increase pre-weaning growth on post-weaning performance. The magnitude of response is important in economic decisions. For example, the increase in weaning weight that results from increasing the valine level in the sow's diet (Pettigrew, 1995) is rather small, perhaps about 300 g/pig. Knowledge of the economic value of that increase is required in order to intelligently decide whether to increase the dietary valine level, which may also increase diet cost.

Also knowledge about whether the value of increased pre-weaning growth varies with weaning age is required. This is of special interest now, with the rapid adoption of segregated early weaning (SEW) and related technologies.

### Prospects for increasing pre-weaning growth

As noted above, the industry appears to have made impressive improvements in pre-weaning growth. The best prospects for further increasing pre-weaning growth are simply continuations of the improvements that have been made to date.

#### *Genetic selection*

It appears that something has been done in genetic selection that has improved pre-weaning growth. It is not clear whether that improvement has come from direct selection for pre-weaning growth (milk production) or as a correlated response to selection for leanness or lean growth rate. The observation of Sauber *et al.* (1994) that sows from a line selected for lean growth produced more milk than those from a control line suggests that such correlated responses may be important. Perhaps there are common physiological conditions that contribute to both lean growth and milk production, such as high circulating concentrations of growth hormone or IGF-I.

#### *Health*

Improving the health of starter pigs through SEW and related technologies has produced astounding improvements in growth rate (Walker and Wiseman, 1994). Improving the health of grower-finisher pigs through all in-all out production systems and attention to sanitation has also produced impressive increases in growth (Williams, 1994). Perhaps the industry's adoption of all in-all out animal flow in farrowing rooms, coupled with increased focus on farm quarantine procedures and sanitation, has contributed to increased pre-weaning growth. It is likely that further attention to these factors will bring further improvements in the future.

#### *Thermal stress*

Maintaining separate and appropriate micro-environments for the sow and her nursing litter has always been a challenge for pork producers, but progress has been made. If piglets are cold, they are unlikely to nurse vigorously, so various methods of heating the piglets' zone are now used to good effect. If sows are hot, they reduce their feed intake and that may reduce their milk production. In addition, results of recent Australian research (Black *et al.*, 1993; M.L. Lorsch *et al.*, personal communication) show that heat stress also has a direct effect on milk production, reducing production even when feed intake is equalized.

In practice, it is less expensive to prevent cold stress than to prevent heat stress, so the biggest remaining challenge is to provide cooling during hot weather. Drip cooling reduces heat stress in lactating sows, and should be used in all climates in which pigs are raised. Use of flooring materials that conduct heat from the sow can also help. However, in hot climates these currently available technologies are inadequate, and more effective ones are required.

### Sow diets

Improvements in pre-weaning growth, regardless of the underlying causes, can be realized only if the sows are provided adequate nutrients. When potential milk production is increased, the nutrient requirements to reach that potential are also increased.

There are two challenges in providing adequate nutrients to lactating sows. The first is ensuring adequate feed intake; the second is proper formulation of the diet.

Energy supply is controlled largely by feed intake. Intake should be encouraged by management interventions including control of ambient temperature, providing feeders that facilitate eating by the sow, ensuring adequate water flow, and feeding frequently. Allowing sows to mix feed with water also increases intake (Koketsu *et al.*, 1994). Digestible energy intake should not be reduced by use of fibrous ingredients. Increasing dietary energy density by incorporation of fat increases pre-weaning growth of the litter, but the effect is small. The effect of fat supplementation on subsequent reproductive performance is still not known.

Dietary amino acid levels must be much higher than suggested by NRC (1988) in order to achieve high rates of pre-weaning growth. Actual levels vary with the rate of potential litter growth (Pettigrew, 1993).

From a broader perspective, nutrient intake by the lactating sow influences not only the pre-weaning growth rate of the litter, but also subsequent reproductive performance of the sow. The nutrient requirements of the sow for milk production can be estimated using classical input-output calculations (Pettigrew, 1993). However, there remains a question of whether requirements to maximize subsequent reproduction are higher. These requirements cannot be estimated from input-output relationships. Recent Australian data (Campbell, 1995) show that very high dietary concentrations of amino acids are needed to maximize subsequent litter size, but it is not clear that the levels necessary to maximize reproduction are greater than those needed to maximize litter growth.

### Summary

There is a need to understand why some evidence suggests that pre-weaning growth is limited by the sow, whereas other evidence suggests it is limited by the nursing litter. Quantitative estimates of the effect of interventions to increase pre-weaning growth on post-weaning performance are required. There are good prospects for further increases in pre-weaning growth rate by continued attention to genetic selection, health, the thermal environment, and sow diets.

### References

- AGRICULTURAL RESEARCH COUNCIL (1981). "Nutrient Requirements of Pigs". (Commonwealth Agricultural Bureau: Slough, UK).
- AHERNE, F.X., DANIELSEN, V. and NIELSEN, H.E. (1982). The effects of creep feeding on pre- and post-weaning pig performance. *Acta Agriculturae Scandinavica*. 32:155-160.
- ALGERS, B. and JENSEN, P. (1985). Communication during suckling in the domestic pig. Effects of continuous noise. *Applied Animal Behaviour Science*. 14:49-61.
- ALGERS, G. and JENSEN, P. (1991). Teat stimulation and milk production in early lactation of sows. *Canadian Journal of Animal Science*. 71:51-60.
- APPLEBY, M.C., PAJOR, E.A. and FRASER, D. (1992). Individual variation in feeding and growth of piglets: Effects of increased access to creep food. *Animal Production*. 55:147-152.
- ASH, R.W. and HEAP, R.B. (1973). The induction and synchronization of parturition in sows treated with I.C.I. 79,939, an analogue of prostaglandin F<sub>2a</sub>. *Journal of Agricultural Science*. 81:365-368.
- ATKINSON, S.A., SCHNURR, C.M., DONOVAN, S.M. and LONNERDAL, B. (1989). The non-protein nitrogen components in human milk: Biochemistry and potential functional role. In: "Protein and non-protein nitrogen in human milk", pp. 117-133, eds S.A. Atkinson and B. Lonnerdal. (CRC Press: Florida).
- ATWOOD, C.S. (1993). The measurement of fat, protein, lactose and cellular metabolites in milk for the assessment of lactocyte function during the lactation cycle of the sow. PhD Thesis. The University of Western Australia.
- ATWOOD, C.S. and HARTMANN, P.E. (1992). Collection of fore- and hind-milk from the sow. *Journal of Dairy Research*. 59:287-298.
- ATWOOD, C.S. and HARTMANN, P.E. (1993). The concentration of fat, protein and lactose in sows' colostrum from suckled and unsuckled glands during lactogenesis II. *Australian Journal of Agricultural Research*. 44:1457-1465.



- ATWOOD, C.S., TOUSSAINT, J.K. and HARTMANN, P.E. (1995). The assessment of mammary gland metabolism in the sow: Cellular metabolites in the mammary secretion and plasma during lactogenesis II. *Journal of Dairy Research*. 62:207-220.
- AULDIST, D.E., CARLSON, D., MORRISH, L., WAKEFORD, C. and KING, R.H. (1995). Effect of increased suckling frequency on mammary development and milk yield of sows. In "Manipulating Pig Production V", p. 137, eds D.H. Hennessy and P.D. Cranwell. (Australasian Pig Science Association: Werribee).
- AULDIST, D.E., MORRISH, L., THOMPSON, M. and KING, R.H. (1994). Response of sows to varying litter size. *Proceedings of the Nutrition Society of Australia*. 18:175.
- BALDWIN, R.L. and LOUIS, S. (1975). Hormonal actions on mammary metabolism. *Journal of Dairy Science*. 58:1033-1043.
- BARNETT, K.L., KORNEGAY, E.T., RISLEY, C.R. LINDEMANN, M.D. and SCHURIG, G.G. (1989). Characterization of creep feed consumption and its subsequent effects on immune response. *Journal of Animal Science*. 67:2698-2708.
- BLACK, J.L., MULLAN, B.P., LORSCHY, M.L. and GILES, L.R. (1993). Lactation in the sow during heat stress. *Livestock Production Science*. 35:153-170.
- BOYD, R.D., KENSINGER, R.S., HARRELL, R.J. and BAUMAN, D.E. (1995). Nutrient uptake and endocrine regulation of milk synthesis by mammary tissue of lactating sows. *Journal of Animal Science*. 73 (Supplement 1): (In press).
- CAMPBELL, R.G. (1995). Lactation sow nutrition - impact on subsequent reproductive performance. In supplement, Seminar Session 10, "Management and Nutrition of the Lactating Sow." *Proceedings of the 26th Annual Meeting of the American Association of Swine Practitioners*, March 4-7, Omaha, Nebraska.
- CAMPBELL, R.G. and TAVERNER, M.R. (1988). Genotype and sex effects on the relationship between energy intake and protein deposition in growing pigs. *Journal of Animal Science*. 66:676-686.
- CARLSON, D. (1995). Effect of suckling demand on milk yield. BAgSc (Hons) Thesis. Royal Veterinary and Agricultural University, Copenhagen, Denmark.
- CHARD, T. (1974). Radioimmunoassay of oxytocin. In: "Methods of Hormone Radioimmunoassay", pp. 209-241, eds B.M. Jaffe and H.R. Behrman. (Academic Press: New York).
- COMBER, M.F. and HARTMANN, P.E. (1993). Preliminary examination of sow's milk by nuclear magnetic resonance. In "Manipulating Pig Production IV", p. 263, ed. E.S. Batterham. (Australasian Pig Science Association: Attwood).
- COWIE, A.T., FORSYTH, I.A. and HART, T.C. (1980). Lactation. In: "Hormonal control of Lactation." *Monographs of Endocrinology*. 15:144-229, eds F. Gross, A. Labhart, T. Mann and J. Zander. (Springer-Verlag: New York).
- CROSS, B.A., GOODWIN, R.F.W. and SILVER, I.A. (1958). A histological and functional study of the mammary gland in normal and agalactic sows. *Journal of Endocrinology*. 17:63-74.
- DALE, H.H. (1909). The action of extracts of the pituitary body. *Biochemical Journal*. 4:427-447.
- de PASSILLE, A.M.B., PELLETIER, G., MÉNARD, J. and MORISSET, J. (1989). Relationships of weight gain and behaviour to digestive organ weight and enzyme activities in piglets. *Journal of Animal Science*. 67:2921-2929.
- de PASSILLE, A.M.B., RUSHEN, J., FOXCROFT, G.R., AHERNE, F.X. and SCHAEFER, A. (1993). Performance of young pigs: Relationships with periparturient progesterone, prolactin and insulin of sows. *Journal of Animal Science*. 71:179-184.
- DIEHL, J.R., GODKE, R.A., KILLIAN, D.B. and DAY, B.N. (1974). Induction of parturition in swine with prostaglandin F<sub>2α</sub>. *Journal of Animal Science*. 38:1229-1234.
- DODD, S.C., FORSYTH, I.A., BUTTLE, H.L., GURR, M.I. and DILS, R.R. (1994). Milk whey proteins in plasma of sows: variation with physiological state. *Journal of Dairy Research*. 61:21-34.
- DOURMAD, J.Y., ETIENNE, M. and NOBLET, J. (1991). A contribution to the study of amino acid requirements for lactation in sows. *Journées de Recherche Porcine en France*. 23:61-68.
- DUEE, P.H. and JUNG, J. (1973). Amino acid composition of sow's milk. *Annales Zootechnie*. 22:243-247.
- EASTHAM, P.R., SMITH, W.C., WHITTEMORE, C.T. and PHILLIPS, P. (1988). Responses of lactating sows to food level. *Animal Production*. 46:71-77.
- ELLENDORFF, F., FORSLING, M.L. and POULAIN, D.A. (1982). The milk ejection reflex in the pig. *Journal of Physiology*. 333:577-594.
- ELLENDORFF, F. and POULAIN, D. (1984). A means to assess nursing efficiency in the pig: the study of the milk ejection reflex. *Annales de Recherches Veterinaires*. 15:271-274.
- ELLENDORFF, F., TAVERNE, M., ELSAESSER, F., FORSLING, M., PARVIZI, N., NAAKGEBOREN, C. and SMIDT, D. (1979). Endocrinology of parturition in the pig. *Animal Reproduction Science*. 2:323-334.
- ELLIOTT, R.F., VANDER NOOT, G.W., GILBREATH, R.L. and FISHER, H. (1971). Effect of dietary protein level on composition changes in sow colostrum and milk. *Journal of Animal Science*. 32:1128-1137.
- ELSLEY, F.W.H. (1970). Nutrition and lactation in the sow. In "Lactation", pp 393 - 411, ed. I. R. Falconer. (Butterworths: London).
- FAHMY, M.H. (1972). Comparative study of colostrum and milk composition of seven breeds of swine. *Canadian Journal of Animal Science*. 52:621-627.
- FERGUSON, J.K.W. (1941). A study of the motility of the intact uterus at term. *Surgery, Gynaecology and Obstetrics*. 73:359-366.
- FOLLEY, S.J. (1969). The milk-ejection reflex: a neuroendocrine theme in biology, myth and art. *Proceedings of the Society for Endocrinology*. 44:476-490.
- FORSLING, M.L., TAVERNE, M.A.M., PARVIZI, N., ELSAESSER, F., SMIDT, D. and ELLENDORFF, F. (1979). Plasma oxytocin and steroid concentrations during late pregnancy, parturition and lactation in the miniature pig. *Journal of Endocrinology*. 82:61-69.
- FORSYTH, I.A. (1986). Variation among species in the endocrine control of mammary growth and function: The roles of prolactin, growth hormone, and placental lactogen. *Journal of Dairy Science*. 69:886-903.
- FRASER, D. (1984). The role of behaviour in swine production: A review of research. *Applied Animal Ethology*. 11:317-339.
- FRASER, D. (1990). Behavioural perspectives on piglet survival. In "Control of Pig Reproduction III", pp. 355-370, eds D.J.A. Cole, G.R. Foxcroft and B.J. Weir. (Journal of Reproduction and Fertility Ltd: Essex).
- FRASER, D. and MORLEY JONES, R. (1975). The "teat order" of suckling pigs. 1. Relation to birth weight and subsequent growth. *Journal of Agricultural Science*. 84:387-391.

- FUCHS, A.R. (1985). Oxytocin in Animal Parturition. In: "Oxytocin: Clinical and Laboratory Studies", pp. 207-235, eds J.A. Amico and A.G. Robinson. (Elsevier Science Publishers: New York).
- GATEL, F. and GUION, P. (1990). Effects of monosodium L glutamate on diet palatability and piglet performance during the suckling and weaning periods. *Animal Production*. 50:365-372.
- GILBERT, C.L., GOODE, J.A. and MCGRATH, T.J. (1994). Pulsatile secretion of oxytocin during parturition in the pig: temporal relationship with fetal expulsion. *Journal of Physiology*. 475:129-137.
- GLENCROSS, B.D., TOUSSAINT, J.K., TUCKEY, R.C. and HARTMANN, P.E. (1994). Progesterone withdrawal, plasma lactose concentrations and piglet performance: Is there a relationship? *Proceedings of the Australian Society for Reproductive Biology*. 26:79.
- GOONERATNE, A., HARTMANN, P.E., MCCAULEY, I. and MARTIN, C.E. (1979). Control of parturition in the sow using progesterone and prostaglandin. *Australian Journal of Biological Science*. 32:587-595.
- GOONERATNE, A.D., HARTMANN, P.E. and NOTTAGE, H.M. (1982). The initiation of lactation in sows and the mastitis-metritis-agalactia syndrome. *Animal Reproduction Science*. 5:135-140.
- HAN, S.W. and PARK, C.S. (1984). Studies of the patterns of nursing, rest and sleep in the lactating sow. *Research Reports of Agricultural Science and Technology*. 11:2.
- HARRELL, R.J., THOMAS, M.J. and BOYD, R.D. (1993). Limitations of sow milk yield on baby pig growth. *Proceedings of the 1993 Cornell Nutrition Conference for Feed Manufacturers*, pp. 156-164. (Cornell University: Ithaca, NY)
- HARTMAN, D.A., LUDWICK, T.M. and WILSON, R.F. (1962). Certain aspects of lactation performance in sows. *Journal of Animal Science*. 21:883-886.
- HARTMANN, P.E., ATWOOD, C.S., COX, D.B. and DALY, S.E.J. (1995). Endocrine and autocrine strategies for the control of lactation in women and sows. Hannah Research Institute Conference "Intracellular Signalling in the Mammary Gland", pp. 203-225, eds C. Wilde, D.H. Knight and M. Peaker. (Plenum Publishing Company: New York)
- HARTMANN, P.E. and HOLMES, M.A. (1989). Sow lactation. In "Manipulating Pig Production II". pp. 72-97, eds J.L. Barnett and D.P. Hennessy. (Australasian Pig Science Association: Werribee).
- HARTMANN, P.E., TREVENTHAN, P. and SHELDON, J. (1973). Progesterone and oestrogen and the initiation of lactation in the ewe. *Journal of Endocrinology*. 59:249-259.
- HARTMANN, P.E., WHITELEY, J.L. and WILLCOX, D.L. (1984). Lactose in plasma during lactogenesis, established lactation and weaning in sows. *Journal of Physiology*. 347:453-463.
- HEAD, R.H., BRUCE, N.W. and WILLIAMS, I.H. (1991). More cells might lead to more milk. In "Manipulating Pig Production III", p. 76, ed. E.S. Batterham. (Australasian Pig Science Association: Attwood).
- HEAD, R.H. and WILLIAMS, I.H. (1991). Mammogenesis is influenced by pregnancy nutrition. In "Manipulating Pig Production III", p. 33, ed. E.S. Batterham. (Australasian Pig Science Association: Attwood).
- HEESOM, K.J., SOUZA, P.F.A., ILIC, V. and WILLIAMSON, D.H. (1992). Chain-length dependency of interactions of medium-chain fatty acids with glucose metabolism in acini isolated from lactating rat mammary glands. *Biochemical Journal*. 281:273-278.
- HEMSWORTH, P.H., WINFIELD, C.G. and MULLANEY, P.D. (1976). Within litter variation in the performance of piglets to three weeks of age. *Animal Production*. 22:351-357.
- HODGE, R.M.W. (1974). Efficiency of food conversion and body composition of the preruminant lamb and the young pig. *British Journal of Nutrition*. 32:113-126.
- HOLDEN, P.J., LUCAS, E.W., SPEER, V.C. and HAYS, V.W. (1968). Effect of protein level during pregnancy and lactation on reproductive performance in swine. *Journal of Animal Science*. 27:1587-1590.
- HOLMES, M.A. (1991). Biochemical investigations into milk secretion and milk removal in the lactating pig. PhD Thesis. The University of Western Australia.
- HUGHES, P.E. and VARLEY, M.A. (1980). Lactation. In: "Reproduction in the Pig", pp. 136-168, eds P.E. Hughes and M.A. Varley. (Butterworths: London).
- JOHNSTON, L.J., ORR, D.E., TRIBBLE, L.F. and CLARK, J.R. (1986). Effect of lactation and rebreeding phase energy intake on primiparous and multiparous sow performance. *Journal of Animal Science*. 63:804-814.
- KENDRICK, K.M., KEVERNE, E.B., HINTON, M.R. and GOODE, J.A. (1991). Cerebrospinal fluid and plasma concentrations of oxytocin and vasopressin during parturition and vaginocervical stimulation in the sheep. *Brain Research Bulletin*. 26:127-141.
- KENSINGER, R.S., COLLIER, R.J. and BAZER, F.W. (1986). Ultrastructural changes in porcine mammary tissue during lactogenesis. *Journal of Anatomy*. 145:49-59.
- KENSINGER, R.S., COLLIER, R.J., BAZER, F.W., DUCSAY, C.A. and BECKER, H.N. (1982). Nucleic acid, metabolic and histological changes in gilt mammary tissue during pregnancy and lactogenesis. *Journal of Animal Science*. 54:1297-1308.
- KEVERNE, E.B. (1988). Central mechanisms underlying the neural and neuroendocrine determinants of maternal behaviour. *Psychoneuroendocrinology*. 13:127-141.
- KING, R.H. and DUNKIN, A.C. (1986). The effect of nutrition on the reproductive performance of first-litter sows. 3. The response to graded increases in food intake during lactation. *Animal Production*. 42:119-125.
- KING, R.H., RAYNER, C.J. and KERR, M. (1993). A note on the composition of sow's milk. *Animal Production*. 57:500-502.
- KING, R.H., TONER, M.S. and DOVE, H. (1989). Pattern of milk production in sows. In "Manipulating Pig Production II", p. 98, eds J.L. Barnett and D.P. Hennessy. (Australasian Pig Science Association: Werribee).
- KING, R.H., TONER, M.S., DOVE, H., ATWOOD, C.S. and BROWN, W.G. (1993). The response of first-litter sows to dietary protein level during lactation. *Journal of Animal Science*. 71:2457-2463.
- KING, R.H. and WILLIAMS, I.H. (1984a). The effect of nutrition on the reproductive performance of first-litter sows. 1. Feeding level during lactation, and between weaning and mating. *Animal Production*. 38:241-247.
- KING, R.H. and WILLIAMS, I.H. (1984b). The effect of nutrition on the reproductive performance of first-litter sows. 2. Protein and energy intakes during lactation. *Animal Production*. 38:249-256.
- KNIGHT, C.H. and PEAKER, M. (1982). Development of the mammary gland. *Journal of Reproduction and Fertility*. 65:521-536.

- KOKETSU, Y. (1994). Influence of feed intake and other factors on the lactational and postweaning reproductive performance of sows. PhD Thesis. University of Minnesota.
- KOKETSU, Y., DIAL, G.D., MARSH, W.E., PETTIGREW, J.E. AND KING, V.L. (1994). A field survey of the effects of equipment and genotype on lactation feed intake in commercial swine herds. *Journal of Animal Science*. 72 (Supplement 1):333.
- KUHN, N.J. and LOWENSTEIN, J.M. (1967). Lactogenesis in the rat: Changes in metabolic parameters at parturition. *Biochemical Journal*. 105:995-1002.
- KULSKI, J.K., SMITH, M. and HARTMANN, P.E. (1977). Perinatal concentrations of progesterone, lactose and  $\alpha$ -lactalbumin in the mammary secretion of women. *Journal of Endocrinology*. 74:509-510.
- KUSINA, J., PETTIGREW, J.E., SOWER, A.F., CROOKER, B.A., WHITE, M.E., HATHAWAY, M.R. and DIAL, G.D. (1995). The effect of protein (lysine) intake during gestation and lactation on lactational performance of the primiparous sow. *Journal of Animal Science*. 73 (Supplement 1):85.
- LODGE, G.A. (1959). The energy requirements of lactating sows and the influence of level of food intake upon milk production and reproduction and reproductive performance. *Journal of Agricultural Science*. 53:177-191.
- LUCAS, I.A.M. and LODGE, G.A. (1961). "The Nutrition of the Young Pig". Technical Communication No. 22. (Commonwealth Bureau of Animal Nutrition: Farnham Royal, Slough).
- MABRY, J.W., COFFEY, M.T. and SEERLEY, R.W. (1983). A comparison of an 8- versus 16-hour photoperiod during lactation on suckling frequency of the baby pig and maternal performance of the sow. *Journal of Animal Science*. 57:292-295.
- MAHAN, D.C. AND LEPINE, A.J. (1991). Effect of pig weaning weight and associated nursery feeding programs on subsequent performance to 105 kilograms body weight. *Journal of Animal Science*. 69:1370-1378.
- MAO, F.C. and BREMEL, R.D. (1995). Prediction of milk yields by serum  $\beta$ -lactoglobulin concentrations in pregnant heifers. *Journal of Dairy Science*. 78:284-295.
- MARTIN, C.E., HARTMANN, P.E. and GOONERATNE, A. (1978). Progesterone and corticosteroids in the initiation of lactation in the sow. *Australian Journal of Biological Science*. 31:517-525.
- MARTYN, P. and HANSEN, I.A. (1980). Initiation of fatty acid synthesis in rat mammary glands. *Biochemistry Journal*. 198:187-192.
- MATZAT, P.D., HOGBERG, M.G., FOGWELL, R.L. and MILLER, E.R. (1990). Lactation performance in high producing sows fed in excess of ad libitum. In "Report of Swine Research", AS-SW-8904, pp. 36-40. (Michigan State University).
- McDONALD, P., EDWARDS, R.A. and GREENHALGH, J.F.D. (1966). "Animal Nutrition". (Oliver and Boyd: Edinburgh).
- MELLOR, D.J. (1986). A comparison of energy metabolism in the newborn infant, piglet and lamb. *Quarterly Journal of Experimental Physiology*. 71:361-379.
- MOLENAAR, A.J., DAVIS, S.R. and WILKINS, R.J. (1992). Expressions of  $\alpha$ -lactalbumin,  $\alpha$ -S1-casein and lactoferrin genes is heterogeneous in sheep and cattle mammary tissue. *Journal of Histochemistry and Cytochemistry*. 40:611-618.
- MULLAN, B.P. and WILLIAMS, I.H. (1989). The effect of body reserves at farrowing on the reproductive performance of first-litter sows. *Animal Production*. 48:449-457.
- MURPHY, G., ARIYANAYAGAN, A.D. and KUHN, N.J. (1973). Progesterone and the metabolic control of the lactose biosynthetic pathway during lactogenesis in the rat. *Biochemical Journal*. 136:1105-1116.
- NARA, B.S. and FIRST, N.L. (1981). Effect of indomethacin and prostaglandin F-2 $\alpha$  on parturition in swine. *Journal of Animal Science*. 52:1360-1370.
- NARA, B.S., WELK, F.A., RUTHERFORD, J.E., SHERWOOD, O.D. and FIRST, N.L. (1982). Effect of relaxin on parturition and frequency of live births in pigs. *Journal of Reproduction and Fertility*. 66:359-365.
- NELSEN, J.L., LEWIS, A.J., PEO, Jr., E.R. and CRENSHAW, J.D. (1985). Effect of dietary energy intake during lactation on performance of primiparous sows and their litters. *Journal of Animal Science*. 61:1164-1171.
- NIWA, T., SUKEYUKI, I., YOKOYAMA, H. and OTSUKA, M. (1951). Studies on the milk secretion of the sow. I. On the habits of nursing, milk yield and the constituents of milk, etc. of the sow. *Bulletin of the Japan National Institute of Animal Industry*. 1:149-150.
- NOBLET, J. and ETIENNE, M. (1986). Effect of energy level in lactating sows on yield and composition of milk and nutrient balance of piglets. *Journal of Animal Science*. 63:1888-1896.
- NOBLET, J. and ETIENNE, M. (1987). Metabolic utilization of energy and maintenance requirements in lactating sows. *Journal of Animal Science*. 64:774-781.
- NATIONAL RESEARCH COUNCIL (1988). "Nutrient Requirements of Swine", 9th revised edn (National Academy Press: Washington, D.C.).
- OKAI, D.B., AHERNE, F.X. and HARDIN, R.T. (1976). Effects of creep and starter composition on feed intake and performance of young pigs. *Canadian Journal of Animal Science*. 56:573-586.
- OWENS, F.N., DUBESKI, P. and HANSON, C.F. (1993). Factors that alter the growth and development of ruminants. *Journal of Animal Science*. 71:3138-3150.
- PAJOR, E.A., FRASER, D. and KRAMER, D.L. (1991). Consumption of solid food by suckling pigs: individual variation and relation to weight gain. *Applied Animal Behavioural Science*. 32:139-151.
- PEAKER, M. and WILDE, C.J. (1987). Milk secretion: Autocrine control. *News In Physiological Science*. 2:124-126.
- PEDERSEN, C.A. and PRANGE, A.J.(Jr) (1979). Induction of maternal behaviour in virgin rats after intracerebroventricular administration of oxytocin. *Proceedings of the National Academy of Science USA*. 76:6661-6665.
- PETTIGREW, J.E. (1981). Supplemental dietary fat for periparturient sows: A review. *Journal of Animal Science*. 53:107-117.
- PETTIGREW, J.E. (1993). Amino acid nutrition of gestating and lactating sows. "Biokyowa Technical Review - 5" (Nutri-Quest Inc.: St Louis, USA).
- PETTIGREW, J.E. (1995). The influence of substrate supply on milk production in the sow. In "Manipulating Pig Production V", pp. 101-106, eds D.P. Hennessy and P.D. Cranwell. (Australasian Pig Science Association: Werribee).

- PETTIGREW, J.E., McNAMARA, J.P., TOKACH, M.D., KING, R.H. and CROOKER, B.A. (1993). Metabolic connections between nutrient intake and lactational performance in the sow. *Livestock Production Science*. 35:137-152.
- PETTIGREW, J.E. and STAIRS, J.T.F. (1991). The impact of starter diets on the subsequent growth of swine. Proceedings of the 52nd Minnesota Nutrition Conference, September 16-18, 1991, Bloomington, Minnesota, p. 163.
- PLAUT, K.I., KENSINGER, R.S., GRIEL, L.C. and KAVANAUGHT, J.F. (1989). Relationships among prolactin binding, prolactin concentrations in plasma and metabolic activity of the porcine mammary gland. *Journal of Animal Science*. 67:1509-1519.
- PLUSKE, J.R. (1993). Psychological and nutritional stress in pigs at weaning: Production parameters, the stress response, and histology and biochemistry of the small intestine. PhD Thesis. The University of Western Australia.
- PLUSKE, J.R., WILLIAMS, I.H. and AHERNE, F.X. (1995b). Nutrition of the piglet. In "The Neonatal Pig: Development and Survival", pp. 187-235, ed. M.A. Varley. (CAB International: Wallingford).
- PLUSKE, J.R., WILLIAMS, I.H., CEGIELSKI, A.C., CLOWES, E.C., ZAK, L.J. and AHERNE, F.X. (1995a). Super-alimentation of first litter sows during lactation. In "Manipulating Pig Production V", p.129, eds D.P. Hennessy and P.D. Cranwell. (Australasian Pig Science Association: Werribee).
- PRAWIRODIGDO, S. (1989). Techniques for estimating milk production by sows. MAgRSc Thesis. The University of Melbourne.
- RANFORD, J.L., REVELL, D.K., MULLAN, B.P., WILLIAMS, I.H. and TOUSSAINT, J.K. (1994). Milk yield, but not milk composition, may be influenced by body fatness in primiparous sows. *Journal of Animal Science*. 72 (Supplement 1):389.
- REALE, T.A. (1987). Supplemental liquid diets and feed flavours for young pigs. MAgRSc Thesis. The University of Melbourne.
- REESE, D.E., MÖSER, B.D., PEO, Jr., E.R., LEWIS, A.J., ZIMMERMAN, D.R., KINDER, J.E. and STROUP, W.W. (1982). Influence of energy intake during lactation on the interval from weaning to first estrus in sows. *Journal of Animal Science*. 55:590-598.
- RENNER, E. (1989). "Micronutrients in Milk and Milk-Based Food Products". (Elsevier Applied Science: New York).
- RICHERT, B.T., GOODBAND, R.D., TOKACH, M.D. and NELSEN, J.L. (1995). Valine and lysine independently improve sow productivity during lactation. *Journal of Animal Science*. 73 (Supplement 1):85.
- RICHERT, B.T., GOODBAND, R.D., TOKACH, M.D., NELSEN, J.L., JOHNSTON, L.J., WALKER, R.D., PETTIGREW, J.E. and BLUM, S.A. (1994). Determining the valine requirement of high producing sows. *Journal of Animal Science*. 72 (Supplement 1):389.
- ROBERTS, J.S. and SHARE, L. (1968). Oxytocin in plasma of pregnant, lactating and cycling ewes during vaginal stimulation. *Endocrinology*. 83:272-278.
- ROBERTSON, H.A. and KING, G.J. (1974). Plasma concentrations of progesterone, oestrone, oestradiol-17 $\beta$  and of oestrone sulphate in the pig at implantation, during pregnancy and at parturition. *Journal of Reproduction and Fertility*. 40:133-141.
- SAAKE, R.G. and HEALD, C.W. (1974). Cytological aspects of milk formation and secretion. In "Lactation: A Comprehensive Treatise", Volume 2, pp. 147-186, eds B.L. Larson and V.R. Smith. (Academic Press: New York).
- SAUBER, T.E., STAHLY, T.S., EWAN, R.E. and WILLIAMS, N.H. (1995). Maximum lactational capacity of sows with a moderate and low genetic capacity for lean tissue growth. Swine Research Report 1994, pp 60-62. (Iowa State University).
- SMITH, N.A. (1994). Biochemical and physiological investigations of parturition and lactation in the pig. PhD Thesis. The University of Western Australia.
- STONE, C.C., BROWN, M.S. and WARING, G.H. (1974). An ethological means to improve swine production. *Journal of Animal Science*. 39:137.
- THALER, R.C., WOERMAN, R.L. and BRITZMAN, D.B. (1992). Effect of lysine level in lactation diets on sow performance and milk composition. *Journal of Animal Science*. 70 (Supplement 1):238.
- THRELFALL, W.R., DALE, H.E. and MARTIN, C.E. (1974). Porcine blood and hypophyseal prolactin values. *American Journal of Veterinary Research*. 35:1491-1493.
- TOKACH, M.D., GOODBAND, R.D., NELSEN, J.L. and KATS, L.J. (1993). Valine - a deficient amino acid in high lysine diets for the lactating sow. *Journal of Animal Science*. 71 (Supplement 1):68.
- TOKACH, M.D., PETTIGREW, J.E., CROOKER, B.A., DIAL, G.D. and SOWER, A.F. (1992). Quantitative influence of lysine and energy intake on yield of milk components in the primiparous sow. *Journal of Animal Science*. 70:1864-1872.
- TONER, M.S., KING, R.H., DOVE, H., HARTMANN, P.E. and ATWOOD, C. (1991). The effect of growth hormone on milk production in first litter sows. In "Recent Advances in Animal Nutrition in Australia, 1991", p.23A, ed. D.J. Farrell. (University of New England: Armidale).
- TRITTON, S.M., KING, R.H., CAMPBELL, R.G. and EDWARDS, A.C. (1993). The effects of dietary protein on the lactation performance of first-litter sows. In "Manipulating Pig Production IV", p.265, ed. E.S. Batterham. (Australasian Pig Science Association: Attwood).
- TROTTIER, N.L. and EASTER, R.A. (1995). Daily amino acid uptake by the mammary gland in the lactating sow: A new approach for estimating amino acid requirements. *Journal of Animal Science*. 73 (Supplement 1):85.
- TURNER, C.W. (1952). "The Mammary Gland. I. The Anatomy of the Udder of Cattle and Domestic Animals." (Lucas Brothers:Columbia, Missouri).
- van der STEEN, H.A.M. and de GROOT, P.N. (1992). Direct and maternal breed effects on growth and milk intake of piglets: Meishan versus Dutch breeds. *Livestock Production Science*. 30:361-373.
- van LANDEGHEM, A.A.J. and van de WIEL, D.F.M. (1978). Radio-immunoassay for porcine prolactin: Plasma levels during lactation, suckling and weaning and after TRH administration. *Acta Endocrinologica*. 88:653-667.
- VERNON, R.G. (1989). Endocrine control of metabolic adaptation during lactation. *Proceedings of the Nutrition Society*. 48:23P.

- WALKER, N. (1977). The effects of induction of parturition in sows using an analogue of prostaglandin F<sub>2α</sub>. *Journal of Agricultural Science*. **89**:267-271.
- WALKER, R.D. and WISEMAN, B.S. (1994). Growth performance of segregated early weaned (SEW) pigs compared to their conventionally weaned littermates. *Journal of Animal Science*. **72** (Supplement 1):377.
- WELDON, W.A., THULIN, A.J., MACDOUGALD, O.A., JOHNSTON, L.J., MILLER, E.R. and TUCKER, H.A. (1991). Effects of increased dietary energy and protein during late gestation on mammary development in gilts. *Journal of Animal Science*. **69**:194-200.
- WHITELY, J.L. (1989). The endocrine control of the initiation of parturition and lactation in the sow. PhD Thesis. The University of Western Australia.
- WHITELY, J.L., HARTMANN, P.E., WILLCOX, D.L., BRYANT-GREENWOOD, G.D. and GREENWOOD, F.C. (1990). Initiation of parturition and lactation in the sow: Effects of delaying parturition with medroxyprogesterone acetate. *Journal of Endocrinology*. **124**:475-484.
- WHITTEMORE, C. (1993). "The Science and Practice of Pig Production." (Longman Scientific and Technical:Essex).
- WHITTLESTONE, W.G. (1954a). Intramammary pressure changes in the lactating sow. I. The effect of oxytocin. *Journal of Dairy Research*. **21**:19-30.
- WHITTLESTONE, W.G. (1954b). Intramammary pressure changes in the lactating sow. II. The effects of vasopressin and acetylcholine. *Journal of Dairy Research*. **21**:183-187.
- WILLCOX, D.L., ARTHUR, P.G., HARTMANN, P.E. and WHITELY, J.L. (1983). Perinatal changes in plasma oestradiol-17β, cortisol and progesterone and the initiation of lactation in sows. *Australian Journal of Biological Science*. **36**:173-181.
- WILLIAMS, I.H. (1976). Nutrition of the young pig in relation to body composition. PhD Thesis. University of Melbourne.
- WILLIAMS, N.S. (1994). Impact of genetic and environmental factors on the growth of the young pig. Proceedings of the 25th Annual Meeting, American Association of Swine Practitioners, pp. 185-190.

## THE VOLUNTARY FOOD INTAKE OF LACTATING SOWS IN FIVE COMMERCIAL HERDS

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Voluntary food intake (VFI) during lactation is often low and is usually insufficient to meet the metabolic demands of lactation and maintenance. Tritton *et al.* (1993) reported that first-litter sows in a large commercial herd in NSW consumed an average of 4.47 kg/d during a 23-day lactation. However there is a lack of information on VFI of lactating sows among commercial farms. The aim of this study was to measure the VFI of lactating sows in commercial herds.

The VFI of lactating sows at five farms in southern Queensland was monitored between August 1994 and April 1995. All sows were based on the "Hyfarm" genotype and were offered their diets at least twice daily during the 3 to 4 week lactation periods. The diets offered during lactation were all cereal-based with meat and bone meal and soya bean meal as the major sources of protein, and contained a minimum of 13.8 MJ DE/kg, 160 g CP/kg and 7 g available lysine/kg. Calibrated buckets were used to supply the diets and intakes at each feeding were recorded in 0.5 kg increments on individual food intake cards. Feed was offered as a dry mash and intake was adjusted on a daily basis depending upon disappearance of feed from the feed trough. The average lactation length ranged from 23.5 to 27.6 d among farms.

In addition to the significant effect of farm on VFI during lactation, first-litter sows consumed significantly less feed than older sows (5.46 vs 6.19 kg/d;  $P < 0.001$ ).

**Table 1. Voluntary food intake (kg/d) of lactating sows in five commercial herds.**

Farm	A	B	C	D	E	Overall
First-litter sows Mean	6.19 (13) <sup>1</sup>	4.27 (12)	5.24 (49)	5.32 (43)	6.31 (7)	5.46 (124)
SE	0.20	0.20	0.10	0.11	0.27	0.07
Older sows Mean	6.66 (40)	5.53 (39)	6.36 (64)	5.84 (63)	6.52 (60)	6.19 (274)
SE	0.10	0.14	0.09	0.09	0.09	0.04

<sup>1</sup> Figures in parenthesis are the number of sows.

Average VFI was approximately 20% higher in this study compared to the report by Tritton *et al.* (1993). Whittemore and Morgan (1990) suggested that daily intakes of at least 5 kg were necessary to ensure that milk production of sows is not adversely affected by lactation feeding strategy. The results of this study suggest that the majority of sows on these farms consumed sufficient amounts of nutrients during lactation to support optimum milk production and reproductive performance under the range of environmental and management conditions.

### References

- TRITTON, S.M., KING, R.H., CAMPBELL, R.G. and EDWARDS, A.C. (1993). In "Manipulating Pig Production IV", p. 265, ed. E.S. Batterham. (Australasian Pig Science Association: Attwood).  
 WHITTEMORE, C.T. and MORGAN, C.A. (1990). *Livestock Production Science*. 26:1-37.

## BODY FATNESS REDUCES VOLUNTARY FEED INTAKE AND ALTERS PLASMA METABOLITES DURING LACTATION

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The more a sow eats during pregnancy, the less she eats during lactation. Low voluntary feed intake (VFI) during lactation appears to be linked to the body fatness of the sow (Williams and Smits, 1991) and may be due to specific metabolic signals that limit VFI during lactation. The hypothesis for the following experiment was that the plasma concentrations of non-esterified fatty acids (NEFA), glycerol and insulin increase with body fatness, and that there is a negative relationship between VFI during lactation and the plasma concentrations of these metabolites.

During gestation, Landrace × Large White gilts were fed either 2.3 kg/d of a low-protein diet (5.8% CP, 14.6 MJ DE/kg) or 1.7 kg/d of a high-protein diet (15.6% CP, 14.5 MJ DE/kg) to produce fat or lean animals of similar weights at farrowing. Immediately after farrowing, fat sows (n=19) weighed  $157 \pm 2.3$  kg (mean ± SE) with  $24.8 \pm 0.76$  mm backfat, while lean sows (n=19) weighed  $152 \pm 2.0$  kg with  $17.9 \pm 0.52$  mm backfat. During a 4-week lactation, the sows were fed *ad libitum* either a low-protein diet (LP; 7.9% CP, 15.5 MJ DE/kg) or a high-protein diet (HP; 19.0% CP, 15.6 MJ DE/kg). Blood samples were collected on day 110 of gestation, at mid lactation (day 14), and late lactation (no less than 2 d before weaning) after feed was removed from sows at 1600 h the previous day.

On day 110 of gestation, the plasma concentration of glycerol was 80% higher ( $P < 0.01$ ) in fat sows than lean sows ( $182$  vs  $99$   $\mu$ M), plasma NEFA was 30% higher, although not significant ( $655$  vs  $505$   $\mu$ M), and insulin and glucose did not differ between fat and lean sows (average concentrations; insulin  $6.6$   $\mu$ U/ml and glucose  $4.1$  mM).

**Table 1.** Average voluntary feed intake (VFI) during lactation and the average concentration of glycerol, NEFA, insulin and glucose in plasma collected at mid- and late-lactation for fat and lean sows fed a low-protein (LP) or high-protein (HP) diet during lactation.

Body fatness	Lactation diet	No. sows	VFI (kg/day)	NEFA ( $\mu$ M)	Glycerol ( $\mu$ M)	Insulin ( $\mu$ U/ml)	Glucose (mM)
Fat	HP	10	3.83	960	143	3.8	3.1
	LP	9	3.27	750	131	4.8	3.3
Lean	HP	9	5.38	450	113	4.0	3.0
	LP	10	4.93	530	81	3.6	3.2
SEM <sup>1</sup>			0.252	85	15.2	0.74	0.16
<b>Probability values for main effects<sup>2</sup></b>							
Fat vs Lean			<0.01	<0.01	0.07	0.64	0.62
HP vs LP			0.17	0.60	0.61	0.80	0.38

<sup>1</sup> SEM = standard error between the means. <sup>2</sup> Interaction term not significant.

Throughout lactation, fat sows ate on average 1.5 kg/d less than lean sows, irrespective of the protein content of the diet fed during lactation (Table 1), and this corresponded to fat sows having 74% higher NEFA and 41% higher glycerol. Plasma insulin and glucose did not differ between fat and lean sows during lactation. It is concluded that body fat is regulating feed intake possibly through the signals, glycerol and NEFA.

### References

WILLIAMS, I.H. and SMITS, R.J. (1991). In "Manipulating Pig Production III", p. 73, ed. E.S. Batterham. (Australasian Pig Science Association: Attwood).

## SUPER-ALIMENTATION OF GILTS DURING LACTATION

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Sows attempt to meet the sudden and large metabolic demand of lactation by increasing their level of voluntary food intake. However, the voluntary food intake of most sows during lactation generally does not provide sufficient energy or protein to meet their requirements for maintenance and milk production. By providing multiparous sows with extra food during lactation (via a gastric cannula) they remained in an anabolic state, and both milk production and piglet growth were increased (Matzat *et al.*, 1990). Do gilts respond in the same way as sows to extra food in lactation? Gilts are smaller than sows and would be expected to have a greater impetus for body growth because they have not reached their mature body size. If gilts are provided with extra food during lactation, they might partition more of it into body tissue and less into milk. Gilts, fitted with gastric cannulas, were used to test the hypothesis that they would respond differently from the sows of Matzat *et al.* (1990) to extra food in lactation.

Thirty-six PIC, Camborough × Canabrid gilts were fitted with gastric cannulas between 70-85 d of gestation (Pluske *et al.*, 1995). After farrowing they were allocated to one of three treatments: (i) *restricted* - gilts fed ≈ 50% of *ad-libitum* intake; (ii) *ad libitum* - gilts were encouraged to eat as much food as possible; and (iii) *super-alimented* - gilts were infused seven times daily through their cannulas to achieve a 25-30% increase in food intake above that of gilts fed *ad libitum*. Gilts farrowed at an average weight of 176.5 kg with 17.8 mm of backfat. During lactation they were fed a diet based on cereals, soya bean meal, fishmeal and sugar (18.5% CP and 14.4 MJ ME/kg). Milk production was estimated in early (10-14 d) and late (20-24 d) lactation using a modification of the D<sub>2</sub>O dilution technique (Pluske *et al.*, unpublished). Piglets were weaned at an average age of 28 d.

**Table 1. Gilt performance, milk yield and piglet growth during lactation.**

	Restricted	<i>Ad libitum</i>	Super-alimented	SEM	P
<b>Lactation</b>					
Δ Live-weight (kg)	-38.1 <sup>a</sup>	-13.6 <sup>b</sup>	10.9 <sup>c</sup>	5.77	<0.001
Δ Backfat (mm)	-8.6 <sup>a</sup>	-3.6 <sup>b</sup>	1.8 <sup>c</sup>	1.09	<0.001
Food intake (MJ ME/d)	41.8 <sup>a</sup>	74.9 <sup>b</sup>	103.8 <sup>c</sup>	2.59	<0.001
<b>Milk yield (kg/d)</b>					
Early lactation	9.0	9.4	9.4	0.76	0.84
Late lactation	7.5	8.9	8.0	0.91	0.97
Litter size	8.4	8.5	8.6	0.47	0.37
Piglet growth (g/d)	215	242	236	14.9	0.13

abcMean values in the same row with different superscripts are significantly different.

Super-alimentation during lactation provided gilts with 38% more energy ( $P < 0.001$ ) than their counterparts fed on an *ad libitum* basis. This increase in intake was associated with marked gains in both live-weight (10.9 kg) and backfat (1.8 mm) in lactation. Increasing energy intake of gilts above that of their *ad libitum*-fed counterparts did not stimulate milk production and suggests that, unlike the sows of Matzat *et al.* (1990), gilts are likely to partition extra energy into body growth than into milk production.

### References

- MATZAT, P.D., HOGBERG, M.G., FOGWELL, R.L. and MILLER, E.R. (1990). In "Report of Swine Research", pp. 36-40, AS-SW-8904 (Michigan State University).  
 PLUSKE, J.R., WILLIAMS, I.H., CEGIELSKI, A.C. and AHERNE, F.X. (1995). *Canadian Journal of Animal Science*. (In press).



## TOTAL CREATINE IN SOWS' COLOSTRUM AND MILK

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Although the major milk nutrients have been extensively researched, less is known of the minor milk components. Nevertheless, a number of these latter components are of importance to the growth and development of young mammals (Harzer and Haschke, 1989). A preliminary nuclear magnetic resonance study in our laboratory unexpectedly showed the presence of creatine phosphate in sows' milk (Comber and Hartmann, 1993) implying that creatine also may be present in sows' milk. Concentrations of both creatine phosphate and creatine in sows' milk, from farrowing to day 28 of lactation, were measured using enzymatic methods (Bergmeyer, 1985). Colostrum and milk was collected from all functional glands of sows prior to farrowing and throughout lactation. The days of lactation, number of sows and the concentrations of total creatine (that is, the sum of creatine phosphate plus creatine) are shown in Table 1.

**Table 1. The concentration of total creatine (mM) in sows' colostrum and milk (mean  $\pm$  SE) throughout lactation.<sup>1</sup>**

Lactation (d)	-0.5	0	1	2	3	4	5	7	11	18	28
Mean	1.6 <sup>a</sup>	1.3 <sup>a</sup>	2.0 <sup>b</sup>	1.9 <sup>b</sup>	2.4 <sup>c</sup>	2.6 <sup>c</sup>	2.3 <sup>c</sup>	2.5 <sup>c</sup>	2.4 <sup>c</sup>	2.7 <sup>c</sup>	2.5 <sup>c</sup>
SE	0.20	0.06	0.06	0.13	0.27	0.03	0.11	0.09	0.08	0.11	0.20
n	5	3	3	3	3	3	3	11	11	11	3

<sup>1</sup> Within a row, means with the different superscripts are significantly different ( $P < 0.05$ ).

The total creatine concentration significantly increased from 1.6 mM in colostrum prior to farrowing to 2.4 mM by day 3 of lactation ( $P < 0.05$ ), where it remained unchanged throughout lactation to day 28 (Table 1). The total creatine concentrations varied significantly ( $P < 0.5$ ) among sows prior to farrowing (range = 1.2-2.3 mM). However, from farrowing to day 28 there were no significant differences among sows. The variation in total creatine concentration among glands within a sow prior to farrowing was small ( $CV < 6\%$ ) with a much larger variation being observed on day 0 to day 28 ( $CV = 15-30\%$ ).

Although creatine phosphate is converted to creatine during digestion, the total creatine concentration has not previously been measured in the milk of any species. However, the concentration of creatine alone is up to four times greater in sows' milk than either human or cows' milk. Although the piglet's requirement for creatine has not been shown, creatine is an important component for the development of skeletal muscle, the neonatal brain and many cellular functions. The higher concentration of total creatine in sows' milk may be related to the rapid growth of the piglet. The piglet doubles its birth weight in 7 d, whereas, the human infant and calf require 180 d and 60 d, respectively, to double their birth weights. During this period of rapid growth it is possible that the piglet is unable to synthesise sufficient creatine to adequately meet its creatine demand.

### References

- BERGMEYER, H.U. (1985). "Methods of Enzymatic Analysis", eds, H.U. Bergmeyer, J. Bermeyer and M. Grabl. 3rd edn. (Verlag Chemie: Weinheim).
- COMBER, M.F. and HARTMANN, P.E. (1993). In "Manipulating Pig Production IV", p. 263, ed. E.S. Batterham. (Australasian Pig Science Association: Attwood).
- HARZER, G. and HASCHKE, E. (1989). In "Micronutrients in milk and milk-based food products", pp. 125-237, ed. E. Renner. (Elsevier Applied Science: London and New York).

## EFFECTS OF DIETARY LYSINE DURING THE FIRST LACTATION ON SUBSEQUENT LITTER SIZE OF SOWS

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Previous work Tritton *et al.*, (1994), showed that lactating, first-litter sows required diets containing at least 8.4 g lysine/kg to maximise lactation performance. Subsequent litter size was significantly greater (10.7 vs 9.6,  $P < 0.05$ ) for sows offered diets containing at least 13.1 g lysine/kg vs sows offered 8.4 g lysine/kg or less (Tritton *et al.*, 1994). The present experiment was designed to determine if ovulation rate or embryo survival may be positively influenced by dietary lysine intake during the preceding lactation.

Two hundred and ten first-cross hybrid gilts with a mean ( $\pm$  SEM) post-partum body-weight and backfat thickness (at 6.5 cm from the midline) of  $186.4 \pm 1.5$  kg and  $21.5 \pm 0.4$  mm respectively, were randomly allocated at parturition to one of two diets with dietary lysine contents of 8.1 and 13.3 g/kg. Both diets contained 14.3 MJ DE/kg and were offered *ad libitum* during a  $24.1 \pm 0.4$  d lactation. Litter size 2 d after farrowing was  $10.1 \pm 0.1$  piglets. A sub-sample of 48 sows were slaughtered 33-37 d after mating and the number of corpora lutea and viable embryos were recorded.

**Table 1. Effect of dietary lysine levels in diets during lactation on performance parameters of first-litter sows.**

	Number of sows	Dietary lysine (g/kg)		SED
		8.1	13.3	
Feed intake (kg/d)	191	4.46	4.35	0.11
Sow weight loss (kg)	191	20.2	17.8	1.4
Sow backfat loss (mm)	191	3.6	3.6	0.5
Piglet growth rate (g/d)	191	207	205	5.0
Weaning - mating interval (d)	191	6.8	6.9	0.4
2nd litter size born	103	10.8	10.3	0.5
Corpora lutea	48	14.6	16.1	0.9
Embryo survival (%)	48	73.3	70.8	5.5

Dietary lysine content had no significant effect on voluntary feed intake, sow body composition changes, piglet growth rate or subsequent reproductive efficiency (Table 1). The failure to repeat the response of subsequent litter size to dietary lysine level observed earlier (Tritton *et al.*, 1994) may have been as the result of the lack of dietary lysine effect on body tissue catabolism during lactation in the present study. Sows which were offered the low protein diet in the present study experienced less nutritional stress because sow body-weight loss during lactation was greater during the previous study by Tritton *et al.* (1994) despite a shorter lactation period.

### Reference

TRITTON, S.M., KING, R.H., CAMPBELL, R.G. and EDWARDS, A.C. (1994). *Proceedings of the Australian Association of Pig Veterinarians Programme, Australian Veterinary Association Conference, Sydney 1994*, pp. 29-32.

## THE DIGESTIBILITY OF AMINO ACIDS IN HUMAN MILK

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The gross amino acid (AA) composition of human milk (HM) is widely accepted as the standard upon which to base the AA requirements of human infants. Implicit in this is the assumption that the AA in HM are completely absorbed during digestion. There are a number of proteins present in HM, however, that are thought to resist digestion. Thus, the gross AA composition of HM may not necessarily reflect the AA requirements of the infant. The profile of absorbed AA from HM may be more appropriate. It is difficult to accurately determine the absorbed AA composition of HM using the breast-fed infant, as ileal digestibility cannot be determined routinely. The piglet has been shown to be a suitable model for studying aspects of protein digestion in the human infant (Moughan *et al.*, 1992; Darragh and Moughan, 1995), and in the present study, three-week-old, male, Large White  $\times$  Landrace piglets ( $3.82 \pm 0.15$  kg body-weight) were used to determine true (corrected for endogenous ileal AA flows) ileal AA digestibility values.

Following a preliminary period of 7 d, during which time the piglets were trained to drink an infant formula from a bottle with a rubber teat attached, piglets were fed either HM (n=6) for a further 6 d, or a 50:50 mix of the infant formula and a protein-free (PF) diet for 3 d, followed by the PF diet (n=6) for a further 3 d. Chromic oxide was used as an indigestible marker. At the end of the six-day period samples of ileal digesta were collected from the terminal ileum of each piglet (average age, 21 d) at slaughter. Endogenous ileal excretions of AA determined in piglets fed the PF diet, and assumed to be representative of endogenous AA flows occurring in the piglets fed HM (Darragh, 1995), were used to determine true digestibility values. The mean true digestibilities of total nitrogen and AA nitrogen were 88% and 95%, respectively. True ileal digestibilities for the individual AA were subsequently used to determine an absorbed AA composition of HM, which is compared with the gross composition in Table 1.

**Table 1.** Gross and absorbed amino acid composition of human milk (mg/100 ml; mean  $\pm$  SEM).

Amino acid	Composition		Amino acid	Composition	
	Gross	Absorbed		Gross	Absorbed
Threonine	52 $\pm$ 1.6	44 $\pm$ 1.4	Phenylalanine	43 $\pm$ 1.9	39 $\pm$ 1.8
Valine	58 $\pm$ 1.4	52 $\pm$ 1.3	Histidine	26 $\pm$ 0.6	25 $\pm$ 0.6
Isoleucine	57 $\pm$ 1.0	56 $\pm$ 1.0	Lysine	70 $\pm$ 1.5	68 $\pm$ 1.5
Leucine	104 $\pm$ 1.8	103 $\pm$ 1.8	Methionine	16 $\pm$ 0.4	16 $\pm$ 0.4

Whereas HM protein was well digested, certain AA such as threonine, valine and phenylalanine were not absorbed as well as other AA. The proteins in HM that are thought to resist digestion in the infant have a high proportion of these AA in their structures (Harzer and Bindels, 1987). Because the absorbed profile of AA in HM was notably lower in these specific AA when compared with the gross AA composition of HM would suggest that a proportion of the protein in HM escapes digestion. Therefore, the absorbed profile of AA may be a better basis for determining the AA requirements of the human infant.

### References

- DARRAGH, A.J. (1995). The amino acid composition of human milk - towards determining the amino acid requirements of the human infant. PhD Thesis. Massey University.
- DARRAGH, A.J. and MOUGHAN, P.J. (1995). *Journal of Pediatric Gastroenterology and Nutrition*. (In press).
- HARZER, G. and BINDELS, J.G. (1987). In "New aspects of nutrition in pregnancy, infancy and prematurity", pp. 83-94, ed. M. Xanthou. (Elsevier: Amsterdam).
- MOUGHAN, P.J., BIRTLES, M.J., CRANWELL, P.D., SMITH, W.C. and PEDRAZA, M. (1992). *World Review of Nutrition and Dietetics*. 67:40-113.

## THE EFFECT OF FOOD INTAKE DURING LACTATION ON NITROGEN METABOLISM OF FIRST-LITTER SOWS

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Nutrient intake, even when sows are fed *ad libitum*, is usually insufficient to meet the metabolic demands of lactation. King *et al.* (1993) established that sows required a diet containing at least 202 g CP/kg and 12.8 g lysine/kg to maximise N balance during lactation when they were offered 57 MJ DE/day. However N balance was below zero and energy may still have been limiting the lactation performance and N balance of sows. The aim of this experiment was to define the relationship between energy intake and N balance in lactating first-litter sows given a diet containing sufficient protein and essential amino acids to support optimum N balance.

Thirty gilts were allocated at parturition to six levels of feeding ranging from 2.5 kg/d up to 5.5 kg/d. The common diet given during lactation contained 15.3 MJ DE/kg, 238 g CP/kg, 213 g digestible CP/kg and 14.5 g lysine/kg and was considered adequate in protein (King *et al.*, 1993). The live-weight and P2 backfat of sows at farrowing were 157.2 kg and 24.3 mm respectively. All sows suckled nine pigs which were weaned at 28 days of age. Milk yield, by the deuterium oxide dilution technique, and N balance were estimated between day 23 and day 28 of lactation.

Table 1. Effect of energy intake on N metabolism and performance during lactation.

	Dietary energy intake (MJ DE/d)						Significance <sup>1</sup>		SED
	38.0	46.9	56.0	62.1	69.1	77.9	Linear	Quad	
Backfat loss (mm)	9.8	7.7	7.1	8.1	5.8	6.5	*	NS	1.9
Live-weight loss (kg)	30.9	24.0	17.5	18.8	12.0	5.0	***	NS	4.1
Piglet growth (g/d)	215	227	237	254	249	267	***	NS	14
Milk Yield (kg/d)	9.18	8.91	9.80	10.25	9.95	11.45	**	NS	0.83
N metabolism (g/d)									
Milk	83.7	81.9	78.3	90.8	86.5	102.9	*	NS	8.0
Balance	-25.4	-14.3	4.5	7.8	19.3	21.7	***	NS	10.3

<sup>1</sup> NS, non-significant, \*P<0.05, \*\*P<0.01, \*\*\*P<0.001.

Lactation performance and N metabolism of sows improved in response to increasing daily energy intake. Based on intercept values of the relationships between N metabolism and energy intake, zero N balance occurred at 59.2 MJ DE/d. At this energy intake the digestible N intake was 131.5 g N/d and N output in milk was 86.5 g N/d. Consequently, the efficiency of conversion of digestible N into milk N was 66%. Commercial pig production should offer protein adequate diets and aim for voluntary food intakes of at least 5.5 kg/d during lactation to maximise performance and N conservation in first-litter sows.

### References

KING, R.H., TONER, M.S., DOVE, H., ATWOOD, C.S. and BROWN, W.G. (1993). *Journal of Animal Science*. 71:2457-2463.

## POTENTIAL MILK PRODUCTION IN GILTS

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After manipulating the body composition of gilts by changing their protein and energy intakes during pregnancy their mammary tissue was measured at farrowing and found that lean sows compared to fat sows had twice as many alveolar or secretory cells (Head *et al.*, 1991) and four times as much DNA (Head and Williams, 1991). Hacker and Hill (1972) found that the number of alveolar cells did not increase during lactation and so the maximum number of secretory cells is probably fixed at parturition. Is this important? There is a high correlation between cell number in the mammary gland and milk production in rats (Tucker, 1981) and, if a similar relationship exists in pigs, it suggests that lean animals have a greater potential to produce milk than fat animals because they have more secretory machinery. The hypothesis that more mammary cells at parturition would allow a higher output of milk was tested in this experiment. The experimental model was fat and lean gilts on which were fostered heavy piglets to help stimulate maximum milk production.

During pregnancy the gilts were fed either 2.3 kg/d of a low-protein diet (6.3% CP and 16.6 MJDE/kg as fed) or 1.7 kg/d of a high-protein diet (15.8% CP and 14.1 MJDE/kg as fed) to produce fat (n=6) or lean (n=7) sows. The diets and feeding regimens were the same as those used earlier (Head and Williams, 1991). Within 24 h of farrowing after the gilts' own litters had obtained colostrum they were replaced with 14-day-old piglets weighing about 5 kg. All 13 gilts during lactation were offered *ad libitum* a diet containing 18.9% CP and 14.3 MJ DE/kg as fed.

**Table 1. Mean growth ( $\pm$  SEM) of litters that were fostered on to lean (n=7) and fat (n=7) sows within 24 h of farrowing.**

	Lean	Fat	Significance <sup>1</sup>
Number of piglets fostered/litter	9.1	9.3	
Weight of litter at start (kg)	51.1 $\pm$ 1.17	51.0 $\pm$ 2.09	NS
Weight of litter after 14 d (kg)	82.2 $\pm$ 1.97	71.3 $\pm$ 2.09	**
Litter daily gain (kg)	2.22 $\pm$ 0.137	1.45 $\pm$ 0.099	**
Sow weight (kg):			
at farrowing	170 $\pm$ 1.6	176 $\pm$ 1.4	*
at weaning	148 $\pm$ 3.0	137 $\pm$ 6.0	NS
Sow backfat (mm):			
at farrowing	28.0 $\pm$ 1.68	34.7 $\pm$ 0.66	**
at weaning	22.2 $\pm$ 1.04	33.3 $\pm$ 2.7	**

<sup>1</sup> NS, non significant, P>0.05; \* P<0.05; \*\* P<0.01.

Piglets sucking the lean sows grew 50% faster (2.22 vs 1.45 kg/d) than those sucking the fat sows suggesting that milk output of the lean sows was much greater. This assumes no change in milk composition. If account is taken of the high maintenance costs of the 2-week-old, heavy piglets the calculated milk production of the lean sows was about 13 L milk per day; a very high output for the first two weeks of lactation. Advice is commonly given to commercial producers to limit the maternal gain of gilts in pregnancy to about 20 kg of which half might be fat and half lean. The fat sows in this study are equivalent to this in their lean gain ( $\approx$  14 kg) but they gained between two and three times the amount of fat. Thus it appears that reducing lean gain in pregnancy can limit mammaryogenesis thereby reducing the number of secretory cells available to produce milk.

### References

- HACKER, R.R. and HILL, D.L. (1972). *Journal of Dairy Science*. 55:1295-1299.  
 HEAD, R.H., BRUCE, N.W. and WILLIAMS, I.H. (1991). In "Manipulating Pig Production III", p. 76, ed. E.S. Batterham. (Australasian Pig Science Association: Werribee).  
 HEAD, R.H. and WILLIAMS, I.H. (1991). In "Manipulating Pig Production III", p. 33, ed. E.S. Batterham. (Australasian Pig Science Association: Werribee).  
 TUCKER, H.A. (1981). *Journal of Dairy Science*. 64:1403-1421.

## CAN NUTRITION IN PREGNANCY AND LACTATION AFFECT THE DEVELOPMENT OF THE MAMMARY GLAND ?

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Changes in DNA in mammary tissue of gilts suggest that most of the cell growth takes place between day 75 and 90 of pregnancy with few changes occurring thereafter (Kensinger *et al.*, 1982). Head and Williams (1991) showed that the number of milk secretory cells can be reduced if lean gain is restricted during pregnancy by low-protein diets. Revell *et al.* (1995) report that feeding high-protein diets in lactation can stimulate milk yield, even in sows where mammary growth has been presumably retarded by low-protein diets during pregnancy. It is suggested that the number of secretory cells might be stimulated during lactation by feeding high-protein diets and that the stimulation might be greatest when secretory tissue growth has been retarded during pregnancy.

Eleven Large White  $\times$  Landrace first-litter sows were fed 2.3 kg/d of a low-protein diet (5.8% CP, 14.6 MJ DE/kg) and 13 sows were fed 1.7 kg/d of a high-protein diet (15.6% CP, 14.5 MJ DE/kg) during pregnancy to produce fat or lean sows. At the start of lactation fat sows weighed  $158 \pm 2.4$  kg (mean  $\pm$  SE) with a backfat at the P2 of  $24 \pm 0.9$  mm. Lean sows weighed  $148 \pm 2.0$  kg with a backfat of  $18 \pm 0.5$  mm. During a 4-week lactation, the sows were fed *ad libitum* either a low-protein diet (7.9% CP, 15.5 MJ DE/kg) or a high-protein diet (19.0% CP, 15.6 MJ DE/kg). Biopsies of mammary tissue (2 g) were taken on day 105 of pregnancy and from sucked teats on days 14 and 28 of lactation, frozen in liquid nitrogen and analysed for DNA.

**Table 1. DNA in mammary tissue and litter growth as an average over 4 weeks for sows fed high- and low-protein diets in pregnancy and lactation (Mean  $\pm$  SEM).**

Dietary protein in:		DNA concentration (mg/g wet tissue) during			Litter growth kg/d
Pregnancy	Lactation	Late pregnancy	Mid-lactation	Late lactation	
Low	Low	$2.6 \pm 0.29$	$2.5 \pm 0.32$	$2.1 \pm 0.34$	$1.5 \pm 0.04^a$
Low	High	$1.9 \pm 0.31$	$2.7 \pm 0.19$	$2.2 \pm 0.20$	$1.7 \pm 0.17^{ab}$
High	Low	$1.8 \pm 0.36$	$3.0 \pm 0.25$	$3.1 \pm 0.24$	$1.6 \pm 0.08^{ab}$
High	High	$2.4 \pm 0.22$	-	$2.1 \pm 0.21$	$2.0 \pm 0.13^b$

<sup>ab</sup>Mean values within a column with different superscript differ significantly ( $P < 0.05$ ).

At day 105 of pregnancy all sows had similar amounts of DNA in their mammary glands; an unexpected result in view of previous work (Head and Williams, 1991). Sows in the previous work had 24 and 36 mm backfat at the end of pregnancy, whereas the present sows were leaner with only 18 and 24 mm. Live-weight at the end of pregnancy was similar in both experiments, suggesting that lean gain during pregnancy in the current experiment was sufficient to not compromise the production of cells in the mammary gland. The DNA concentration also failed to respond to the high-protein diet during lactation. However, milk output, indicated by litter growth, did respond particularly in the final week of lactation ( $P < 0.05$ ). As this extra milk was not associated with an increase in DNA, it is concluded that factors other than the number of secretory cells, such as the supply of substrates for milk synthesis, are more important determinants of milk yield and consequently piglet growth.

### References

- HEAD, R.H. and WILLIAMS, I.H. (1991). In "Manipulating Pig Production III", p. 33, ed. E. S. Batterham. (Australasian Pig Science Association: Attwood).
- KENSINGER, R.S., COLLIER, R.J., BAZER, F.W., DUCSAY, C.A. and BECKER, H.N. (1982). *Journal of Animal Science*, **54**:1297-1308.
- REVELL, D.K., WILLIAMS, I.H., RANFORD, J.L., MULLAN, B.P. and SMITS, R.J. (1995). In "Manipulating Pig Production V", p. 128, eds D.P. Hennessy and P.D. Cranwell. (Australasian Pig Science Association: Werribee).

## A HIGH-PROTEIN DIET MAXIMIZES MILK OUTPUT AND MINIMIZES WEIGHT LOSS IN LACTATION

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In most commercial piggeries, sows do not consume sufficient feed to maintain body weight during lactation and hence body reserves are an important source of substrates for milk production. This experiment tested the hypothesis that milk yield and composition are maintained by sows using body reserves of fat and protein when their voluntary feed intake (VFI) is low. Thirty eight gilts were fed during gestation so that they were either fat or lean at farrowing and, during a 4-week lactation, were fed *ad libitum* either a low-protein (LP; 7.9% CP) or high-protein (HP; 19.0% CP) diet (described by Revell *et al.*, 1995). After cross-fostering, the average litter size was 8.7. Milk yield was calculated by the deuterium oxide technique (Pettigrew *et al.*, 1987) and milk samples were collected in early (day 4-6) and late (day 25-27) lactation. The milk data presented are the average values for early and late lactation. The losses of body fat and protein were estimated from changes in body-weight and backfat thickness (Mullan, 1992).

Sows fed the HP diet during lactation maintained their body protein while those fed the LP diet lost 150 g/d (Table 1). All sows lost body fat during lactation with the greatest losses occurring with sows fed the LP diet. Milk composition was not affected by either body fatness or the protein content of the diet fed to the sows during lactation. The average concentrations of fat, protein and lactose were  $54.3 \pm 3.09$ ,  $46.0 \pm 1.86$  and  $53.8 \pm 1.53$  g/L (mean  $\pm$  SE) respectively. Average milk yield over the entire lactation depended on both body fatness of the sows and the diet fed during lactation; on average lean sows produced 16% more than fat sows ( $P=0.01$ ), and sows fed the HP diet produced 19% more milk than sows fed the LP diet ( $P=0.02$ ).

**Table 1. Average voluntary feed intake (VFI), weight loss, estimated loss of body fat and protein, and milk yield during a 4-week lactation for fat and lean sows fed a low-protein (LP) or high-protein (HP) diet during lactation.**

Body fatness	Lactation diet	No. sows	VFI (kg/d)	Weight loss (g/d)	Fat loss (g/d)	Protein loss (g/d)	Milk yield (kg/d)
Fat	HP	10	3.83	221	221	0	8.00
	LP	9	3.28	1239	571	150	6.04
Lean	HP	9	5.38	86	68	4	9.01
	LP	10	4.93	1007	350	150	8.29
SEM <sup>1</sup>			0.252	113.8	57.9	32.8	0.385
<b>Probability values for main effects<sup>2</sup></b>							
Fat vs Lean			<0.01	0.27	0.03	0.89	0.01
HP vs LP			0.17	<0.01	<0.01	<0.01	0.02

<sup>1</sup>SEM = standard error between the means. <sup>2</sup>Interaction term not significant.

Sows used body reserves to maintain milk composition but they did not mobilize sufficient energy to prevent milk yield from decreasing when feed intake decreased. Given the dependency of milk output on feed intake, production strategies should ensure that VFI during lactation is maximized and that lean tissue gain during pregnancy is sufficient so that mammary development is not compromised and body reserves of protein are high.

### References

- MULLAN, B.P. (1992). *Pig News and Information*. 12:221-225.  
 REVELL, D.K., WILLIAMS, I.H., RANFORD, J.L., MULLAN, B.P. and SMITS, R.J. (1995). In "Manipulating Pig Production V", p. 128, eds D.P. Hennessy and P.D. Cranwell. (Australasian Pig Science Association: Werribee).  
 PETTIGREW, J.E., CORNELIUS, S.G., MOSER, R.L. and SOWER, A.F. (1987). *Livestock Production Research*. 16:163-174.

## EFFECT OF INCREASED SUCKLING FREQUENCY ON MAMMARY DEVELOPMENT AND MILK YIELD OF SOWS

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The major factor which appears to control milk yield in sows is sucking demand. Auldish *et al.* (1994) have shown milk yield to be linearly related to litter size but this is mainly a function of an increased number of functional glands rather than production per gland. This experiment was conducted to determine if milk yield per gland remains constant or if it can be increased by imposing a greater sucking demand.

At parturition, 18 first-litter sows with an average post-partum live-weight and backfat thickness of  $143.3 \pm 2.2$  kg (mean  $\pm$  SE) and  $19.1 \pm 0.7$  mm respectively, were allocated to three treatments; litter size 6, litter size 12 and a cross-suckle treatment involving two litters, each of 6 pigs. Litter sizes of either 6 ( $n=12$ ) or 12 ( $n=6$ ) piglets were established within 36 h of farrowing. To ensure that sows with the litters of 6 or 12 pigs had only 6 or 12 functional teats, respectively, excess teats were taped from day 1 until they regressed. On day 6, half of the sows with a litter of six were allocated to the cross-suckle treatment by providing them with another litter of 6 pigs. The two litters of 6 pigs were alternated every 30 min to be with the sow from day 6 to 27. On day 27 all sows were slaughtered one h after a successful suckling. The udder was removed from each sow and individual glands dissected out for later analysis.

Table 1. Effect of sucking demand on piglet performance and mammary development on a dry matter (DM) basis.

	Litter treatment			SED
	6	12	6 + 6	
Litter ADG (g/d)	1762 <sup>a</sup>	2571 <sup>b</sup>	2195 <sup>ab</sup>	251
Mean gland weight (g, DM)	110.5 <sup>a</sup>	64.9 <sup>b</sup>	124.0 <sup>a</sup>	8.8
Total functional tissue (g, DM)	662	855	759	75
Gland lactose (g/100 g, DM)	4.2 <sup>b</sup>	4.5 <sup>b</sup>	6.1 <sup>a</sup>	0.7
Gland milk (g/100 g, DM)	72.0 <sup>b</sup>	74.2 <sup>b</sup>	104.7 <sup>a</sup>	9.1
Suckling interval (min)	46.3 <sup>b</sup>	47.4 <sup>b</sup>	38.6 <sup>a</sup>	1.6

<sup>1</sup> Within rows, means with different superscripts are significantly different ( $P < 0.05$ )

The six glands of sows in the cross-suckle treatment supported similar piglet growth as the 12 glands of sows with litters of 12 (Table 1). One gram of dry matter of functional tissue supported similar piglet growth of between 2.6 and 3.0 g/d across all treatments. The higher milk content of glands of sows with the cross-suckle litter (which suckled more frequently) suggests a greater rate of milk synthesis in these animals.

Manipulation of sucking demand in commercial pig production through encouraging greater suckling frequency may lead to improvements in sow milk yield, individual milk consumption and piglet weaning weights.

### References

AULDISH, D.E., MORRISH, L., THOMPSON, M. and KING, R.H. (1994). *Proceedings of the Nutrition Society of Australia*. 18:175.



## THE EFFECT OF DIETARY LYSINE ON THE COMPOSITION OF MILK IN GILTS

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Stahly *et al.* (1990) found that the average weight of piglets at weaning increased linearly by approximately 0.65 kg as the level of dietary lysine increased from 4.2 to 9.2 g/kg in the diet during lactation. The aim of this experiment was to investigate whether the high lysine to energy ratio fed during the sows' first lactation had an effect on milk protein concentrations.

Forty gilts were selected from the study of Tritton *et al.* (1995) and randomly allocated between two diets to determine the effect of a high lysine diet on milk protein concentrations during the first lactation. The diets were formulated to contain either 13.3 or 8.1 g lysine/kg and similar amounts of protein and digestible energy (DE). One week prior to allocation to the farrowing crates all gilts were fed 1.8 to 2.0 kg of the feed. On the day of parturition, the treatment group (n=20) were fed the treatment diet (lysine 13.3 g/kg, DE 14.3 MJ/kg) while the control group received a standard lactating sow diet (lysine 8.1 g/kg, DE 14.3 MJ/kg). The average lysine:DE ratios were 0.93 and 0.57 g/kg for the treatment and control diets respectively throughout the 24 d lactation. The feed during lactation increased until *ad libitum* intake was achieved. Colostrum and milk were collected at five time points throughout lactation at 0-3, 72, 168, 336 and 504 h post-partum from all functional glands after an intra-muscular injection of 10 mg oxytocin. The concentrations of milk protein were determined by the Lowry method as described by Atwood and Hartmann (1992).

**Table 1.** Protein concentration (g/100 ml) in milk collected between 3 and 504 h post-partum from gilts fed a diet with either a high or standard lysine content.

Diet	Hours post-partum				
	0-3	72	168	336	504
13.3 g lysine/kg	10.95	6.27*	5.74*	4.90*	5.06*
8.1 g lysine/kg	11.22	4.69	4.40	3.37	3.28
SEM	0.62	0.28	0.31	0.30	0.23

\*Indicates significant difference between diets at (P<0.001)

The results shown in the table indicate a significant difference between milk protein content of the gilts given high or a low lysine diet over 72-504 h post-partum. The percentages of total solids in milk from treated and control gilts were 23.89, 20.36, 19.96, 19.13, 20.17 vs 22.27, 18.98, 19.32, 19.73, 19.48 respectively for the five time points, these differences were not significantly different (P>0.05). Tritton *et al.* (1995) reported no increase in the growth rate of piglets suckling gilts given the high lysine diet. The increase in milk protein reported in this study may not have been sufficient to influence piglet growth rate. Further work will be required to determine the effect of a high lysine diet on milk protein concentrations and growth rate.

### References

- ATWOOD, C.S. and HARTMANN, P.E. (1992). *Journal of Dairy Research*. 59:287.  
 STAHLY, T.S., CROMWELL, G.L. and MONEGUE, H.J. (1990). *Journal of Animal Science*. 68:369.  
 TRITTON, S. M., KING, R.H., CAMPBELL, R.G., HUGHES, P.E. and KERSHAW, S.S. (1995). In "Manipulating Pig Production V", p. 131, eds D.P. Hennessy and P.D. Cranwell. (Australasian Pig Science Association: Werribee).

## A REVIEW - INTESTINAL SPIROCHAETAL INFECTIONS OF PIGS: AN OVERVIEW WITH AN AUSTRALIAN PERSPECTIVE

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### Introduction

Intestinal spirochaetes have become recognized over the last 25 years as an important group of enteric pathogens. These bacteria cause disease in a variety of animal species, especially pigs, poultry, dogs and human beings (Hampson and Stanton, 1996). In pigs, the bacteria cause two well-recognized conditions, swine dysentery (SD), and intestinal spirochaetosis (IS) (Taylor *et al.*, 1980; Hampson, 1991). A third condition, referred to here as spirochaetal colitis (SC), is less clearly defined, but is associated with certain weakly beta-haemolytic spirochaetes other than those causing IS.

Swine dysentery is one of the most significant production-limiting diseases of pigs and is a common problem throughout the world. The significance of IS and SC in reducing production is less clear; certainly, clinical manifestations of the conditions are much less severe than with SD. The prevalence of the diseases is not known, but the authors' observations suggest that IS occurs commonly in pigs in Australia and North America, whilst cases of IS and SC also have been reported in Europe (Taylor, 1992).

### General description of spirochaetes

Spirochaetes are chemoheterotrophic bacteria, characterized by a unique cellular anatomy and a distinctive morphology. They are spiral-shaped, with their main structural component being a coiled protoplasmic cylinder consisting of cytoplasmic and nuclear regions, surrounded by a cytoplasmic membrane-cell wall complex. Periplasmic flagellae, that are wound around the helical protoplasmic cylinder, run from each end of the cell, and overlap near its middle. The outermost structure is the outer sheath or outer cell envelope, which completely surrounds both the protoplasmic cylinder and the periplasmic flagellae (Canale-Parole, 1977; Canale-Parole, 1984).

Spirochaetes belong in the order *Spirochaetales* (Canale-Parole, 1984). Intestinal spirochaetes belong in the family *Spirochaetaceae*, within this order. This family includes the genera *Spirochaeta*, *Borrelia*, *Cristispira*, *Brachyspira*, *Treponema*, and *Serpulina* (Canale-Parole, 1977; Hovind-Hougen *et al.*, 1982; Stanton *et al.*, 1991; Stanton, 1992). Another genus, *Anguillina*, containing the spirochaetes associated with IS, has been proposed (Lee *et al.*, 1993b), but this seems more likely to be a species of *Serpulina* (Duhamel *et al.*, 1993b; Stanton *et al.*, 1995; Trott, Stanton, Jensen, Duhamel, Johnson, and Hampson, unpublished data). The spirochaete that causes SD (*Serpulina hyodysenteriae*), and a non-pathogenic porcine intestinal spirochaete (*Serpulina innocens*), were originally placed in the genus *Treponema*, based on their requirements for anaerobic growth conditions, their morphology and their habitat (Harris *et al.*, 1972; Kinyon and Harris, 1979). Subsequently, based on the results of extensive genetic analysis, it was considered that the organisms belong to a distinct and new genus, named *Serpula* (Stanton *et al.*, 1991). The genus name was later modified to *Serpulina* (Stanton, 1992).

The term "intestinal spirochaete" implies that the organisms share a habitat, rather than that they are a specific genetic group. In the case of the porcine intestinal spirochaetes, discussion will be limited to intestinal organisms currently regarded by us as belonging to the genus *Serpulina*. Other organisms, such as the small, non-pathogenic porcine intestinal spirochaete *Treponema succinifaciens* (Cwyk and Canale-Parole, 1979), will not be considered.

## Historical perspectives

Spirochaetes were recognized in the intestines of swine well before the turn of the last century (Thomson and Thomson, 1914). In the first descriptions of SD, enormous numbers of spirochaetes were observed in the faeces of diseased animals (Whiting *et al.*, 1921), but these failed to induce disease when inoculated into two experimental pigs. Their significance as pathogens was only firmly established in the early 1970s, when the strongly beta-haemolytic spirochaete *Serpulina* (formerly *Treponema*) *hyodysenteriae* was isolated, and used to reproduce SD in pigs (Taylor and Alexander, 1971; Harris *et al.*, 1972). This discovery led to a period of intense research into both SD, and the intestinal spirochaetes. Throughout the 1970s it became clear that other morphologically-similar, but weakly haemolytic spirochaetes inhabit the porcine large intestine (Hudson *et al.*, 1976; Kinyon *et al.*, 1977; Kinyon and Harris, 1979). Generally, these were considered to be non-pathogenic, and consequently were named *Serpulina* (formerly *Treponema*) *innocens* (Kinyon and Harris, 1979).

In 1980, Taylor and colleagues described and used weakly haemolytic spirochaetal isolates to reproduce a distinct porcine colonic infection, which they called spirochaetal diarrhoea (now generally termed as IS). This led to a re-evaluation of the porcine intestinal spirochaetes. It is now clear that the weakly haemolytic organisms are a diverse group, comprising at least four species within the genus *Serpulina*, and include both pathogenic and non-pathogenic organisms (Lemcke and Burrows, 1981; Binek and Szykiewicz, 1984; Lymbery *et al.*, 1990; Lee *et al.*, 1993b; Ramanathan *et al.*, 1993; Fellström and Gunnarsson, 1994; Fellström *et al.*, 1994; Stanton *et al.*, 1995; Trott, Stanton, Jensen, Duhamel, Johnson and Hampson, unpublished data). The diversity and differing pathogenic potential of the porcine intestinal spirochaetes must be considered when investigating the epidemiology of SD, IS, and SC, and when devising means for their diagnosis and control.

This review describes recent improvements in the understanding of the organisms associated with intestinal spirochaetal infections of pigs. Areas requiring more research are highlighted, and the Australian situation in relation to these bacteria is discussed.

## Swine dysentery (SD)

### *Clinical description*

Typical cases of SD are characterized by severe mucohaemorrhagic colitis in grower pigs, and sometimes in weaner pigs. Clinical manifestations vary greatly, and include mild and sub-clinical disease. This variation is seen both in individual pigs, and at the herd level. Factors influencing the variation are outlined in the section on the epidemiology of SD.

In typical cases, infected pigs initially show a slight depression, and a reduced food intake. They develop diarrhoea, which is grey to black, and sometimes watery, but is more often soft and porridge-like. The nature of this diarrhoea progresses to contain mucus plugs, fibrin, epithelial cell casts, and flecks of fresh blood. Affected animals have faecal staining of the hindquarters, become dehydrated, and appear gaunt, with a tucked-in abdomen and an arched back. About 10% of affected pigs die within five days of first showing clinical signs, if treatment is not implemented, but death rates exceeding 50% have been reported (Fisher and Olander, 1981; Raynaud *et al.*, 1981a; 1981b). The majority of animals recover over a period of one to several weeks, but their growth rate remains depressed.

### *Economic significance*

In the mid 1980s, SD was identified as being the most economically significant endemic disease of pigs in Australia (Cutler and Gardner, 1988). The condition results in major losses through growth rate depression, especially during the grower phase, as well as in the costs associated with medication and mortalities. Decreases in feed conversion efficiencies of 10-90%, and reductions in average daily live-weight gains of 13-62%, have

been recorded in experimentally infected pigs (Taylor and Davey, 1980). Less tangible costs arise from the necessity to implement preventative measures in herds that do not have the disease, and particularly from the disruption to the supply and movement of pigs when the disease becomes introduced into stock in large breeding company herds. In the latter situation, the company's losses potentially can be enormous.

Overall, Cutler and Gardner (1988) estimated that in infected herds, SD could be costed at \$100 per sow per year. They suggested that 36% of Victorian sow herds were infected. More recently, a serological survey in Western Australia suggested that 33% of herds were infected (Mhoma *et al.*, 1992). Swine dysentery clearly has a major economic impact on the Australian pig industry.

#### *Aetiology of swine dysentery*

Swine dysentery results from infection of the pig's large intestine by *Serpulina hyodysenteriae*, a Gram negative, oxygen-tolerant anaerobic spirochaete (Harris *et al.*, 1972; Stanton, 1992). The bacteria are slender, loosely-coiled rods, approximately 6-11  $\mu\text{m}$  in length, and 0.32-0.38  $\mu\text{m}$  in diameter. They have between 7-14 periplasmic flagellae, which are inserted in two rows at each end of the cell, and overlap in the centre. The bacteria grow on blood agar, and, after a period of three or more days, produce flat, translucent colonies surrounded by areas of clear haemolysis. The organisms can be propagated readily in pre-reduced anaerobic broth containing cholesterol, and either foetal calf serum or pig faecal extract (Kunkle *et al.*, 1986). The addition of 1% oxygen enhances growth (Stanton and Lebo, 1988). Important features which distinguish this spirochaete are outlined in the section on diagnosis of SD.

Swine dysentery has been reproduced experimentally in gnotobiotic pigs challenged with *S. hyodysenteriae* in the absence of other micro-organisms, although colonization by the spirochaete is enhanced by the presence of other anaerobic bacteria (Whipp *et al.*, 1979; 1982). In natural cases of SD, other members of the colonic microflora, such as the protozoan *Balantidium coli*, also may colonize the disrupted colonic epithelium, and exacerbate the lesions of SD.

#### *Pathological changes*

Pigs with chronic SD appear dehydrated and emaciated. Gelatinous oedema sometimes may be found separating the coils of the spiral colon, whilst the drainage lymph nodes are enlarged, moist, and congested. The lesions of SD are confined to the large intestine, and their distribution and severity are very variable. They are usually most marked in the colon, often extending as far as the rectum, but the caecum may only be mildly effected (Lysons, 1992). At the microscopic level, early lesions include hyperaemia and oedema of the *lamina propria*, with goblet cells prominent and distended with mucus. The mucosa appears roughened and corrugated, and subsequently develops areas of inflammation and epithelial cell necrosis. Pseudomembranes consisting of fibrin, mucus, necrotic enterocytes, leukocytes and erythrocytes overlay these eroded areas. With time, these fibrinous plaques become thicker and drier, and may adhere to the eroded epithelium.

#### *Pathogenesis*

Pigs become affected with SD following ingestion of dysenteric faeces containing *S. hyodysenteriae*. An inoculum of  $10^5$  colony forming units (cfu) is usually sufficient to produce SD (Kinyon and Harris, 1979), although much higher dose rates (eg.,  $10^{10}$  cfu) often are used for experimental challenge (eg., Hampson *et al.*, 1993). Optimal colonization is achieved using actively motile bacterial cells in mid-log phase, and repeating the oral challenge daily over two or three days. The bacteria presumably survive the acidic environment of the stomach protected in faeces, and eventually arrive at the large intestine. *Serpulina hyodysenteriae* has the ability to dispose of NADH in several ways, and can utilise substrate amounts of oxygen (Stanton, 1989). This metabolic versatility may enhance its ability to colonize mucosal sites. The organism displays a

chemotactic response to mucus, and its active motility allows it to penetrate the mucus layer and to invade colonic crypts (Kennedy *et al.*, 1988). At these sites its presence stimulates an outpouring of mucus into the lumen. Clinical signs and lesions of SD start to develop as numbers of spirochaetes reach  $10^6/\text{cm}^2$  of mucosa (Hughes *et al.*, 1977; Whipp *et al.*, 1979). Spirochaetes first appear in the faeces one to four days before diarrhoea commences (Kinyon *et al.*, 1977). At this time, there is a shift in the composition of the rest of the colonic microflora, from predominantly Gram positive bacteria in healthy animals, to mainly Gram negative organisms in pigs with dysentery (Pohlenz *et al.*, 1984).

*Serpulina hyodysenteriae* enters goblet cells in the colonic crypts, and penetrates intracellular gaps in the epithelium (Sueyoshi and Adachi, 1990). There is an associated loss of cohesion between colonic enterocytes, with subsequent necrosis and shedding of the epithelium. The organisms attach to the luminal surface and enter these disrupted epithelial cells. Some spirochaetes also may be observed in the *lamina propria*, particularly around blood vessels. Bleeding occurs from small vessels located under areas of eroded epithelium, which also may be invaded by other members of the colonic microflora.

In SD, small intestinal function is normal, and colonic mucosal permeability is unaffected; however, there is a decrease in colonic absorption, resulting in a severe loss of sodium, chloride, bicarbonate and water from the infected colon (Argenzio *et al.*, 1980; Schmall *et al.*, 1983). The mechanisms whereby these changes occur are unknown, and they do not correspond to the action of any known bacterial toxin. The mechanisms responsible for the enterocyte disruption and subsequent necrosis, which are central features of SD, also are not fully understood. Two possible toxic components of *S. hyodysenteriae* may be involved in the changes. These are: (1) the endotoxic effects of the organism's lipopolysaccharide (LPS) (Nuessen *et al.*, 1982; Nuessen *et al.*, 1983; Greer and Wannemuehler, 1989a), possibly acting through the production of tumour necrosis factor or interleukin-1 (Greer and Wannemuehler, 1989b), and, (2) the cytotoxic effects of its haemolysin(s) on colonic and ileal enterocytes (Lysons *et al.*, 1991; ter Huurne *et al.*, 1993; Bland *et al.*, 1995). Strains of *S. hyodysenteriae* with mutations constructed in the *tlyA* gene (encoding one of the haemolysins) have reduced virulence in pigs, suggesting that the haemolysin has a role in the pathogenesis of SD (Hyatt *et al.*, 1994).

### Immunity

Pigs that have recovered from SD are reported to be protected against experimental challenge with *S. hyodysenteriae* for up to 17 weeks (Joens *et al.*, 1979). The existence of this immunity is encouraging for vaccine development. Nevertheless, a proportion of recovered pigs (7-43%) remain susceptible (Jenkins, 1978; Joens *et al.*, 1978a; Rees *et al.*, 1989a), and about 10% may only become fully protected after two previous bouts of disease (Rees *et al.*, 1989b). This may explain why SD often is observed to recur at intervals of three to four weeks in groups of grower pigs (Kinyon *et al.*, 1977; Harris and Lysons, 1992).

Immunity to *S. hyodysenteriae* appears to be quite strongly serotype-specific, directed against LPS antigens present in the cell envelope. This observation has been important for vaccine development, since it suggests the requirement for LPS components in vaccines. Joens *et al.* (1983) demonstrated the LPS-serotype-specific nature of immunity to SD, by infecting pigs with one or another of four serotypes of the organism, and allowing them to recover. They then used isolates from the four serotypes to challenge a series of surgically-prepared colonic loops in each of these pigs. Only loops inoculated with isolates of the same serotype used initially to infect a given pig did not develop lesions. Limited protection against serotypes, other than those used for infection, has been observed (Kennedy *et al.*, 1992; Nuessen and Joens, 1982; Parizek *et al.*, 1985). This suggests that protective immune responses also are directed at other components that are common to isolates of different serotypes. In Australia it is uncommon for herds to be infected with more than one strain of *S. hyodysenteriae* (Combs *et al.*, 1992), so that serotype-specific immunity is unlikely to be important in preventing a given strain proliferating, whilst allowing another of a different serotype to establish itself.

Changes occur in both antibody titres and in cell mediated immunity in pigs with SD, but their importance is unclear. Titres of serum IgG correlate with the duration of clinical signs, whilst IgA titres in colonic washes are indicative of recent exposure (Rees *et al.*, 1989a). Neither of these titres are strongly correlated with protection from developing SD (Joens *et al.*, 1982; Fernie *et al.*, 1983; Rees *et al.*, 1989a). Other studies suggest that complement components, in conjunction with immune serum, may be involved in the clearance of *S. hyodysenteriae* from the colon (Joens *et al.*, 1985). Cell mediated immunity also may be involved in protection, since there is evidence of inhibition of peripheral blood leukocyte migration, a delayed hypersensitivity response, and a T-cell proliferative response to *S. hyodysenteriae* antigens in pigs convalescent from SD (Jenkins *et al.*, 1982; Kennedy *et al.*, 1992). Nevertheless, in mouse models of the disease, there are no significant changes in T-cell subsets in the *lamina propria* (Nibbelink and Wannemuehler, 1990). The role of immune-mediated components to the lesions of SD is uncertain, as in mouse models, the changes observed in numbers of mast cells in the *lamina propria*, are not correlated with lesion development (Nibbelink and Wannemuehler, 1990). Further work is required to understand the mechanisms involved in host immunity to *S. hyodysenteriae*.

### Epidemiology

#### *Species affected*

*Serpulina hyodysenteriae* is a highly adapted parasite of the mucosa of the large intestine. Pigs are considered to be the main host for the organism, although both mice and rats living on piggeries can harbour the infection (Joens and Kinyon, 1982; Hampson *et al.*, 1991; Duhamel *et al.*, 1992). Although generally it is assumed that the rodents became infected from the pigs, it has been suggested that they are the natural hosts of the spirochaete (Blaha and Gunter, 1985). Experimentally-infected mice show signs of disease, with an outpouring of mucus into their caecae (Joens and Glock, 1979). They continue to shed the organism in their faeces for up to 180 days, and this material has been shown to be infectious for pigs (Joens, 1980). Certain avian species also may be infected with *S. hyodysenteriae*. For example, in the USA, rheas suffering from natural cases of typhlocolitis were shown to be infected with *S. hyodysenteriae* (Sagartz *et al.*, 1992; Atyeo, Combs and Hampson, unpublished data; N.S. Jensen, T.B. Stanton and D.E. Swayne, personal communication). Experimentally, starlings can be transiently colonized (Glock *et al.*, 1978), whilst infection with *S. hyodysenteriae* causes typhlitis in young chicks (Adachi *et al.*, 1985). The organism also causes lesions in the caecae of guinea pigs (Joens *et al.*, 1978b), and in rabbit ileal loops (Knoop, 1979). Dogs have been experimentally colonized for 13 days (Glock *et al.*, 1978), and the organism has been isolated from a dog that had eaten infected pig faeces (Songer *et al.*, 1978).

*Serpulina hyodysenteriae* is able to persist in the environment for limited periods, and this is an important consideration when attempting to eradicate the disease from a piggery. The organism can survive for up to 48 days in dysenteric faeces stored between 0 and 10°C, and for 61 days when diluted 1/10 with water and stored at 5°C (Chia and Taylor, 1978). It only survives for seven days at 25°C, and for 24 hours at 37°C. It is very susceptible to desiccation, and to disinfectants such as phenol and sodium hypochloride (Chia and Taylor, 1978).

#### *Distribution*

Swine dysentery has been reported from all the major pig-producing countries (Roncalli and Leaning, 1976). It was first recorded in Australia in 1938 (McLennan, 1938), and now is recognized in all states (Buddle, 1985; Hampson *et al.*, 1994). There is some evidence that around one third of Australian herds are infected (Cutler and Gardner, 1988; Mhoma *et al.*, 1992). Similarly, a prevalence of 40% was recorded in a serological survey conducted in the mid-west of the USA (Egan *et al.*, 1982). In the UK, SD remains the second most commonly diagnosed disease, after enteric colibacillosis (Lysons, 1992). Confirmed diagnoses in the UK increased by 33% in 1994, after an apparent reduction in 1993 (Anon, 1995).

Within-herd prevalence of SD is influenced by a number of factors. Two serological surveys reported that 18% of pigs were seropositive for *S. hyodysenteriae* (Egan *et al.*, 1983; Mhoma *et al.*, 1992). In one of these studies, rates were 31% seropositive in market age animals, 16% in adults, and 0.5% in weaners (Egan *et al.*, 1983). This reflects the fact that SD occurs most commonly in grower pigs, although it may affect pigs of all age groups (Alexander and Taylor, 1969). Antimicrobial medication used primarily to control respiratory disease may delay the occurrence of clinical disease until about two weeks after weaners are moved to grower accommodation.

#### *Strain variation and distribution*

In recent years, it has become apparent that the species *S. hyodysenteriae* is made up of a variety of different strains. Much of this analytical work has been conducted in Australia. Eighteen Australian isolates were examined and divided into two groups, based on variation in a 37 kDa outer membrane protein (Smith *et al.*, 1990). The use of multilocus enzyme electrophoresis (MEE), indicated that the species has a similar mean genetic diversity (at 0.26) to that reported for other species of pathogenic porcine bacteria (Lee *et al.*, 1993a). These researchers were able to subdivide 98, mainly Australian isolates, into 28 electrophoretic types, contained in four genetic groups. In this study, there were strong indications that the species was clonal. When 91 isolates from 62 piggeries in Australia were examined using serotyping and DNA restriction endonuclease analysis (REA), they were divided into eight serogroups and 31 different REA patterns (Combs *et al.*, 1992). The greatest numbers of different strains was found in Victoria, where 12 strains were isolated from 19 piggeries. In Queensland 10 strains were recovered from 24 piggeries. Overall, only three of 31 strains were found in more than one state, indicating limited geographical dispersion of strains, belonging to related clonal groups. When 210 Australian isolates were serotyped, 47% belonged to serogroup B, 23% to serogroup D, 12% to serogroup A, 11.4% were untypable, and a few isolates were from serogroups E-I (Hampson *et al.*, 1994). This information has practical application for the preparation of appropriate bacterin vaccines for use in Australia, and it was recommended that these be multivalent, containing representative strains from serogroups A, B and D.

Relatively little similar data is available from other countries. Strains of serotypes 1, 2, 5, 6 and 7 (from serogroups A and B) have been reported in the USA (Baum and Joens, 1979; Mapother and Joens, 1985), serotypes 3 (serogroup C), 8 and 9 (unknown serogroups) from Canada (Baum and Joens, 1979; Hampson *et al.*, 1989; Li *et al.*, 1991), and serogroups B, D and E from England (Lau and Hampson, 1992). In Quebec, 70% of 30 isolates belonged to serotypes 8 and 9 (Li *et al.*, 1991). Forty-three field isolates from The Netherlands were divided into six REA patterns (ter Huurne *et al.*, 1992). In Canada, 21 isolates were divided into seven restriction endonuclease patterns, or into four different ribotypes (Harel *et al.*, 1994). Restriction fragment polymorphism (RFLP) analysis of 21 isolates from three farms in the mid-west of the USA demonstrated that a single RFLP type was responsible for each of the outbreaks under investigation (Duhamel *et al.*, 1992). To date, Australian strains have all been distinct from a range of non-Australian strains that have been examined (Combs *et al.*, 1992).

New methods of identifying individual strains has proved to be important in the investigation of the epidemiology of SD. Duhamel *et al.* (1992) demonstrated that isolates from the environment, a mouse, and from affected pigs all shared the same RFLP type. Similarly, in Australia, REA was used to show that a strain isolated from a rat, shot on a piggery that was attempting to eradicate SD, was identical to the strain that had been infecting pigs on the site for the previous six years (Hampson *et al.*, 1991).

The biological properties of individual strains also may vary. In Australia, various isolates have been identified that are resistant to either lincomycin, tylosin, dimetridazole or tiamulin (Smith *et al.*, 1991b; Buller and Hampson, 1994). This resistance is not obviously linked to the serotype, REA pattern or electrophoretic type of the organism (Buller and Hampson, 1994). Variations in virulence also occur: for example, avirulent strains of *S. hyodysenteriae* have been isolated from healthy pigs both in England (Lysons *et al.*, 1982) and in Australia (Lee *et al.*, 1993a). The type strain, B78, has been reported to be less virulent than other strains, when assayed in rabbit ileal loops (Knoop, 1979) or in

pigs (Jensen and Stanton, 1993). The basis of the differences in virulence among strains is not known, however laboratory attenuation of a strain resulted in the loss of a high molecular weight band from silver-stained lipooligosaccharide preparations (Halter and Joens, 1988), whilst two avirulent field isolates have been shown to have reduced motility in porcine mucus, and therefore may not have the same capacity to colonize infected pigs (Milner and Sellwood, 1994).

#### *Factors influencing patterns of disease*

Serological surveys have suggested that infection of herds with *S. hyodysenteriae* is much more common than is the occurrence of overt disease (Mhoma *et al.*, 1992). Even at the individual pig level, there may be considerable variation in susceptibility to disease development. Clearly, the widespread use of prophylactic antimicrobial medication in pig foodstuffs may be preventing clinical expression of the disease. For example, in Sweden, SD has become much more widespread since implementation in 1986 of a ban on the use of in-feed medication (Lysons, 1992). Other factors undoubtedly influence the outcome of infection. Hampson *et al.* (1992) demonstrated that an *S. hyodysenteriae* isolate, recovered from a gilt in a herd that had not experienced clinical SD for five years, and which was not medicating for SD, was fully virulent in experimentally-infected pigs from another piggery. Whether infectivity was modulated by components of the microflora was not determined but, work in experimentally-infected mice has shown that certain members of the colonic microflora can inhibit the growth of *S. hyodysenteriae* (Suenaga and Yamazaki, 1984). The microflora also may be influenced by the diet (Varel, 1987). In laboratory mice, one type of commercial diet was shown to increase the susceptibility of mice to infection with *S. hyodysenteriae* (Nibbelink and Wannemuehler, 1992), whilst the addition of zinc was protective (Zhang *et al.*, 1995). In pigs, deficiencies in vitamin E and selenium increase their susceptibility to SD (Teige, 1984), as does the presence of aflatoxin in the diet (Joens *et al.*, 1981). In another study, SD ceased to be a clinical problem on a commercial piggery after the usual maize-based diet was ensiled (Prohaszka and Lukacs, 1984). This protection was thought to have resulted from the low base value of the ensiled diet interacting with volatile fatty acids produced by fermentation to produce an intestinal environment that inhibited the growth of *S. hyodysenteriae*. Studies in Australia by Siba *et al.* (1994) demonstrated a different effect. In three separate trials, these workers found that feeding a highly digestible diet, based on cooked rice and animal protein, completely protected pigs from developing SD. Colonization appeared to be inhibited, and this may have resulted from alterations in other components of the colonic microflora. This may explain why certain chemotherapeutic agents with no effect on *S. hyodysenteriae* can be used to control SD, since they are thought to inhibit components of the microflora that normally interact to enhance colonization by the spirochaetes (Meyer, 1978).

Other stressors may enhance the spread of *S. hyodysenteriae* and increase the severity of SD. These include cold temperatures, overcrowding, transportation, introduction of new stock, and the stress of farrowing (Harris, 1984; Griffin and Hutchings, 1980; Songer and Harris, 1978). Such factors vary greatly depending on the type and quality of piggery management.

#### *Transmission of swine dysentery*

Swine dysentery can be controlled within herds, whilst its spread among herds can be reduced by appropriate means. The most obvious source of infection is the faeces of acutely affected pigs. Transmission is enhanced by close contact between animals, and by open drainage between pens. Recirculation of effluent from contaminated slurry, waste lagoons or stored manure also aids the spread of infection (Olsen, 1992; Chia and Taylor, 1978; Songer and Harris, 1978). The ability of the organisms to survive in the environment (Chia and Taylor, 1978), and in rodent reservoirs (Joens and Kinyon, 1982; Hampson *et al.*, 1991), also enhances its potential for transmission.

The major means of transmission of SD between herds is the movement of carrier pigs (Fisher and Olander, 1981; Rutter, 1985). This has been demonstrated in England, where 22 of 25 outbreaks of SD were attributable to the introduction of pigs for fattening or breeding (Windsor and Simmons, 1981). Similarly, in Western Australia, a postal



survey of piggeries demonstrated a high correlation between the purchase of replacement or fattening pigs from saleyards, and serological evidence of SD (Robertson *et al.*, 1992). *Serpulina hyodysenteriae* also may be introduced into herds via contaminated vehicles, or the movement of personnel with contaminated boots or clothing (Windsor and Simmons, 1981). Provision of boots and protective clothing for visitors, the presence of a footbath for disinfection at the entrance to the piggery, and the existence of an external security fence are all strongly correlated with the absence of SD from Western Australian piggeries (Robertson *et al.*, 1992).

### Diagnosis

A presumptive diagnosis of SD can be made on the basis of characteristic clinical signs, epidemiological features of the disease, and pathological findings. Clinically affected grower pigs have reduced appetites, are depressed, and have diarrhoea containing fresh blood and mucus. There may be a history of introduction of new stock, or stresses associated with mixing and moving. The disease can be confused with salmonellosis, proliferative enteritis, intestinal spirochaetosis, or, in recently-weaned pigs, colibacillosis. The possibility of diseases exotic to Australia, such as swine fever, also should be considered.

Since SD is a disease with a severe economic impact, and is notifiable in several Australian states, it is important to obtain a definitive diagnosis so that appropriate control measures can be taken. Currently, it is recommended that diagnosis be made by isolating *S. hyodysenteriae* from affected pigs. Nucleic acid-based tests also have considerable diagnostic potential (Duhamel and Joens, 1994). Isolation of *S. hyodysenteriae* is not straightforward, since the organism requires an anaerobic environment, and visible growth on agar plates requires incubation for three or more days. The organisms also become overgrown with other faecal flora, unless faecal extracts either are passed through a series of filter membranes through which only the spirochaetes can pass, or are cultured on special selective agar plates. The use of filters is cumbersome, so selective media are normally used for isolation; typically these are either trypticase soy agar supplemented with 5% bovine (or ovine) blood, 400 µg/ml spectinomycin, and 25 µg/ml each of vancomycin and colistin (Jenkinson and Wingor, 1981); or trypticase soy agar supplemented with 5% blood, and 200 µg/ml spectinomycin, 25 µg/ml spirromycin, 12.5 µg/ml rifampin, and 6.25 µg/ml each of vancomycin and colistin (Kunkle and Kinyon, 1988). The latter so-called BJ medium appears to be the best available for isolating *S. hyodysenteriae* from faeces (Achacha and Messier, 1991). The spirochaetes will grow at 37°C, but tend to outgrow contaminants better if they are incubated at 41°C.

The second main problem with using culture to support diagnosis, is that other intestinal spirochaetes, similar to *S. hyodysenteriae*, present in the faeces of both healthy and diseased pigs (Joens *et al.*, 1980), may be isolated on the same medium. It therefore becomes necessary to clearly identify an isolate as *S. hyodysenteriae*. Phenotypic properties typically used to identify the organism are: (1) strong beta-haemolysis, (2) production of indole, and (3) alpha-glucosidase activity but lack of alpha-galactosidase activity in the commercial API-zym test kit (Hunter and Wood, 1979; Lee *et al.*, 1993a). The inability of *S. hyodysenteriae* to ferment fructose has been suggested as a definitive test (eg., Kinyon and Harris, 1979), however fructose may be utilized as an energy source by *S. hyodysenteriae* cells grown in liquid media (Stanton and Lebo, 1988). The degree of haemolysis may not be absolutely definitive, since a strongly haemolytic non-*S. hyodysenteriae* porcine intestinal spirochaete, that does not cause disease in conventional pigs (Neef *et al.*, 1991; Lysons *et al.*, 1992), but does induce mucoid diarrhoea in gnotobiotic pigs (Neef *et al.*, 1994a), has recently been described. Some non-pathogenic spirochaetes also can produce a degree of haemolysis that can be difficult to differentiate from that of *S. hyodysenteriae* (Olson and Fales, 1983; Torp and Thorensen, 1992). Haemolysis by *S. hyodysenteriae* is enhanced by either removing a plug of agar, or slashing the surface of the growth, and reincubating the plate (Kunkle and Kinyon, 1988; Bélanger and Jacques, 1991). Some weakly haemolytic intestinal spirochaetal isolates also may produce indole (Lemcke and Burrows, 1981), and some can have a "typical" *S. hyodysenteriae* biochemical profile in the API-zym test (Lee *et al.*, 1993a; Milner *et al.*, 1995).

Alternative approaches to the diagnosis of SD include serological tests and/or assays of cell mediated immunity to provide evidence of infection, or the detection or demonstration of specific antigens or nucleic acids in the faeces of affected animals. Serological assays include a microtitre agglutination test (MAT) (Joens *et al.*, 1978a), various ELISA tests (Joens *et al.*, 1982; Wright *et al.*, 1989), and Western immunoblot analysis (Smith *et al.*, 1990). The ELISA tests are more sensitive than MAT (Egan *et al.*, 1983). The ELISA plate-coating antigens used have been either spirochaetal cell extracts or purified LPS preparations. Use of the former antigens may result in cross-reactivity and false positive diagnosis, in cases where other intestinal spirochaetes infect pigs, whilst LPS-based ELISAs are serotype-specific. Therefore knowledge of the serotypes of *S. hyodysenteriae* present in the area or herd to be tested is needed, with the possible necessity of testing each serum against a range of LPS extracts. For example, a serological survey was conducted in Western Australia using LPS from the three main serogroups (A, B and E) in that state (Mhoma *et al.*, 1992). Smith *et al.* (1991a), working in Victoria, used a whole cell extract as antigen in ELISA. They then tested the positive sera in Western blot analysis with whole outer membrane extracts, using reactivity with the LPS to confirm their results. It is generally accepted that none of these serological tests can reliably identify individual infected pigs, but they can be used to determine whether SD is present in a herd. Generally, at least 40 serum samples should be tested per herd (Egan *et al.*, 1983; Mhoma *et al.*, 1992). Assays of cell mediated immunity also have been developed to provide evidence of infection (Jenkins *et al.*, 1982), but generally they are not convenient for routine diagnostic use.

Numerous assays that detect specific antigens of *S. hyodysenteriae* have been developed to assist with the diagnosis of SD. These mainly have been used to identify culture isolates, but some have been applied directly to clinical material. These tests have utilized polyclonal rabbit antiserum raised against formalised *S. hyodysenteriae* cells. This serum has been used for identification of *S. hyodysenteriae* isolates in a growth inhibition test (Lemcke and Burrows, 1979), and a microtitre agglutination test (Lemcke and Burrows, 1981). Unfortunately, when this serum is used in indirect immunofluorescence tests (IFAT) directly on pig faeces, cross-reaction may be seen with other intestinal spirochaetes. The sera must therefore be cross-absorbed with one or more of these organisms before it can be used (Hudson and Alexander, 1976; Lemcke *et al.*, 1982). The process of absorption reduces the sensitivity of the test, and, together with the non-availability of commercially-prepared and standardised absorbed antiserum, has resulted in IFAT being used only at a few specialized centres, mainly in the UK. The absorbed serum also has been used in a slide agglutination test (Burrows and Lemcke, 1981) and in a microscopic agglutination test (Lysons, 1991) to help identify cultured isolates. Monoclonal antibodies (Mabs) have been raised against *S. hyodysenteriae*. The use of Mabs in the above tests, by capture ELISA or labelled immunomagnetic beads, may have more potential than polyclonal sera for detecting the organisms. A Mab developed against a 16 kDa outer membrane polypeptide of *S. hyodysenteriae* has been used in a capture ELISA (Sellwood *et al.*, 1992), but unfortunately this antigen may not be expressed *in vivo*, and not all strains possess the gene encoding it (Sellwood *et al.*, 1995; Turner *et al.*, 1995). Monoclonal antibodies against LPS also have been prepared (Alderton *et al.*, 1993; Lee and Hampson, 1994a), but these are serotype restricted. Recently, a Mab against an outer membrane protein of *S. hyodysenteriae* has been prepared (Lee and Hampson, unpublished data), and this has considerable potential for use in the diagnosis of SD.

There has been considerable interest in developing nucleic acid-based diagnostic reagents for detecting and identifying *S. hyodysenteriae*. These have involved either the use of specific nucleic acid probes, or the development of polymerase chain reaction (PCR) tests. Such approaches have the potential advantage of being extremely sensitive and specific, so that they can detect carrier animals, and of being rapid, without the need for culturing faeces. To date, no one test has met all these criteria, although considerable progress has been made. The first probe was based on a radiolabelled plasmid-like DNA molecule that is present in many isolates of *S. hyodysenteriae* (Joens and Marquez, 1988). Subsequently probes based on 16S ribosomal RNA (Jensen *et al.*, 1990), whole chromosomal DNA (Combs and Hampson, 1991), and various random chromosomal

sequences (Combs and Hampson, 1992; Sotiropoulos *et al.*, 1993) have been developed (the latter three in Australia). These probes may be applied directly to faecal samples, and have been reported to detect around  $10^3$  to  $10^4$  *S. hyodysenteriae* cells in 0.1 g of faeces (Jensen *et al.*, 1990; Sotiropoulos *et al.*, 1993). Unfortunately, these probes are only available at a few specialized centres, are quite technically difficult to use, and are not readily available for routine diagnostic purposes. More recently, PCR assays, in which specific *S. hyodysenteriae* sequences are amplified before detection, have been developed for diagnosis. A PCR based on the sequence for the 16 kDa outer membrane protein described by Sellwood and colleagues (1992) failed to detect all strains of *S. hyodysenteriae* (Atyeo, 1992). Details of a much more sensitive PCR assay recently were published (Elder *et al.*, 1994). This was said to detect 1-10 *S. hyodysenteriae* cells in 0.1 g of faeces, but since it also relied on the use of Southern hybridization to achieve these levels of detection, it is not a particularly practical technique. A DNA probe and PCR also have been developed by Canadian researchers. Whilst this PCR could detect as little as 10 *S. hyodysenteriae* whole cells, the detection limit in seeded faeces was only  $10^4$  cells per 0.1 g of faeces (Harel and Forget, 1995). Workers in the authors' laboratory have developed a PCR assay that consistently detects  $10^2$  -  $10^4$  cells in 0.2 g of faeces, without the use of Southern hybridization (Atyeo, Combs and Hampson, unpublished data). This is a relatively straightforward procedure, and, with suitable further modification, has considerable potential for diagnostic use.

### Treatment

In outbreaks of SD, all pigs in an infected group should be treated by water medication if possible, whilst severely affected animals should be isolated and treated by injection. Treatment should continue for 1-2 weeks, after which antibiotics should be administered at prophylactic concentrations via the feed for at least another 2-3 weeks. The disease may become more severe if ineffective drugs are used, and problems may occur following removal of a partially-effective treatment regimen (Olsen and Rodabaugh, 1978). In Australia, the most commonly used medications are tiamulin, lincomycin/spectinomycin, dimetridazole, and tylosin. Carbadox is no longer available. Strains that are resistant to one or other of the four commonly-used drugs have been isolated in Australia, but multiple-drug-resistant strains have not been reported (Smith *et al.*, 1991b; Buller and Hampson, 1994). Tiamulin is the most effective drug *in vitro* (Méssier *et al.*, 1990), and to date only one resistant strain has been identified, from a property in Queensland (Buller and Hampson, 1994). Dimetridazole is a relatively cheap drug, but soon may no longer be available for use in Australian pigs. Although *in vitro* resistance to lincomycin is commonly found, the drug still may be effective *in vivo* (Smith *et al.*, 1991b), possibly through its inhibitory action on other members of the colonic microflora. It is important to reduce the possibility of reinfection of treated pigs from the environment, by thorough cleaning and disinfection of pens and dung channels.

### Prevention and control

#### Control in infected herds

There are two major approaches for controlling SD in infected herds. These are to control the disease through medication and management, or to eradicate the infection from the herd. The approach taken depends on a number of factors, including the primary function of the herd (eg., high-health status breeding, versus mainly fattening); the ongoing costs of the disease to the herd; the size of the herd (eradication is more difficult in very large herds); the capital resources that are available to invest in controlling the disease; whether or not other infected piggeries are located nearby (especially within a three kilometre radius); the design of the piggery; the design and state of repair of the buildings and associated equipment; the presence of other production-limiting diseases in the herd; the quality of the herd's genetics; and the availability of good management, co-operative piggery staff, and experienced veterinary advice.

Swine dysentery can be eradicated from herds and, although the procedure is expensive, it is generally cost-effective, due to improvements in subsequent productivity and profitability (Wood and Lysons, 1988). Several methods have proved successful, depending on the circumstances of the piggery. These are:

(a) Depopulation of the herd, followed by thorough cleaning and disinfection, removal of rodents, and maintaining the property free of stock for at least six weeks, if possible. This is followed by restocking with high-health status pigs, of good genotype. This procedure is most beneficial when other diseases are present, and the genotype is poor. Destocking is best undertaken when pig prices are high (ie., July to December), while restocking is best undertaken when pig prices are low. The procedure is expensive, and requires good management skills.

(b) Medication of all pigs with drugs such as tiamulin, at full therapeutic levels, for three to 10 weeks. Concurrently there should be a programme of thorough cleaning and disinfection, removal of effluent, and rodent control. When disease no longer appears to be present, it is advisable to remove medication to determine whether SD has been eradicated from the herd. This procedure does not always work, and disease may reappear later. Once effective vaccines become available, these may be used to reduce the level of infection in the herd prior to medication.

(c) Application of medicated-early-weaning. Here piglets, from medicated sows, are medicated, weaned at 10 days of age, or older, and removed to weaner sheds at sites at least three kilometres from the breeding herd (Alexander *et al.*, 1980). The piglets are grown out there, or at other sites. Variations on these procedures are now used widely in the USA, and have proved effective at controlling a number of infectious diseases, resulting in greatly-improved growth performance.

In herds that are unable to eradicate the infection, control can be achieved by using prophylactic levels of medication in the feed, and managerial changes to reduce losses. Lowering stocking density reduces stresses on the pigs, and decreases the rate of transmission. Modification to pen design and effluent disposal, as well as staff education and provision of protective clothing and boot-dips, can help to reduce spread between groups. In these situations the use of vaccines may also be very helpful. Serotype-specific inactivated vaccines have been developed in Australia (Coloe *et al.*, 1989; Hampson *et al.*, 1993), and their design and application have been greatly assisted by a thorough knowledge of the number and distribution of *S. hyodysenteriae* serotypes in Australia (Hampson *et al.*, 1994). Inactivated vaccines do not give complete protection from SD, but their use may increase overall herd performance (Hampson, 1989). *Serpulina hyodysenteriae* bacterins have occasionally been reported to exacerbate the severity of the disease (Olsen and Dayalu, 1994). In the USA, a protein-reduced LPS/endotoxin extract has been used experimentally to confer a high level of protection against SD (Wannemuehler *et al.*, 1990). This preparation will soon be available commercially. Subunit vaccines developed through recombinant DNA technology also have been prepared. An endoflagellar protein, produced in *Escherichia coli*, was shown to protect mice from developing lesions of SD (Boyden *et al.*, 1989). This vaccine is not commercially available for use in pigs. Recently, colonization with haemolysin-negative mutants of *S. hyodysenteriae*, which have reduced virulence, has been shown to provide partial protection against challenge with fully-virulent strains (Hyatt *et al.*, 1994). This may provide another possible approach to increasing productivity in endemically-infected herds. Finally, feeding highly-digestible diets can inhibit colonization by *S. hyodysenteriae* (Siba *et al.*, 1994; Siba, Pethick, and Hampson, unpublished data), and may provide an alternative means of control, if economically-viable protective diets can be identified.

#### *Prevention of swine dysentery in herds free of infection.*

Herds that are free of SD should take precautions to prevent its entry. The best measure is to maintain a closed herd. Where pigs must be introduced, they should come from a source that is certified free of SD. Incoming pigs should be quarantined in

separate accommodation, and fed antimicrobial-free food, for at least a month before entry into the main herd. It is useful to house the animals with a few cull sows which may act as indicators of the introduction of a new disease. Genetic improvement also can be achieved through the use of artificial insemination. In the future, the use of PCR or other techniques to identify carrier animals should greatly assist control. Other sources of infection also should be prevented from entering the herd. It is advisable to remove rodents, and to prevent entry of food trucks, commercial salespeople, and other visitors, by the use of external security fences and gates. Essential visitors should be provided with clean clothing and boots. It may be advisable to restrict the use of antimicrobial therapy, so that if infection is introduced it can be rapidly identified and controlled.

## Intestinal spirochaetosis and spirochaetal colitis

### Introduction

Porcine colitis is a general term for diarrhoea of swine in which the large intestine is the only organ affected, and clinical signs and pathological changes are generally mild. Where weakly beta-haemolytic intestinal spirochaetes distinct from the strongly haemolytic *S. hyodysenteriae* are involved in the aetiology, the condition has been termed intestinal spirochaetosis (IS), although a range of other terms also have been used, including spirochaetal diarrhoea (Taylor *et al.*, 1980), spirochaetal colitis (SC) (Hampson, 1991), and colonic spirochaetosis (Duhamel *et al.*, 1995c; Girard *et al.*, 1995). In this review, the terms IS and SC are used to describe separate conditions caused by distinct types of weakly beta-haemolytic intestinal spirochaetes. Collectively, the conditions are distinct from non-specific colitis, which is a non-infectious condition, which appears to be predisposed to by the physical nature of the diet, particularly pelleted feed (Smith and Nelson, 1987; Connor, 1992).

Pigs with IS develop soft faeces, or diarrhoea with little or no blood, lose condition and have reduced weight gain. Histologically, IS often is characterized by the presence of large numbers of spirochaetal cells attached end-on to the colonic epithelium, forming a false-brush border (Taylor *et al.*, 1980; Spearman *et al.*, 1988; Jacques *et al.*, 1989). This characteristic pathological appearance has been recorded in humans with diarrhoea, where again it has been referred to as IS (Harland and Lee, 1967; Lee *et al.*, 1971). Although the end-on attachment of spirochaetes is considered to be pathognomonic for IS, it may not always be apparent (Taylor *et al.*, 1980). Furthermore, in human beings the non-pathogenic intestinal spirochaete, *Brachyospira aalborgi*, also shows a similar pattern of attachment (Hovind-Hougen *et al.*, 1982; Nielsen *et al.*, 1983).

Intestinal spirochaetosis of swine was first documented in 1980, when Taylor *et al.* (1980) experimentally infected pigs with pure cultures of a weakly beta-haemolytic spirochaete strain (P43/6/78) that was isolated from a pig with mucoid diarrhoea. The infected pigs developed mucoid diarrhoea, containing a small amount of blood. Histological lesions were consistent with colitis, including the end-on attachment of large numbers of spirochaetes to the colonic epithelium. Although the disease has since been reported in several other countries (Spearman *et al.*, 1988), few attempts have been made either to characterize the spirochaetes involved in IS, or determine their relationships to *S. hyodysenteriae* and the non-pathogenic weakly haemolytic *S. innocens*. Current knowledge suggests that members of a distinct species of weakly beta-haemolytic spirochaetes, with the proposed name *Serpulina pilosicoli*, are the principal aetiological agent of IS in both pigs and humans (Trott, Stanton, Jensen, Duhamel, Johnson and Hampson, unpublished data).

Although SC has a similar clinical presentation to IS, other groups of weakly haemolytic spirochaetes are involved in the aetiology, and the end-on attachment of spirochaetes to the colonic epithelium is not a feature (Binek and Szynekiewicz, 1984; Fellström and Gunnarsson, 1994). The condition of SC is poorly documented, and has only been experimentally reproduced in ligated porcine colonic loops and gnotobiotic pigs (Binek and Szynekiewicz, 1984; Neef *et al.*, 1994a). To date, infection of conventional pigs with intestinal spirochaetes other than *S. hyodysenteriae* and *S. pilosicoli* has not resulted in diarrhoea or inflammatory change in the colon. In the field, SC is less frequently encountered than IS, and therefore the condition will not be discussed in detail.

To clarify the significance of IS and SC to the Australian pig industry, the research efforts at Murdoch have been concentrated on taxonomic characterization of the weakly beta-haemolytic intestinal spirochaetes, and the development of rapid diagnostic tests that can be used to differentiate the various groups of porcine intestinal spirochaetes. Animal models have been used to test isolates for pathogenicity, and molecular typing methods for epidemiological studies. These recent developments will be discussed with particular reference to IS and SC in Australia. Although an understanding of the disease processes has been significantly improved, many aspects of IS and SC, particularly host susceptibility and response, pathogenicity, and epidemiology, remain to be investigated.

#### *Clinical description*

The clinical signs of IS and SC are almost identical; and are similar to those seen in other forms of porcine colitis. They may also mimic the early stages of SD (Taylor *et al.*, 1980; Duncan and Lysons, 1987). The disease IS commonly affects pigs in the immediate post-weaning stage, although it has been reported, during the growing and finishing period. Adult pigs are rarely affected, however outbreaks have been reported in breeding stock recently-introduced into herds (Taylor, 1992). The condition has been reported in well-managed herds (Wilkinson and Wood, 1987), and in pigs suffering with concurrent illness, such as pneumonia, salmonellosis, trichuriasis, or intestinal adenopathy (Jacques *et al.*, 1989; Taylor, 1992; Girard *et al.*, 1995). A common pre-disposing factor is the introduction of new feed in the preceding week (Spearman *et al.*, 1988).

In experimental infections, diarrhoea associated with IS occurs following an incubation period of between 3 and 20 days (Taylor *et al.*, 1980; Andrews and Hoffman, 1982; Trott and Hampson, unpublished data). In the early stages of infection, affected pigs generally develop loose, sticky faeces that adhere to the pen floor. The consistency of the faeces then changes to that of fresh cement or porridge, and may take on a glistening appearance. Some individuals may not develop further clinical signs, but most pigs rapidly develop mucoid diarrhoea. Occasionally, flecks of blood and plugs of mucus may be present in the faeces, although dysentery is not a characteristic feature. Descriptions of grey-green diarrhoea and grey scours also have been reported (Taylor, 1980; Andrews and Hoffman, 1982; Spearman *et al.*, 1988). Diarrhoea is usually self-limiting, and lasts between 2 and 14 days, although recovered animals may relapse and develop clinical signs again. Affected pigs are characterized by faecal staining around the perineum, appear ill-thrifty ("hairy"), have a tucked-up appearance, and are often febrile (Taylor *et al.*, 1980; Taylor, 1992). Inappetance usually is not apparent. Pigs that develop loose faeces may lose weight, in contrast to pigs with chronic diarrhoea which have reduced live-weight gain and poor feed efficiency. Whilst morbidity may be high, mortality is rare, although pregnant sows and chronically affected weaners have been found dead with the only lesions being suggestive of IS (Taylor, 1980).

Clinical signs associated with SC are not well documented, although in studies where the aetiological agent was a weakly beta-haemolytic spirochaete which otherwise phenotypically resembled *S. hyodysenteriae*, the main clinical presentation was reported to be a mucoid, grey-green diarrhoea (Binek and Szykiewickz, 1984; Fellström and Gunnarsson 1994). In pathogenicity tests in gnotobiotic pigs, Neef *et al.*, (1994a) found that pathogenic weakly haemolytic intestinal spirochaete strains not associated with IS caused mucoid diarrhoea and occasional dysentery, whereas those associated with IS caused watery diarrhoea.

#### *Economic significance*

The economic significance of IS and SC is unknown, however the severity and morbidity of the diseases are less than that associated with SD (Taylor, 1992). The greatest economic concern is probably the loss of production associated with a failure to gain weight, and a reduced feed conversion ratio. There also may be significant costs associated with treatment, as the conditions often return upon the cessation of antimicrobial therapy. The diseases can be difficult to eradicate from a herd, although major control measures are not usually undertaken.

### Aetiology

#### *The taxonomic classification of the weakly beta-haemolytic spirochaetes.*

The weakly beta-haemolytic porcine intestinal spirochaetes are genetically heterogeneous, although they share many similar phenotypic properties. Although they were all originally thought to belong to the non-pathogenic species, *Serpulina innocens*, multilocus enzyme electrophoresis (MEE) was used to divide these spirochaetes into four distinct genetic groups (Lee *et al.*, 1993b) (Figure 1). As well as *S. innocens*, two new species were proposed (*S. intermedius* and *S. murdochii*), together with a new genus and species (*Anguillina coli*). These names were provisional, and required verification by other methods of genomic comparison. *Anguillina coli* was genetically distinct from the other groups of spirochaetes, and contained only isolates with 4-6 periplasmic flagellae (*S. hyodysenteriae* isolates and *S. innocens* isolates have 8-14 periplasmic flagellae). The group included a large number of isolates obtained from pigs suffering from IS-like conditions, including the first strain recovered from the condition (P43/6/78). Intestinal spirochaetes isolated from human beings with diarrhoea also were included in this group (Lee *et al.*, 1993c; Lee and Hampson, 1994b).

*Anguillina coli* was confirmed to represent a distinct genetic group by using DNA-DNA reassociation assays (Lee *et al.*, 1993c), and 16S ribosomal DNA sequence analysis (Stanton *et al.*, 1995). Sequencing of the 16S ribosomal DNA gene was performed on eleven intestinal spirochaetes, including P43/6/78, other strains of *A. coli*, and representatives from *S. hyodysenteriae*, *S. innocens*, *S. intermedius*, and *S. murdochii*. A high degree of sequence homology was found between these intestinal spirochaetes, suggesting that they all belonged within a single genus. Therefore P43/6/78 and other spirochaetes with 4-6 periplasmic flagellae were not sufficiently distinct to constitute a new genus, and should be placed within a new species in the genus *Serpulina*. This finding also was suggested by Duhamel *et al.* (1993b), who used DNA-DNA reassociation to compare porcine and human strain, all with 4-6 periplasmic flagellae, with the type strains of *S. hyodysenteriae* and *S. innocens*.

#### *Intestinal spirochaetosis*

Additional genetic and phenotypic characterization of P43/6/78 and other porcine and human isolates with 4-6 periplasmic flagellae (formally proposed as *Anguillina coli*), isolated from cases of IS, was undertaken to confirm the results obtained from 16S rDNA sequencing and DNA-DNA reassociation (Trott, Stanton, Jensen, Duhamel, Johnson and Hampson, unpublished data). Whilst many of the tests were performed for taxonomic purposes, the identification of simple characteristic traits that were specific to these organisms also was important for diagnostic reasons. The results confirmed that these organisms belonged within the genus *Serpulina*, but that they were distinct from *S. hyodysenteriae*, *S. innocens*, and spirochaetes in the two proposed groups, *S. intermedius* and *S. murdochii*. It is therefore proposed to name the group *Serpulina pilosicoli* sp. nov. (*pilosicoli*: L gen. n. "of the hairy colon"). The strain P43/6/78 was chosen to be the type strain, and was lodged with the American Type Culture Collection under the accession number ATCC 51139.

*Serpulina pilosicoli* strains have a characteristic morphology. When compared with the other *Serpulina* species, they have fewer flagellae (4-6), more pointed ends, are more slender (0.18-0.3  $\mu\text{m}$ ), and are generally shorter (4-12  $\mu\text{m}$ ) (Figure 2). *Serpulina pilosicoli* strains grow more rapidly, can metabolise D-ribose, hydrolyze hippurate and are more sensitive to rifampicin and spiramycin than are strains of *S. hyodysenteriae* and *S. innocens*. Strains of *S. pilosicoli* are the only intestinal spirochaetes within the genus *Serpulina* that are known to attach by one end to the colonic epithelium. It is believed that *S. pilosicoli* is the principal agent of IS in both pigs and humans. The severity of disease resulting from infection with *S. pilosicoli* is likely to depend on strain virulence, dietary influences, and the host response and bacterial synergisms occurring in the large intestine.

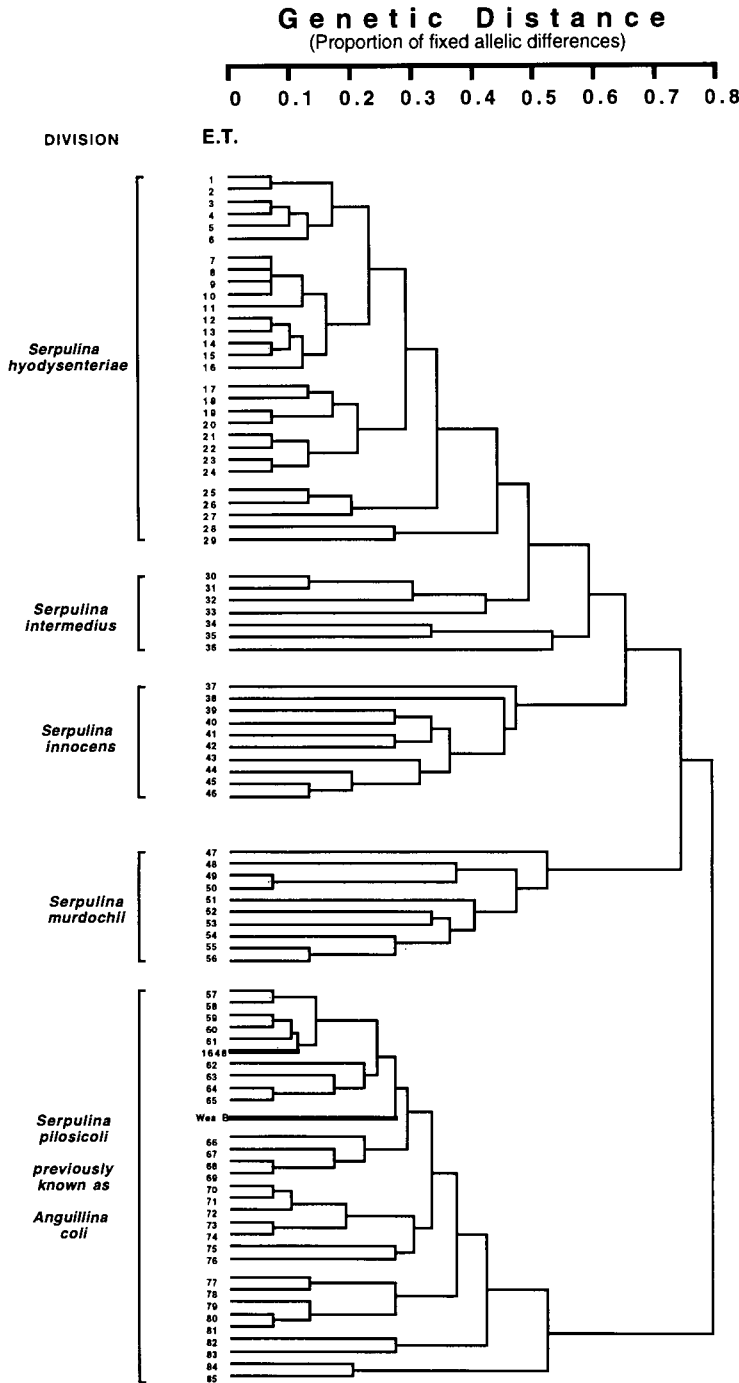


Figure 1. Phenogram of genetic distance (as the percentage of fixed allelic differences) illustrating genetic relationships among 189 porcine intestinal spirochaetes divided into 86 electrophoretic types (ETs 1-85 and 1648) and one human intestinal spirochaete (ET WesB), clustered by the unweighted pair-group method of arithmetic averages. The raw data was obtained by examining and comparing the electrophoretic mobilities of 15 constitutive enzymes for each isolate. Five major groups of spirochaetes are identified. Modified with permission from Lee et al. (1993b).





Figure 2. Transmission electron micrograph of one end of a cell of *Serpulina pilosicoli* strain P43/6/78 showing five periplasmic flagellae and insertion discs, and the pointed end of the cell ( $\times 54,000$ ). This is the strain recovered from the first recorded case of IS (Taylor *et al.*, 1980).

#### *Spirochaetal colitis*

The involvement of the other groups of weakly beta-haemolytic spirochaetes in intestinal disease has not been fully determined, however it would appear that certain isolates of *S. intermedium*, and perhaps *S. innocens*, may be capable of inducing SC. *Serpulina intermedium* was so named because it was phenotypically and genetically intermediate between *S. hyodysenteriae* and *S. innocens* (Lee *et al.*, 1993b). In addition, it closely resembled a group of indole positive, weakly beta-haemolytic spirochaetes designated as *Treponema (Serpulina) hyodysenteriae* biotype 2. These spirochaetes were associated with diarrhoea in pigs in Poland, and were enteropathogenic in ligated porcine colonic loops (Binek and Szykiewickz, 1984). Swedish researchers also consider that these spirochaetes have pathogenic potential (Fellström and Gunnarsson, 1994; Fellström *et al.*, 1994). Despite these findings, pure cultures of an indole positive, weakly beta-haemolytic strain (PWS/A), since suggested as the type strain of *S. intermedium* (ATCC 51140), failed to induce clinical signs or lesions when orally inoculated into experimental pigs (Hudson and Alexander, 1976). Similarly, an indole positive, weakly beta-haemolytic Australian strain (889) did not cause disease in conventional pigs (Lee *et al.*, 1993b). Neef *et al.* (1994a) failed to induce disease in gnotobiotic pigs with a similar isolate. Although *S. innocens* is considered to be non-pathogenic on the basis of pathogenicity testing in small numbers of conventional pigs (Kinyon and Harris, 1979), some isolates designated as *S. innocens* by MEE caused colitis in gnotobiotic pigs (Neef *et al.*, 1994a). Furthermore, a strongly beta-haemolytic spirochaete which was otherwise phenotypically distinct from *S. hyodysenteriae*, and resembled *S. innocens* using MEE, also induced disease in gnotobiotic

pigs, but not in conventional animals (Neef *et al.*, 1991; Lysons, *et al.*, 1992). The pathogenic potential of isolates of *S. murdochii* remains unknown, but epidemiological data generally support their classification as non-pathogenic commensals, not associated with SC (Lee *et al.*, 1993b).

#### *Pathological changes*

##### *Gross lesions*

Gross lesions associated with IS are limited to the caecum and colon, and may be very subtle, particularly in the early stages of the disease. Post-mortem examination soon after the onset of clinical signs often reveals a large increase in the volume of the caecum and colon, which are generally flaccid, whilst the serosal surface may be oedematous (Taylor *et al.*, 1980; Duncan and Lysons, 1987). This may be accompanied by oedema of the mesenteric and colonic lymph nodes (Taylor *et al.*, 1980; Andrews and Hoffman, 1982). The colonic contents are fluid or occasionally mucoid, and may be frothy or distended with gas (Taylor *et al.*, 1980; Spearman *et al.*, 1988; Taylor, 1992). The mucosal surface may appear normal, slightly congested with a glistening appearance, or slightly hyperaemic, with occasional ulcerations (Andrews and Hoffman, 1982; Duncan and Lysons, 1987; Taylor, 1992). In many instances, these are the only gross lesions. In individual animals in which the disease progresses, some visible inflammation may occur, resulting in multifocal ulcerative colitis or mucohaemorrhagic colitis (Girard *et al.*, 1989). The mucosa becomes thickened, and local petechial and ecchymotic haemorrhages may appear on the luminal surface (Taylor, 1992). In chronic cases, and in resolving lesions, occasional mucus clots, necrotic plaques or diptheritic foci may be present (Taylor, 1980; Taylor *et al.*, 1980; Duncan and Lysons, 1987; Wilkinson and Wood, 1987). Gross lesions associated with SC are not documented, but may be similar to those of IS.

##### *Histological lesions*

Histological lesions associated with IS range from being very mild, to severe, resembling those seen in SD. Mild inflammatory changes are generally apparent, and have been described as catarrhal, multifocal erosive, or ulcerative colitis (Andrews and Hoffman, 1982; Girard *et al.*, 1989). The inflammatory response is characterized by diffuse infiltration of mononuclear, or occasionally polymorphonuclear cells into the *lamina propria*, and subepithelial oedema, congestion, and capillary dilatation. The luminal surface may show patchy epithelial necrosis, usually accompanied by desquamation or shallow erosions. A mass of basophilic material (cellular debris or fibrinous exudate), containing large numbers of spirochaetes, is found adherent to the damaged epithelium. Commonly, moderate to large numbers of *Balantidium coli* cells are found in close approximation to epithelial erosions or within the underlying *lamina propria* (Taylor *et al.*, 1980; Andrews and Hoffman, 1982; Duncan and Lysons, 1987; Spearman *et al.*, 1988). Intestinal crypts may be normal (Girard *et al.*, 1989), or dilated and filled with cellular debris, spirochaetes and polymorphonuclear cells (Taylor *et al.*, 1980; Andrews and Hoffman, 1982; Spearman *et al.*, 1988; Taylor, 1992). Crypt hyperplasia and accelerated turnover of epithelial cells may be apparent (Girard *et al.*, 1995). An increase in goblet cells may occur, resulting in an increased production of mucus (Taylor *et al.*, 1980). The most characteristic finding in IS is the intimate, end-on attachment of large numbers of spirochaetes to epithelial cells, to produce a false brush border effect. This attachment may be apparent over the entire colonic surface, or it may be distributed in patches. Spirochaetes may be found within goblet cells, or, rarely, invading the *lamina propria*. The end-on attachment of spirochaetes may not be apparent in every case of IS in which *S. pilosicoli* has been isolated (Taylor *et al.*, 1980; Girard *et al.*, 1995).

Histological lesions associated with SC have not been described in detail. Neef *et al.* (1994a) reported thickening of the *lamina propria*, increased crypt depth, hyperaemia, dilated crypts with occasional abscessation, and mild inflammatory changes similar to those described above. However, as previously mentioned, the spirochaetes associated with SC do not attach end-on to the colonic mucosa, and hence this appearance is not a feature of the condition.

### Electron microscopy

Electron microscopy has been used to examine the colonic mucosa from pigs naturally infected with *S. pilosicoli* (Jacques *et al.*, 1989; Duhamel *et al.*, 1993a). Scanning electron micrographs revealed a carpet of spirochaetes on the luminal surface, making the underlying mucosa scarcely discernable. Transmission electron micrographs showed polar attachment of large numbers of spirochaetes to the apical portions of columnar epithelial cells, which are devoid of microvilli. Bacteria were observed invaginating into the terminal web cytoplasm, however they did not penetrate past the host cell plasmalemma.

### Pathogenesis

The pathogenic mechanisms of *S. pilosicoli* are not well understood, but are thought to differ from those of *S. hyodysenteriae*. The watery diarrhoea that is observed in IS may arise from interference with colonic absorption, but this has not been confirmed. Pathogenic mechanisms of the other weakly beta-haemolytic spirochaetes associated with SC also are not known.

### Pathogenicity testing

Reports of Koch's postulates being fulfilled for *S. pilosicoli* are rare (Taylor *et al.*, 1980; Andrews and Hoffman, 1982; Taylor, 1992). Recent pathogenicity tests have shown that IS can be reproduced in an animal model (Trott *et al.*, 1995). Initial experiments used strain 1648, which was isolated in NSW from a pig with catarrhal colitis, consistent with lesions of IS. This isolate, and a related human strain (Wes B), induced watery diarrhoea and stunting during a 21 day test in day-old chicks, orally inoculated with approximately  $3 \times 10^8$  spirochaetes. Histological and electron microscopic examination of the caecae of chicks from both groups revealed massive colonization of the mucosal surface by intestinal spirochaetes, and pathological changes in the underlying epithelial cells (Figure 3). In contrast, a strain of *S. innocens* did not colonize or produce lesions in a second group of SPF chicks, demonstrating that this animal model can be used to differentiate between pathogenic and non-pathogenic spirochaetes. This day-old SPF chick model has the capacity to be used to determine the pathophysiological mechanisms and virulence potential of *S. pilosicoli* isolates, and also may aid in determining the pathogenic potential of intestinal spirochaete isolates associated with SC.

Strain 1648 also has been used to infect newly-weaned pigs (Trott and Hampson, unpublished data), and the results are summarised in Table 1. In each of two experiments, six pigs were inoculated with approximately  $10^{10}$  spirochaetes in early log phase culture. In the first experiment, two pigs that were colonized with 1648 developed mucoid diarrhoea by five days post inoculation. The weight of the infected pigs was less ( $P < 0.05$ ) than the control group. Two infected pigs and a third pig which became culture positive two days later, but did not develop diarrhoea, had the poorest growth rates. Both pigs had watery caecal and colonic contents, and spirochaetes with the same MEE electrophoretic type as 1648 were cultured from caecal and colonic scrapings. Histologically, mild typhlo-colitis was present in the two infected pigs, but attachment of spirochaetes by one end to the colonic epithelium was not demonstrated. No gross or histological lesions were observed in the remaining pigs.

In a second experiment, colonization initially occurred in all six inoculated pigs., four of which subsequently developed mucoid diarrhoea, but growth rate was not affected. Post-mortem examination revealed hyperaemic typhlo-colitis in one of the colonized pigs. Mild inflammatory changes were present histologically; however, spirochaetes were not attached by one end to the colonic epithelium. Future challenge experiments will examine the pathogenic potential of other isolates, using different diets, and pigs of different genotype, to attempt to reproduce the disease in a more consistent manner.

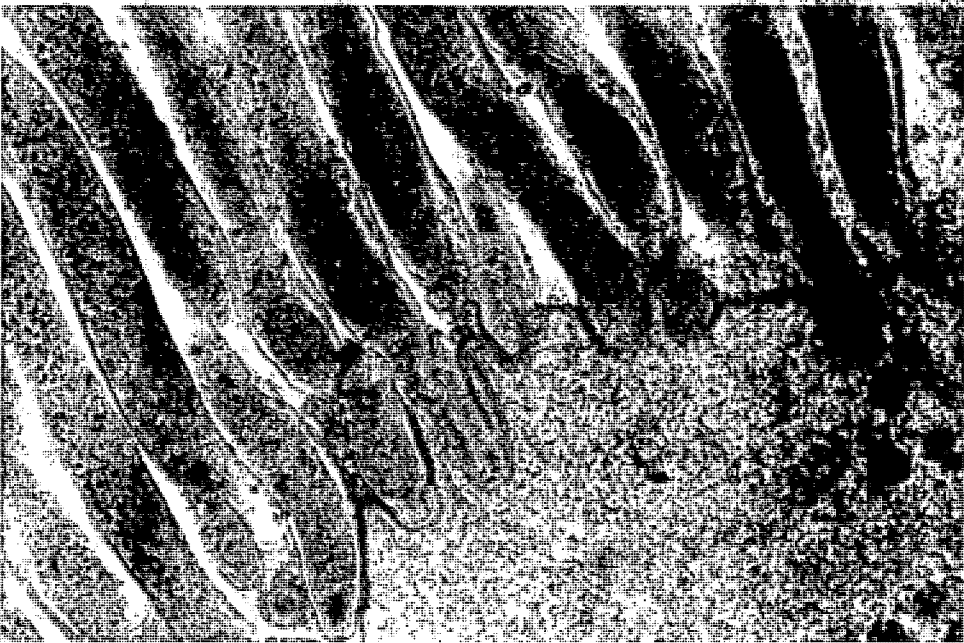
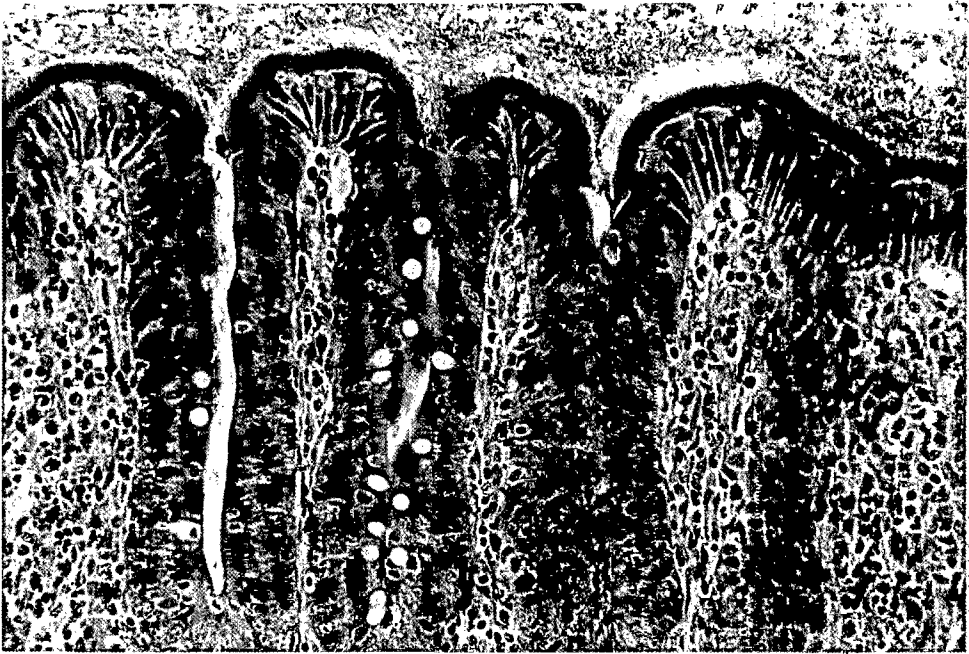


Figure 3. Micrographs showing histological (top panel [ $\times 400$ ]) and transmission electron (bottom panel [ $\times 41,917$ ]) appearance of caecal tissue from chicks infected with *S. pilosicoli* strain WesB. In the top panel, the spirochaetes are seen as a dense fringe on the surface of the columnar epithelium of the villous tips. In the electron micrograph, individual spirochaetes have invaginated into the host cell terminal web cytoplasm, but do not penetrate the cell membrane.

**Table 1. Results of oral infection in two experiments where six pigs were infected with *Serpulina pilosicoli* strain 1648.**

Group	Weight (kg) mean $\pm$ SD	No. of pigs colonized (days <sup>1</sup> post-inoculation)				No. of pigs with:	
		3	7	10	14	Diarrhoea	Lesions <sup>2</sup>
<b>Experiment 1:</b>							
Infected	8.2 $\pm$ 0.9*	2	1	1	3	2	2
Control <sup>3</sup>	9.7 $\pm$ 0.5	-	-	-	-	-	-
<b>Experiment 2:</b>							
Infected	8.4 $\pm$ 1.2	6	3	5	4	4	2
Control <sup>3</sup>	8.7 $\pm$ 0.6	-	-	-	-	-	-

<sup>1</sup>Post-mortem examination was carried out at 14 days post-inoculation. <sup>2</sup>Lesions defined as gross or microscopic evidence of typhlitis or colitis. \*Significantly different from control ( $P < 0.05$ ) in the one-tailed t-test at 5 days post-inoculation. <sup>3</sup>Control (n=6)

#### Pathogenic mechanisms

The ultrastructural appearance of experimentally-induced IS in day-old chicks suggests that diarrhoea may be caused by physical blockage of passive absorption, resulting from large numbers of spirochaetes becoming attached to the epithelium, and disrupting the microvilli. The presence of gap-like lesions between enterocytes, however, suggests that a secretory mechanism also may be involved. This explanation fits well with the clinical signs and histological lesions of IS in humans, but not in pigs. In pigs, direct attachment of spirochaetes to the colonic mucosa is sometimes patchy, or non-existent. Other bacterial virulence factors may be involved in triggering the mild inflammatory response that is seen in histological sections. It is reported that virulent *S. hyodysenteriae* cells are chemotactic to porcine mucin. In contrast, *S. pilosicoli* and non-virulent *S. hyodysenteriae* strains were not chemotactic (Milner and Sellwood, 1994). Unlike *S. hyodysenteriae*, *S. pilosicoli* cells do not colonize the colonic crypts to any great extent. These observations suggest that the two pathogenic spirochaetes have very different biological activities in the colon. The mechanism whereby *S. pilosicoli* causes disease in pigs and humans has not been elucidated. Increased bacterial fermentation in the large intestine may support mucosal colonization by *S. pilosicoli*, similar to *S. hyodysenteriae*. Wilkinson and Wood (1987) showed that IS did not recur in a herd after the energy and protein content of the diet was reduced, and protein of animal origin was added. The hypothesis that bacterial synergism may play a role in IS was supported by the findings of Neef *et al.* (1994a), who successfully reproduced watery diarrhoea and mild inflammatory changes, with no end-on attachment of spirochaetes, in gnotobiotic pigs first colonized with *Bacteroides vulgatus* and then infected with either of two *S. pilosicoli* strains. Variation in the virulence of different strains may be an important factor in governing the outcome of the infection.

#### Immunity

Intestinal spirochaetosis in pigs may be similar to the disease in humans, where immunocompromised patients or those in developing communities with poor health, diet, and/or hygiene, are more susceptible to infection with *S. pilosicoli* (Jones *et al.*, 1986; Tompkins *et al.*, 1986; Ruane *et al.*, 1989; Barrett, 1990; Kasbohrer *et al.*, 1990; Lee and Hampson, 1992). It has been shown previously that Aboriginal children with chronic diarrhoea may be colonized with the same strain of *S. pilosicoli* up to one year after the initial infection. However, it was not possible to determine whether the strain had been present for this extended period or whether its presence was the result of reinfection (Lee and Hampson, 1992; 1994b). Pigs treated for IS may redevelop clinical signs soon after antibiotic therapy has ceased, and this is usually attributed to reinfection (Taylor, 1992). Immunity to *S. hyodysenteriae* is largely serotype specific, and immunity to *S. pilosicoli* also

may depend upon the nature and distribution of cell envelope antigens. In the pathogenicity trials conducted in the authors' laboratory, only some of the pigs became colonized, and these animals subsequently developed clinical signs. These individuals may have not have generated an adequate immune response to the organism, and so became heavily colonized and developed disease. The chronic diarrhoea seen in human beings with IS, and occasionally in swine, may reflect a poorly developed immune response. Alternatively, it may be due to *S. pilosicoli* cells only having the capacity to colonize a damaged gastro-intestinal tract. Specific cellular receptors may be important in enterocyte attachment, as the spirochaetes often are found in massive numbers, closely associated with host cells at the luminal surface. This susceptibility to attachment may be a function of the animal's genotype. Recently a monoclonal antibody to a specific outer membrane protein of *S. pilosicoli* has been developed (Lee and Hampson, 1994a) and this Mab will be used to develop an ELISA to determine antibody titres to *S. pilosicoli*. These assays will be used in pathogenicity experiments to characterize the immune response in infected and convalescent animals.

### Epidemiology

#### *Species affected*

Intestinal spirochaetosis is primarily a disease of pigs and human beings, and *S. pilosicoli* commonly is isolated from both these species. Spirochaetes that are genetically and phenotypically similar to *S. pilosicoli* also have been isolated from poultry (McLaren *et al.*, 1994), and dogs (Duhamel *et al.*, 1995a; Duhamel *et al.*, 1995b), with clinical signs and lesions typical of IS, including the end-on attachment of spirochaetes to the colonic epithelium (Trampel *et al.*, 1994). Isolates of *S. pilosicoli*-like organisms also have been obtained from rheas, and ducks (Trott, Swayne, Stoutenburg and Hampson, unpublished data), and spirochaetes have been observed attached end-on to the colonic mucosa in histological sections obtained from rhesus monkeys (Takeuchi *et al.*, 1974), and opossums (Duhamel *et al.*, 1994).

Spirochaetal colitis is primarily a disease of pigs, however *S. intermedius*-like isolates have been obtained from poultry with intestinal disorders and poor production, and have caused mild typhlitis in experimentally infected birds (Dwars *et al.*, 1992; McLaren *et al.*, 1994).

#### *Incidence*

Confirmed cases of IS have been recorded from Canada (Spearman *et al.*, 1988; Girard *et al.*, 1989; Jacques *et al.*, 1989; Girard *et al.*, 1995), the United States (Duhamel *et al.*, 1993a), continental Europe (Fellström and Gunnarsson, 1994; Fellström *et al.*, 1994), the United Kingdom (Taylor *et al.*, 1980; Wilkinson and Wood, 1987; Taylor, 1992) and Australia (Hampson, 1991). The incidence of the disease in Australia is not known, but it is believed to be common. Intestinal spirochaetosis has been diagnosed from histological specimens obtained from pigs in Queensland, NSW and Victoria; furthermore *S. pilosicoli* has been frequently cultured from faecal samples taken from these herds. When improved methods of diagnosis are developed veterinarians and producers in Australia will recognize that IS is a common disease. A study in Western Australia, to assess the incidence of IS, and the ages at which pigs become susceptible to infection, and to identify risk factors for infection is presently being planned. The diagnostic and epidemiological typing tools that have been developed in the authors' laboratory will enable a clearer understanding of the disease to be obtained.

Spirochaetal colitis appears to be less prevalent than IS, and has been reported only in continental Europe (Binek and Szykiewickz, 1984; Fellström and Gunnarsson, 1995) and the United Kingdom (Neef *et al.*, 1994a).

### Transmission

The faecal/oral route is the most likely method by which IS spreads between pigs. Dogs, birds, human beings and other animal species also may be colonized with *S. pilosicoli*, so there may be the potential for cross-species transmission. It is not considered that pigs suffering with IS pose a significant risk to healthy industry-workers, as *S. pilosicoli* strains sharing the same MEE profile have not been obtained from both pigs and another animal species (Lee and Hampson, 1994b). Rodents may be reservoirs of *S. hyodysenteriae*, and also can be colonized by weakly beta-haemolytic spirochaetes. However, all those that have been examined have belonged to the non-pathogenic group *S. murdochii* (Trott, Swayne, Stoutenburg and Hampson, unpublished data).

### Strain diversity

Using MEE, a considerable genetic diversity has been found amongst strains of *S. pilosicoli* (Lee *et al.*, 1993b). The majority of isolates obtained from confirmed cases of IS, including 1648, are closely related to P43/6/78, the strain originally associated with IS (Taylor *et al.*, 1980). This cluster contains isolates from diverse geographic regions including Canada, the United States, the UK and Australia (Lee *et al.*, 1993b; Lee and Hampson, 1994b). Organisms closely related to P43/6/78 have been the major focus of pathogenicity experiments at Murdoch. More distantly related strains also will be tested to determine if pathogenicity is a feature of the entire genetic group, or whether it is confined to this small cluster of organisms.

### Diagnosis

In the past, diagnosis of IS has involved isolation of weakly beta-haemolytic spirochaetes from pigs with signs of colitis. Diagnostic tests are needed which rapidly and efficiently differentiate *S. pilosicoli* from other intestinal spirochaetes which are either non-pathogenic, or may be involved in SC.

*Serpulina pilosicoli* cells grow on selective trypticase soy blood agar plates, as previously described for SD; however, growth is sometimes slower than for *S. hyodysenteriae*, and plates may require anaerobic incubation for at least six days before visible growth occurs. *Serpulina pilosicoli* cells cannot be differentiated from other spirochaetes on the basis of colony morphology, and only differ from *S. hyodysenteriae* in strength of haemolysis. Media containing spectinomycin alone, spectinomycin, vancomycin and colistin, or BJ media (spectinomycin, vancomycin, colistin, rifampicin and spiramycin) have all previously been used to cultivate *S. pilosicoli*. Recent research indicates that the latter method may have a depressive effect on the rate of cell recovery, because of the increased sensitivity of *S. pilosicoli* strains to rifampicin and spiramycin (Trott and Hampson, unpublished data).

Until recently MEE and transmission electron microscopic examination of bacterial cell ultrastructure, were the only diagnostic methods available for confirming that weakly beta-haemolytic strains belonged to the species *S. pilosicoli*. These are powerful, but laborious techniques that are not suitable for a general diagnostic laboratory. The availability of simple phenotypic tests that are specific for *S. pilosicoli* will aid diagnosis considerably (Trott, Stanton, Jensen, Duhamel, Johnson and Hampson, unpublished data). *Serpulina pilosicoli* cells can ferment D-ribose, a test which can be performed easily by growing test cells in liquid medium with and without D-ribose as the only known energy source. Utilization of this sugar then can be determined by measuring a reduction in pH, or by determining growth spectrophotometrically. The hippurate cleavage test is another simple procedure that appears to be definitive for *S. pilosicoli* (Fellström and Gunnarsson, 1994; Duhamel *et al.*, 1995b; Trott, Stanton, Jensen, Duhamel, Johnson and Hampson, unpublished data). *Serpulina pilosicoli* strains often have a variable reaction in the API-zym test, used for differentiating the other weakly beta-haemolytic spirochaetes, and *S. hyodysenteriae*. Phenotypic criteria for determining the identity of an unknown intestinal spirochaete are shown in Table 2.

All of these procedures still require incubation of plates for at least six days, followed by further growth in liquid media. Clearly, there is a need for the development of rapid, specific diagnostic tests for detecting *S. pilosicoli*. Recently, a specific polymerase

chain reaction (PCR) assay (Park *et al.*, 1995a; Park *et al.*, 1995b), and an indirect fluorescent antibody test (IFAT) (Lee and Hampson, 1994a; 1995) have been developed for these purposes. The PCR was designed using a signature sequence identified in the 16S rDNA region that was specific for *S. pilosicoli* strains. The test is completely specific, and is more sensitive than culture for detecting bacterial cells in clinical samples (Atyeo and Hampson, 1995). Unfortunately, the technique currently used in the authors' laboratory still requires primary incubation of cultures for five days. The IFAT was developed using a monoclonal antibody to an outer membrane protein of *S. pilosicoli* (Lee and Hampson, 1995). This test has been used to detect *S. pilosicoli* cells in the faeces of experimentally infected pigs. The sensitivity of culture, PCR and the IFAT will be compared in future on-farm studies aimed at investigating the epidemiology of IS.

#### Differential diagnosis

The colitis that occurs in IS and SC is similar to that seen in other conditions, some of which have been described only recently. In their early stages, both conditions may be confused with SD, particularly if blood and mucus are passed. Whilst the aetiology of IS is now clear, the role of other intestinal spirochaetal groups in SC also needs further definition. Rapid diagnostic methods are in their infancy, and confirmation of diagnosis is still based upon the degree of haemolysis on trypticase soy blood agar, and the biochemical tests outlined in Table 2. Other non-spirochaetal infectious conditions that could be confused with IS and SC clinically include post-weaning colibacillosis, rotavirus infection, salmonellosis, proliferative enteritis, *Clostridium perfringens* type A diarrhoea, and colitis caused by *Yersinia pseudotuberculosis* (Taylor *et al.*, 1987; Neef and Lysons, 1994). Pathogenic synergisms between intestinal spirochaetes and these organisms have not been reported, although the lesions of trichuriasis, intestinal adenopathy, and salmonellosis may be colonized by intestinal spirochaetes similar to *S. pilosicoli* (Beer and Rutter, 1972; Taylor, 1992; Girard *et al.*, 1995). Non-infectious or non-specific colitis may be caused by the physical form of the feed alone, and may be associated with a form of hypersensitivity to pelleted feed (Smith and Nelson, 1987; Connor, 1992). Neef *et al.* (1994b) reported that a diet implicated in field cases of colitis strongly influenced the development of lesions in the large intestine due to enteropathogenic *E. coli*. The influence of diet on the development of IS and SC requires further investigation.

**Table 2.** Differentiation of the five recognized groups of porcine intestinal spirochaetes by their haemolysis pattern on trypticase soy blood agar, biochemical reactions and utilization of sugars.

Test	<i>S. hyodysenteriae</i>	<i>S. intermedius</i>	<i>S. innocens</i>	<i>S. murdochii</i>	<i>S. pilosicoli</i>
Haemolysis	strong	weak	weak	weak	weak
Indole	+	+	-	-	-
Hippurate	-	-	-	-	+
API-zym*	1	1	2	3	4
Cellubiose*	-	-	+	+	+
L-fucose*	-	+	+	+	+
D-galactose*	+	+	+	v	
D-ribose*	-	-	-	-	+

1, alpha-glucosidase positive, alpha-galactosidase negative. 2, alpha-glucosidase negative, alpha-galactosidase positive. 3, alpha-glucosidase negative, alpha-galactosidase negative. 4, variable reactions including positive reactions for both enzymes. v, variable fermentation

\* Note: only two isolates of each of the proposed species *S. intermedius* and *S. murdochii* have been examined for utilization of soluble sugars, and thus these results require further validation using additional strains.



### Treatment

Treatment and control of IS and SC are generally modelled on procedures developed for SD. Antimicrobial therapy by feed or water medication is generally recommended. The sensitivities to 20 different antimicrobials for three porcine *S. pilosicoli* strains (including P43/6/78 and 1648) have been examined using the agar dilution method of Kitai *et al.* (1979) and compared with the results for *S. hyodysenteriae* and *S. innocens* (Trott, Stanton, Jensen and Hampson, unpublished data). This method is laborious to perform, but gives more reliable results for intestinal spirochaetes than the antimicrobial disc method. Generally, the three *S. pilosicoli* strains were sensitive to all of the common drugs used for treating SD, at the same or lower levels than required for *S. hyodysenteriae*. These drugs included the olaquinox derivative, carbadox (not available for use in Australia but used commonly in the United States for the treatment of SD), lincomycin, metronidazole, tiamulin and tylosin. Interestingly, all strains, including *S. hyodysenteriae* and *S. innocens*, were sensitive to tetracycline, a drug which is not commonly employed for the treatment of SD or IS.

From these observations, drugs used for the treatment of SD should be effective against *S. pilosicoli*, and should also be effective against spirochaetes involved in SC, however, pigs with IS commonly develop clinical signs after treatment has ceased (Wilkinson and Wood, 1987; Spearman *et al.*, 1988). Dimetridazole resistance by an unidentified weakly beta-haemolytic spirochaete associated with IS has been reported, and in this case the condition improved when lincospectin was used (Garden, 1977). Adjunctive therapy, such as improved management and pen hygiene, has given inconsistent results (Smith and Nelson, 1987; Wilkinson and Wood, 1987), whilst dietary modification often has alleviated the problem (Wilkinson and Wood, 1987; Spearman *et al.*, 1988). Similar inconsistent results with treatment have been reported in Australian piggeries where IS has been diagnosed. Reinfection after treatment is common, and may be influenced by the predisposing factors mentioned previously.

### Control

A theoretical basis for control and prevention of IS and SC is largely dependent on a thorough knowledge of epidemiological aspects of the disease, yet these have not been elucidated. The control methods mentioned previously for SD may be effective against IS and SC, but the reduced severity of both diseases generally does not warrant such drastic methods. Taylor (1992) reported that medicated-early-weaning, as proposed by Alexander *et al.* (1980), and previously discussed for SD, is the only recorded practice which has eliminated intestinal spirochaetes from a group of animals. It is intended to investigate the interactions between diet and colonization with weakly beta-haemolytic spirochaetes, as reported for SD, to determine if dietary manipulation can influence or control the development of IS and SC.

### Conclusion

Knowledge and understanding of the intestinal spirochaetes infecting pigs has increased greatly over the last few years. Largely this has been the result of the recent rapid development and application of molecular-based techniques for taxonomic studies, and for strain differentiation. It is now clear that *S. hyodysenteriae* is made up of a variety of different strains, and that these vary in their biological properties. The ability to identify individual strains has allowed detailed epidemiological studies to be undertaken at the level of the farm, state, and the nation. More is known about strain variety and distribution of *S. hyodysenteriae* in Australia than in any other country.

The depth of knowledge of the weakly beta-haemolytic spirochaetes also has been increased enormously. These now have been divided into at least four species, with strains of *S. pilosicoli* being recognized as the causal agents of IS, and some strains of *S. intermedius*, and possibly *S. innocens*, being associated with SC. Using this knowledge it is now possible to study the epidemiology of these conditions, to develop improved methods for diagnosis, and to formulate appropriate control measures.

The diagnosis of SD and IS has been facilitated by the recent development of PCR and/or monoclonal antibody-based techniques. These procedures are rapid and specific, but, more importantly, they may have sufficiently high sensitivity to allow the detection of carrier animals. In turn, the ability to identify these animals will reduce the occurrence of between-herd transmission of infection, and greatly improve control at a national level. The application of new management procedures, particularly medicated-early-weaning, also seem likely to greatly reduce the prevalence of SD and other intestinal spirochaetal infections in Australia, as has occurred in the USA. Although new vaccines are being developed for the control of SD, no completely effective products are currently available. Their eventual widespread use is likely to be coupled with other more traditional, or novel, means of control.

Finally, one of the most interesting and important new developments with implications for the control of SD has been the discovery that pigs fed highly-digestible, low residue diets, are not susceptible to the disease. The availability of such diets, at a reasonable cost, has the potential to revolutionize the control of SD, and possibly other intestinal spirochaetal infections. A detailed study of their mode of action also should shed new light on the pathogenesis of these complex and important infections.

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### References

- ACHACHA, M. and MESSIER, S. (1991). Identification of *Treponema hyodysenteriae* and *Treponema innocens* using two four-hour identification systems. *Journal of Veterinary Diagnostic Investigation*. 3:211-214.
- ADACHI, Y., SUEYOSHI, M., MIYAGAWA, E., MINATO, H. and SHOYA, S. (1985). Experimental infection of young broiler chicks with *Treponema hyodysenteriae*. *Microbiological Immunology*. 29:683-688.
- ALDERTON, M.R., SMITH, S.C. and COLOE, P.J. (1993). Identification of a novel group of *Serpulina hyodysenteriae* isolates by using a lipopolysaccharide-specific monoclonal antibody. *Journal of Clinical Microbiology*. 31:1326-1328.
- ALEXANDER, T.J.L. and TAYLOR, D.J. (1969). The clinical signs, diagnosis and control of swine dysentery. *The Veterinary Record*. 85:59-63.
- ALEXANDER, T.J.L., THORNTON, K., BOON, G., LYSONS, R.J. and GUSH, A.F. (1980). Medicated early weaning to obtain pigs free from pathogens endemic in the herd of origin. *The Veterinary Record*. 106:114-119.
- ANDREWS, J.J. and HOFFMAN, L.J. (1982). A porcine colitis caused by a weakly beta-hemolytic treponeme (*Treponema nosoinnocens*?). *Proceedings of the Annual Convention of the American Association of Veterinary Laboratory Diagnosticians*. pp. 395-402.
- ANON. (1995). Detected increases in swine dysentery. *Pigs*. 11:6.
- ARGENZIO, R.A., WHIPP, S.C. and GLOCK, R.D. (1980). Pathophysiology of swine dysentery: colonic transport and permeability studies. *The Journal of Infectious Diseases*. 142:676-684.
- ATYEO, R.F. (1992). Polymerase chain reaction for the detection of *Serpulina hyodysenteriae*. BSc Hons Thesis. Murdoch University, Western Australia.
- ATYEO, R.F. and HAMPSON, D.J. (1995). Diagnosis of swine dysentery and intestinal spirochaetosis by the use of polymerase chain reaction on faecal samples. In "Manipulating Pig Production V", p. 186, eds D.P. Hennessy and P.D. Cranwell. (Australasian Pig Science Association: Werribee).
- BARRETT, S.P. (1990). Intestinal spirochaetes in a Gulf Arab population. *Epidemiology and Infection*. 104:261-266.
- BAUM, D.H. and JOENS, L.A. (1979). Serotypes of beta-haemolytic *Treponema hyodysenteriae*. *Infection and Immunity*. 25:792-796.
- BEER, R.J. and RUTTER, J.M. (1972). Spirochaetal invasion of the colonic mucosa in a syndrome resembling swine dysentery following experimental *Trichuris suis* infection in weaned pigs. *Research in Veterinary Science*. 29:593-595.
- BELANGER, M. and JACQUES, M. (1991). Evaluation of the An-Ident system and an indole spot test for the rapid differentiation of porcine treponemes. *Journal of Clinical Microbiology*. 29:1727-1729.
- BINEK, M. and SZYMKIEWICZ, Z. (1984). Physiological properties and classification of strains of *Treponema* sp. isolated from pigs in Poland. *Comparative Immunology Microbiology and Infectious Diseases*. 7:141-148.
- BLAHA, T. and GUNTER, H. (1985). On ultrastructure and motility of spirochaetes. *Acta Veterinaria Hungarica*. 33:3-12.
- BLAND, A.P., FROST, A.J. and LYSONS, R.J. (1995). Susceptibility of porcine ileal enterocytes to the cytotoxin of *Serpulina hyodysenteriae* and the resolution of epithelial lesions: an electron microscopic study. *Veterinary Pathology*. 32:24-35.
- BOYDEN, D.A., ALBERT, F.G. and ROBINSON, C.S. (1989). Cloning and characterization of *Treponema hyodysenteriae* antigens and protection in a CF-1 mouse model by immunization with a cloned endoflagellar antigen. *Infection and Immunity*. 57:3808-3815.
- BUDDLE, J.R. (1985). "Bacterial and Fungal Diseases of Pigs", pp. 179-186. (Australian Government Publishing Service: Canberra).

- BULLER, N.B. and HAMPSON, D.J. (1994). Antimicrobial susceptibility testing of *Serpulina hyodysenteriae*. *Australian Veterinary Journal*. **71**:211-214.
- BURROWS, M.R. and LEMCKE, R.M. (1981). Identification of *Treponema hyodysenteriae* by a rapid slide agglutination test. *The Veterinary Record*. **108**:187-189.
- CANALE-PAROLE, E. (1977). Physiology and evolution of spirochaetes. *Bacteriological Reviews*. **41**:181-204.
- CANALE-PAROLE, E. (1984). The Spirochetes. In "Bergey's manual of systematic bacteriology, Volume 1", pp. 38-70, eds J.G. Holt and N.R. Krieg. (Williams and Wilkins: Baltimore).
- CHIA, S.P. and TAYLOR, D.J. (1978). Factors affecting the survival of *Treponema hyodysenteriae* in dysenteric pig faeces. *The Veterinary Record*. **103**:68-70.
- COLOE, P.J., GERRATY, N.L. and GELDARD, H. (1989). Vaccination against *Treponema hyodysenteriae*: artificial and natural challenge results. In "Manipulating Pig Production II", p. 273, eds J.L. Barnett and D.P. Hennessy. (Australasian Pig Science Association: Werribee).
- COMBS, B.G. and HAMPSON, D.J. (1991). Use of a whole chromosomal probe for identification of *Serpulina hyodysenteriae*. *Research in Veterinary Science*. **50**:286-289.
- COMBS, B.G. and HAMPSON, D.J. (1992). Identification of *Serpulina hyodysenteriae* using a cloned DNA sequence. *Proceedings of the 12th International Pig Veterinary Society Congress, The Hague, The Netherlands*. p. 150.
- COMBS, B.G. and HAMPSON, D.J. and HARDERS, S.J. (1992). Typing of Australian isolates of *Treponema hyodysenteriae* by serology and by DNA restriction endonuclease analysis. *Veterinary Microbiology*. **31**:273-285.
- CONNOR, J.F. (1992). Nonspecific colitis. *Proceedings of the Australian Association of Pig Veterinarians Conference, Adelaide*. pp. 79-80.
- CUTLER, R. and GARDNER, I. (1988). "A blueprint for pig health research." (Australian Pig Research Council: Canberra).
- CWYK, W.M. and CANALE-PAROLE, E. (1979). *Treponema succinificiens* sp. nov., an anaerobic spirochete from the swine intestine. *Archives of Microbiology*. **122**:231-239.
- DUHAMEL, G.E., HUNSAKER, B.D., MATHIESON, M.R. and MOXLEY, R.A. (1995a). Intestinal spirochetosis and giardiasis in a Beagle pup with diarrhea. *Veterinary Pathology*. *In press*.
- DUHAMEL, G.E. and JOENS, L.A. (1994). "Laboratory procedures for diagnosis of swine dysentery." (American Association of Veterinary Laboratory Diagnosticians, Inc.: Columbia).
- DUHAMEL, G.E., MATHIESEN, M.R., SCHAFFER, R.W., RAMANATHAN, M. and JOHNSON, J.L. (1993b). Description of a new species of spirochete, *Serpulina coli* sp. nov. associated with intestinal spirochetosis of swine and human beings. *Proceedings of the Annual Meeting of the Conference of Research Workers in Animal Diseases*. Chicago. p. 14.
- DUHAMEL, G.E., MUNIAPPA, N., GARDNER, I., ANDERSON, M.A., BLANCHARD, P.C., DeBEY, B.M., MATHIESON, M.R. and WALKER, R.L. (1995c). Porcine colonic spirochaetosis: a diarrhoeal disease associated with a newly-recognised species of intestinal spirochaetes. *Pig Veterinary Society Spring Meeting*, Stratford.
- DUHAMEL, G.E., MUNIAPPA, N., MATHIESON, M.R., JOHNSON, J.L., TOH, J., ELDER, R.O. and DOSTER, A.R. (1995b). Certain canine weakly beta-hemolytic spirochetes are phenotypically and genotypically related to spirochetes associated with human and porcine intestinal spirochetosis. *Journal of Clinical Microbiology*. *In press*.
- DUHAMEL, G.E., MUNIAPPA, N., TARARA, R.P., ANDERSON, M.A., BLANCHARD, P.C., BARR, B.C. and DeBEY, B.M. (1994). Comparative pathology of naturally occurring intestinal spirochaetosis of human beings and animals. *Veterinary Pathology*. **31**:612.
- DUHAMEL, G.E., RAMANATHAN, M., BERNARD, R.J., NEWMAN, M.C. and ERICKSON, E.D. (1992). Application of restriction fragment length polymorphism typing to epidemiological tracing of *Serpulina hyodysenteriae*. *Proceedings of the 12th International Pig Veterinary Society Congress, The Hague, The Netherlands*. p. 276.
- DUHAMEL, G.E., RAMANATHAN, M., GARDNER, I., ANDERSON, M.A., WALKER, R.L. and HAMPSON, D.J. (1993a). Intestinal spirochetosis of swine associated with weakly  $\beta$ -hemolytic spirochetes distinct from *Serpulina innocens*. *Proceedings of the Annual Convention of the American Association of Veterinary Laboratory Diagnosticians, Las Vegas*. p. 53.
- DUNCAN, A.L. and LYSONS, R.J. (1987). Diagnosis of colitis in pigs. *The Veterinary Record*. **121**:430.
- DWARS, R.M., DAVELAAR, F.G. and SMIT H.F. (1992). Spirochaetosis in broilers. *Avian Pathology*. **21**:261-273.
- EGAN, I.T., HARRIS, D.L. and HILL, H.T. (1982). Prevalence of swine dysentery, transmissible gastroenteritis, and pseudorabies in Iowa, Illinois, and Missouri swine. *Proceedings of the United States Animal Health Association*. pp. 497-502.
- EGAN, I.T., HARRIS, D.L. and JOENS, L.A. (1983). Comparison of the microtitre agglutination test and the enzyme-linked immunosorbent assay for the detection of herds affected with swine dysentery. *American Journal of Veterinary Research*. **44**:1323-1328.
- ELDER, R.O., DUHAMEL, G.E., SCHAFFER, R.W., MATHIESON, M.R. and RAMANATHAN, M. (1994). Rapid detection of *Serpulina hyodysenteriae* in diagnostic specimens by polymerase chain reaction. *Journal of Clinical Microbiology*. **32**:1497-1502.
- FELLSTROM, C. and GUNNARSSON, A. (1994). A biochemical reaction scheme for porcine intestinal spirochetes. *Proceedings of the International Pig Veterinary Society Congress, Bangkok, Thailand*. p. 145.
- FELLSTROM, C., PETERSSON, B., GUNNARSSON, A., UHLEN, M. and JOHANSSON, K. (1994). Classification of intestinal porcine spirochetes by sequence analysis of 16S rRNA and biochemical methods. *Proceedings of the 13th International Pig Veterinary Society Congress, Bangkok, Thailand*. p. 146.
- FERNIE, D.S., RIPLEY, P.H. and WALKER, P.D. (1983). Swine dysentery: protection against experimental challenge following single dose parenteral immunization with inactivated *Treponema hyodysenteriae*. *Research in Veterinary Science*. **35**:217-221.
- FISHER, L.F. and OLANDER, H.J. (1981). Shedding of *Treponema hyodysenteriae*, transmission of disease, and agglutination response of pigs convalescent from swine dysentery. *American Journal of Veterinary Research*. **42**:450-455.
- GARDEN, S. (1977). Treatment of spirochaetosis. *The Veterinary Record*. **101**:250.
- GIRARD, C., JACQUES, M. and HIGGINS, R. (1989). Colonic spirochetosis in piglets. *Canadian Veterinary Journal*. **30**:68.

- GIRARD, C., LEMARCHAND, T. and HIGGINS, R. (1995). Porcine colonic spirochetosis: a retrospective study of eleven cases. *Canadian Veterinary Journal*. **36**:291-294.
- GLOCK, R.D., KINYON, J.M. and HARRIS, D.L. (1978). Transmission of *Treponema hyodysenteriae* by canine and avian vectors. *Proceedings of the 3rd International Pig Veterinary Society Congress, Zagreb*. KB63.
- GREER, J.M. and WANNEMUEHLER, M.J. (1989a). Comparison of the biological responses produced by lipopolysaccharide and endotoxin of *Treponema hyodysenteriae* and *Treponema innocens*. *Infection and Immunity*. **57**:717-723.
- GREER, J.M. and WANNEMUEHLER, M.J. (1989b). Pathogenesis of *Treponema hyodysenteriae*: Induction of interleukin-1 and tumor necrosis factor by a treponema butanol/water extract (endotoxin). *Microbiological Pathology*. **7**:279-288.
- GRIFFIN, R.M. and HUTCHINGS, D.A. (1980). Swine dysentery: observations on the frequency of latent infection. *The Veterinary Record*. **107**:559.
- HALTER, M.R. and JOENS, L.A. (1988). Lipooligosaccharides from *Treponema hyodysenteriae* and *Treponema innocens*. *Infection and Immunity*. **56**:3152-3156.
- HAMPSON, D.J. (1989). Vaccines and the control of swine dysentery. In "Manipulating Pig Production II", pp. 246-248. eds J.L. Barnett and D.P. Hennessy. (Australasian Pig Science Association: Werrisbee).
- HAMPSON, D.J. (1991). New developments in research on swine dysentery and spirochaetal colitis. *Pig News and Information*. **12**:233-235.
- HAMPSON, D.J., COMBS, B.G., HARDERS, S.J., CONNAUGHTON, I.D. and FAHY, V.A. (1991). Isolation of *Treponema hyodysenteriae* from a wild rat living on a piggery. *Australian Veterinary Journal*. **68**:308.
- HAMPSON, D.J., CUTLER, R. and LEE, B.J. (1992). Virulent *Serpulina hyodysenteriae* from a pig in a herd free of clinical swine dysentery. *The Veterinary Record*. **131**:318-319.
- HAMPSON, D.J., MALTAS, C.D., STEPHENS, C.P., MCKECHNIE, K. and BULLER, N.B. (1994). Serogroups of Australian isolates of *Serpulina hyodysenteriae*. *Australian Veterinary Journal*. **71**:347.
- HAMPSON, D.J., MHOMA, J.R.L., COMBS, B. and BUDDLE, J.R. (1989). Proposed revisions to the serological typing systems for *Treponema hyodysenteriae*. *Epidemiology and Infection*. **102**:75-84.
- HAMPSON, D.J., ROBERTSON, I.D. and MHOMA, J.R.L. (1993). Experiences with a vaccine being developed for the control of swine dysentery. *Australian Veterinary Journal*. **70**:18-20.
- HAMPSON, D.J. and STANTON, T.B. (1996). "Intestinal Spirochaetes in Domestic Animals and Humans", (CAB International: Wallingford). *In press*.
- HAREL, J., BELANGER, M., FORGET, C. and JACQUES, M. (1994). Characterisation of *Serpulina hyodysenteriae* isolates of serotypes 8 and 9 from Quebec by restriction endonuclease fingerprinting and ribotyping. *Canadian Journal of Veterinary Research*. **58**:302-305.
- HAREL, J. and FORGET, C. (1995). DNA probe and polymerase chain reaction procedure for the specific detection of *Serpulina hyodysenteriae*. *Molecular and Cellular Probes*. **9**:111-119.
- HARLAND, W.A. and LEE, F.D. (1967). Intestinal spirochaetosis. *British Medical Journal*. **3**:718-719.
- HARRIS, D.L. (1984). The epidemiology of swine dysentery as it relates to the eradication of the disease. *The Compendium on Continuing Education*. **6**:S83-S88.
- HARRIS, D.L., GLOCK, R.D., CHRISTENSEN, C.R. and KINYON, J.M. (1972). Swine Dysentery. I. Inoculation of pigs with *Treponema hyodysenteriae* (new species) and reproduction of the disease. *Veterinary Medicine Small Animal Clinician*. **67**:61-64.
- HARRIS, D.L. and LYSONS, R.J. (1992). Swine Dysentery. In "Diseases of Swine." 7th edn, pp. 599-616. eds A.D. Leman, B.E. Straw, W.L. Mengeling, S. D'Allaire and D.J. Taylor. (Iowa State University Press: Ames, Iowa).
- HOVIND-HOUGEN, K., BIRCH-ANDERSEN, A., HENRIK-NIELSEN, R., ORLDNI, M., PEDERSEN, J.O., TEGLBJAERG, P.S. and THAYSEN, E.H. (1982). Intestinal spirochaetosis: morphological characterisation and cultivation of the spirochaete *Brachyspira aalborgi* gen. nov. sp. nov. *Journal of Clinical Microbiology*. **16**:1127-1136.
- HUDSON, M.J. and ALEXANDER, T.J.L. (1976). Diagnosis of swine dysentery: Spirochaetes which may be confused with *Treponema hyodysenteriae*. *The Veterinary Record*. **99**:498-500.
- HUGHES, R., OLANDER, H.J., KANITZ, D.L. and QURESHI, S. (1977). A study of swine dysentery by immunofluorescence and histology. *Veterinary Pathology*. **14**:490-507.
- HUNTER, D. and WOOD, T. (1979). An evaluation of the API-zym system as a means of classifying spirochaetes associated with swine dysentery. *The Veterinary Record*. **104**:383-384.
- HYATT, D.R., ter HUURNE, A.A.H.M., van der ZIEJST, B.A.M. and JOENS, L.A. (1994). Reduced virulence of *Serpulina hyodysenteriae* haemolysin-negative mutants in pigs and their potential to protect pigs against challenge with a virulent strain. *Infection and Immunity*. **62**:2244-2248.
- JACQUES, M., GIRARD, C., HIGGINS, R. and GOYETTE, G. (1989). Extensive colonization of the porcine colonic epithelium by a spirochete similar to *Treponema innocens*. *Journal of Clinical Microbiology*. **27**:1139-1141.
- JENKINS, E.M. (1978). Development of resistance to swine dysentery. *Veterinary Medicine/Small Animal Clinician*. **73**:931-936.
- JENKINS, E.M., MOHAMMAD, A. and KLESIOUS, P.H. (1982). Evaluation of cell-mediated immune response to *Treponema hyodysenteriae*. *Proceedings of the 6th International Pig Veterinary Society Congress, Mexico City, Mexico*. p. 41.
- JENKINSON, S.R. and WINGAR, C.R. (1981). Selective medium for the isolation of *Treponema hyodysenteriae*. *The Veterinary Record*. **109**:384-385.
- JENSEN, N.S., CASEY, T.A. and STANTON, T.B. (1990). Detection and identification of *Treponema hyodysenteriae* by using oligodeoxynucleotide probes complementary to 16S rRNA. *Journal of Clinical Microbiology*. **28**:2717-2721.
- JENSEN, N.S. and STANTON, T.B. (1993). Comparison of *Serpulina hyodysenteriae* B78, the type strain of the species, with other *S. hyodysenteriae* strains using enteropathogenicity studies and restriction fragment length polymorphism analysis. *Veterinary Microbiology*. **36**:221-231.
- JOENS, L.A. (1980). Experimental transmission of *Treponema hyodysenteriae* from mice to pigs. *American Journal of Veterinary Research*. **41**:1225-1226.
- JOENS, L.A., DEYOUNG, D.W., GLOCK, R.D., MAPOTHER, M.E., CRAMER, J.D. and WILCOX III, H.E. (1985). Passive protection of segmented swine colonic loops against swine dysentery. *American Journal of Veterinary Research*. **46**:2369-2371.

- JOENS, L.A. and GLOCK, R.D. (1979). Experimental infection in mice with *Treponema hyodysenteriae*. *Infection and Immunity*. 25:757-760.
- JOENS, L.A., HARRIS, D.L. and BAUM, D.H. (1979). Immunity to swine dysentery in recovered pigs. *American Journal of Veterinary Research*. 40:1352-1354.
- JOENS, L.A., HARRIS, D.L., KINYON, J.M. and KAEBERLE, M.L. (1978a). Microtitre agglutination for detection of *Treponema hyodysenteriae* antibody. *Journal of Clinical Microbiology*. 8:293-298.
- JOENS, L.A. and KINYON, J.M. (1982). Isolation of *Treponema hyodysenteriae* from wild rodents. *Journal of Clinical Microbiology*. 15:994-997.
- JOENS, L.A. and MARQUEZ B, R. (1988). The diagnosis of swine dysentery using a labelled nucleic acid probe. *Proceedings of the 10th International Pig Veterinary Society Congress*, Rio de Janeiro, Brazil. p. 120.
- JOENS, L.A., NORD, N.A., KINYON, J.M. and EGAN, I.T. (1982). Enzyme-linked immunosorbent assay for detection of antibody to *Treponema hyodysenteriae* antigens. *Journal of Clinical Microbiology*. 15:249-252.
- JOENS, L.A., ROBINSON, I.M., GLOCK, R.D. and MATTHEWS, P.J. (1981). Production of lesions in gnotobiotic mice by inoculation with *Treponema hyodysenteriae*. *Infection and Immunity*. 31:504-506.
- JOENS, L.A., SONGER, J.G., HARRIS, D.L. and GLOCK, R.D. (1978b). Experimental infection with *Treponema hyodysenteriae* in guinea pigs. *Infection and Immunity*. 22:132-135.
- JOENS, L.A., SONGER, J.G., HARRIS, D.L. and KINYON, J.M. (1980). Comparison of selective culture and serological agglutination of *Treponema hyodysenteriae* for diagnosis of swine dysentery. *The Veterinary Record*. 106:245-246.
- JOENS, L.A., WHIPP, S.C., GLOCK, R.D. and NUESSEN, M.E. (1983). Serotype-specific protection against *Treponema hyodysenteriae* infection in ligated colonic loops of pigs recovered from swine dysentery. *Infection and Immunity*. 39:460-462.
- JONES, J.M., MILLER, J.N. and GEORGE, W.L. (1986). Microbiological and biochemical characterisation of spirochetes isolated from the feces of homosexual males. *Journal of Clinical Microbiology*. 24:1071-1074.
- KASBOHRER, A., GELDERBLUM, H.R., ARASTEH, K., HEISE, W., GROSSE, G., L'AGE, M., SCHONER, G., KOCH, M.A. and PAULI, G. (1990). Intestinale spirochaetose bei HIV-infektion-vorkommen, isolierung und morphologie der spirochaeten. *Deutsche medizinische Wochenschrift*. 115:1499-1506.
- KENNEDY, M.J., ROSNICK, D.K., ULRICH, R.G. and YANCEY Jr, R.J. (1988). Association of *Treponema hyodysenteriae* with the porcine intestinal mucosa. *Journal of General Microbiology*. 134:1565-1576.
- KENNEDY, M.J., ROSNICK, D.K., ULRICH, R.G. and YANCEY Jr, R.J. (1992). Identification and immunological characterisation of the major immunogenic antigens of serotypes 1 and 2 of *Serpulina hyodysenteriae*. *Proceedings of the 12th International Pig Veterinary Society Congress*, The Hague, The Netherlands. p. 273.
- KINYON, J.M. and HARRIS, D.L. (1979). *Treponema innocens*, a new species of intestinal bacteria and emended description of the type strain of *Treponema hyodysenteriae* Harris et al. *International Journal of Systematic Bacteriology*. 29:102-109.
- KINYON, J.M., HARRIS, D.L. and GLOCK, R.D. (1977). Enteropathogenicity of various isolates of *Treponema hyodysenteriae*. *Infection and Immunity*. 15:638-646.
- KITAI, K., KASHIWASAKI, M., ADACHI, Y., KUME, T. and ARAKAWA, A. (1979). In vitro activity of 39 antimicrobial agents against *Treponema hyodysenteriae*. *Antimicrobial Agents and Chemotherapy*. 15:392-395.
- KNOOP, F.C. (1979). Experimental infection of rabbit ligated ileal loops with *Treponema hyodysenteriae*. *Infection and Immunity*. 26:1196-1201.
- KUNKLE, R.A., HARRIS, D.L. and KINYON, J.M. (1986). Autoclaved liquid medium for propagation of *Treponema hyodysenteriae*. *Journal of Clinical Microbiology*. 24:669-671.
- KUNKLE, R.A. and KINYON, J.M. (1988). Improved selective medium for the isolation of *Treponema hyodysenteriae*. *Journal of Clinical Microbiology*. 26:2357-2360.
- LAU, T.T.A. and HAMPSON, D.J. (1992). The serological grouping system for *Serpulina (Treponema) hyodysenteriae*. *Epidemiology and Infection*. 109:255-263.
- LEE, B.J. and HAMPSON, D.J. (1994a). A monoclonal antibody reacting with the cell envelope of spirochaetes from intestinal spirochaetosis. *Proceedings of the 13th International Pig Veterinary Society Congress*, Bangkok, Thailand. p. 198.
- LEE, B.J. and HAMPSON, D.J. (1995). A monoclonal antibody reacting with the cell envelope of spirochaetes from cases of intestinal spirochaetosis in pigs and humans. *FEMS Microbiology Letters*. In press.
- LEE, F.D., KRASZWESKI, A., GORDON, J., HOWIE, G.R., McSEVENEY, D. and HARLAND, W.A. (1971). Intestinal spirochaetosis. *Gut*. 12:126-133.
- LEE, J.I. and HAMPSON, D.J. (1992). Intestinal spirochaetes colonising Aborigines from communities in the remote north of Western Australia. *Epidemiology and Infection*. 109:133-141.
- LEE, J.I. and HAMPSON, D.J. (1994b). Genetic characterisation of intestinal spirochaetes and their association with disease. *Journal of Medical Microbiology*. 40:365-371.
- LEE, J.I., HAMPSON, D.J., COMBS, B.G. and LYMBERY, A.J. (1993a). Genetic relationships between isolates of *Serpulina (Treponema) hyodysenteriae*, and comparison of methods for their subspecific differentiation. *Veterinary Microbiology*. 34:35-46.
- LEE, J.I., HAMPSON, D.J., LYMBERY, A.J. and HARDERS, S.J. (1993b). The porcine intestinal spirochaetes: identification of new genetic groups. *Veterinary Microbiology*. 34:273-285.
- LEE, J.I., McLAREN, A.J., LYMBERY, A.J. and HAMPSON, D.J. (1993c). Human intestinal spirochaetes are distinct from *Serpulina hyodysenteriae*. *Journal of Clinical Microbiology*. 31:16-21.
- LEMCKE, R.M. and BURROWS, M.R. (1979). A disc growth-inhibition test for differentiating *Treponema hyodysenteriae* from other intestinal spirochaetes. *The Veterinary Record*. 104:548-551.
- LEMCKE, R.M. and BURROWS, M.R. (1981). A comparative study of spirochaetes from the porcine alimentary tract. *Journal of Hygiene Cambridge*. 86:173-182.
- LEMCKE, R.M., BURROWS, M.R., LYSONS, R.J. and BEW, J. (1982). The differentiation of spirochaetes from the porcine gut. *Proceedings of the 6th International Pig Veterinary Society Congress*, Mexico City, Mexico. p. 237.
- LI, Z., BELANGER, M. and JACQUES, M. (1991). Serotyping of Canadian isolates of *Treponema hyodysenteriae* and description of two new serotypes. *Journal of Clinical Microbiology*. 29:2794-2797.
- LYMBERY, A.J., HAMPSON, D.J., HOPKINS, R.M., COMBS, B. and MHOMA, J.R.L. (1990). Multilocus enzyme electrophoresis for identification and typing of *Treponema hyodysenteriae* and related spirochaetes. *Veterinary Microbiology*. 22:89-99.

- LYSONS, R.J. (1991). Microscopic agglutination: a rapid test for identification of *Serpulina hyodysenteriae*. *The Veterinary Record*. **129**:314-315.
- LYSONS, R.J. (1992). Swine dysentery and other colitides. *The Pig Veterinary Society Proceedings*. **26**:69-74.
- LYSONS, R.J., KENT, K.A., BLAND, A.P., SELLWOOD, R., ROBINSON, W.F. and FROST, A.J. (1991). A cytotoxic haemolysin from *Treponema hyodysenteriae* - a probable virulence determinant in swine dysentery. *Journal of Medical Microbiology*. **34**:97-102.
- LYSONS, R.J., LEMCKE, R.M., BEW, J., BURROWS, M.R. and ALEXANDER, T.J.L. (1982). An avirulent strain of *Treponema hyodysenteriae* isolated from herds free of swine dysentery. *Proceedings of the 6th International Pig Veterinary Society Congress*, Mexico City, Mexico. p. 40.
- LYSONS, R.J., NEEF, N.A. and SMITH, W.J. 1992. "Colitis" of growing pigs. *Proceedings of the International Pig Veterinary Society Congress*, The Hague, The Netherlands. p. 290.
- MAPOTHER, M.E. and JOENS, L.A. (1985). New serotypes of *Treponema hyodysenteriae*. *Journal of Clinical Microbiology*. **22**:161-164.
- McLAREN, A.J., TROTT, D.J., SWAYNE, D.E., STOUTENBURG, J.W. and HAMPSON, D.J. (1994). Characterization of avian intestinal spirochetes. *Proceedings of the 45th North Central Avian Diseases Conference*, Des Moines. p. 66.
- McLENNAN, G.C. (1938). A condition of unknown aetiology affecting pigs. *Australian Veterinary Journal*. **14**:245.
- MESSIER, S., HIGGINS, R. and MOORE, C. (1990). Minimal inhibitory concentrations of five antimicrobials against *Treponema hyodysenteriae* and *Treponema innocens*. *Journal of Veterinary Diagnostic Investigation*. **2**:330-333.
- MEYER, R.C. (1978). Swine dysentery: a perspective. *Advances in Veterinary Science and Comparative Medicine*. **22**:133-158.
- MHOMA, J.R.L., HAMPSON, D.J. and ROBERTSON, I.D. (1992). A serological survey to determine the prevalence of infection with *Treponema hyodysenteriae* in Western Australia. *Australian Veterinary Journal*. **69**:81-84.
- MILNER, J.A. and SELLWOOD, R. (1994). Chemotactic response to mucin by *Serpulina hyodysenteriae* and other porcine spirochetes: potential role in intestinal colonization. *Infection and Immunity*. **62**:4095-4099.
- MILNER, J.A., TRUELOVE, K.G., FOSTER, R.J. and SELLWOOD, R. (1995). Use of commercial enzyme kits and fatty acid production for the identification of *Serpulina hyodysenteriae*: a potential misdiagnosis. *Journal of Veterinary Diagnostic Investigation*. **7**:92-97.
- NEEF, N.A. and LYSONS, R.J. (1994). Pathogenicity of a strain of *Yersinia pseudotuberculosis* isolated from a pig with porcine colitis syndrome. *The Veterinary Record*. **135**:58-63.
- NEEF, N., LYSONS, R.J., MAWDSLEY, A.C. and TRUELOVE, K.G. (1991). Colitis - Diet has been found to influence strongly the severity of colitis. *Annual Report of the Institute for Animal Health, Compton, England*. p. 63.
- NEEF, N.A., LYSONS, R.J., TROTT, D.J., HAMPSON, D.J., JONES, P.W. and MORGAN, J.M. (1994a). Pathogenicity of porcine intestinal spirochaetes in gnotobiotic pigs. *Infection and Immunity*. **62**:2395-2403.
- NEEF, N.A., McORIST, S., LYSONS, R.J., BLAND, A.P. and MILLER, B.G. (1994b). Development of large intestinal attaching and effacing lesions in pigs in association with the feeding of a particular diet. *Infection and Immunity*. **62**:2395-2403.
- NIBBELINK, S.K. and WANNEMUELLER, M.J. (1990). Effect of *Treponema hyodysenteriae* infection on mucosal mast cells and T cells in the murine caecum. *Infection and Immunity*. **58**:88-92.
- NIBBELINK, S.K. and WANNEMUELLER, M.J. (1992). An enhanced murine model for studies of *Serpulina (Treponema) hyodysenteriae*. *Infection and Immunity*. **60**:3433-3436.
- NIELSEN, R.H., ORHÖLM, M., PEDERSEN, J.O., HOVIND-HOUGEN, K., TEGLBJAERG, P.S. and THAYSEN, E.H. (1983). Colorectal spirochaetosis: clinical significance of the infection. *Gastroenterology*. **85**:62-67.
- NUESSEN, M.E., BIRMINGHAM, J.R. and JOENS, L.A. (1982). Biological activity of a lipopolysaccharide extracted from *Treponema hyodysenteriae*. *Infection and Immunity*. **37**:138-142.
- NUESSEN, M.E. and JOENS, L.A. (1982). Serotype-specific opsonisation of *Treponema hyodysenteriae*. *Infection and Immunity*. **38**:1029-1032.
- NUESSEN, M.E., JOENS, L.A. and GLOCK, R.D. (1983). Involvement of lipopolysaccharide in the pathogenicity of *Treponema hyodysenteriae*. *Journal of Immunology*. **131**:997-999.
- OLSEN, L.D. (1992). Survival time of swine dysentery inoculum in a lagoon. *Proceedings of the 12th International Pig Veterinary Society Congress*, The Hague, The Netherlands. p. 282.
- OLSEN, L.D. and DAYALU, K.I. (1994). Exacerbated onset of dysentery in swine vaccinated with inactivated adjuvanted *Serpulina hyodysenteriae*. *American Journal of Veterinary Research*. **55**:67-71.
- OLSEN, L.D. and FALES, W.H. (1983). Comparison of stained smears and culturing for identification of *Treponema hyodysenteriae*. *Journal of Clinical Microbiology*. **18**:950-955.
- OLSEN, L.D. and RÖDABAUGH, D.E. (1978). Clinical and pathological features of various drug-related problems in the control of swine dysentery. *Journal of the American Veterinary Medical Association*. **173**:843-851.
- PARIZEK, R., STEWART, R. and BROWN, K. (1985). Protection against swine dysentery with an inactivated *Treponema hyodysenteriae* bacterin. *Veterinary Medicine*. **80**:80-86.
- PARK, N.Y., CHUNG, C.Y., McLAREN, A.J., ATYEO, A.J. and HAMPSON, D.J. (1995a). PCR for identification of spirochaetes associated with intestinal spirochaetosis. *Proceedings of the 13th International Pig Veterinary Society Congress*, Bangkok, Thailand. p. 197.
- PARK, N.Y., CHUNG, C.Y., McLAREN, A.J., ATYEO, A.J. and HAMPSON, D.J. (1995b). Polymerase chain reaction for identification of human and porcine spirochaetes recovered from cases of intestinal spirochaetosis. *FEMS Microbiology Letters*. **125**:225-230.
- POHLENZ, J.F., WHIPP, S.C., ROBINSON, I.M. and FAGERLAND, J.A. (1984). A hypothesis on the pathogenesis of *Treponema hyodysenteriae* induced diarrhoea in pigs. *Proceedings of the 7th International Pig Veterinary Society Congress*, Ghent, Belgium. p. 178.
- PROHASKA, L. and LUKÁCS, K. (1984). Influence of the diet on the antibacterial effect of volatile fatty acids on the development of swine dysentery. *Zentralblatt für Veterinärmedizin*. **31**:779-785.
- RAMANATHAN, M., DUHAMEL, G.E., MATHIESEN, M.R. and MESSIER, S. (1993). Identification and partial characterisation of a group of weakly  $\beta$ -haemolytic intestinal spirochaetes of swine distinct from *Serpulina innocens* isolate B256. *Veterinary Microbiology*. **37**:53-64.

- RAYNAUD, J.P., BRUNAULT, G. and PATTERSON, E.B. (1981a). A swine dysentery model for evaluation of drug prophylaxis: development of a model involving oral infection plus pen contamination. *American Journal of Veterinary Research*. **42**:49-50.
- RAYNAUD, J.P., BRUNAULT, G. and PATTERSON, E.B. (1981b). A swine dysentery model for evaluation of drug prophylaxis: efficacy of various drugs in the control of swine dysentery. *American Journal of Veterinary Research*. **42**:51-53.
- REES, A.S., LYSONS, R.J., STOKES, C.R. and BOURNE, F.J. (1989a). Antibody production in the colon during infection with *Treponema hyodysenteriae*. *Research in Veterinary Science*. **23**:171-178.
- REES, A.S., LYSONS, R.J., STOKES, C.R. and BOURNE, F.J. (1989b). The effect of parental immunization on antibody production in the pig colon. *Immunology and Immunopathology*. **47**:263-269.
- ROBERTSON, I.D., MHOMA, J.R.L. and HAMPSON, D.J. (1992). Risk factors associated with the occurrence of swine dysentery in Western Australia: results of a postal survey. *Australian Veterinary Journal*. **69**:92-93.
- RONCALLI, R.A. and LEANING, W.H.D. (1976). Geographical distribution of swine dysentery. *Proceedings of the 2nd International Pig Veterinary Society Congress*, Ames, USA. p. 17.
- RUANE, P.J., NAKATA, M.M., REINHARDT, J.F. and GEORGE, W.L. (1989). Spirochete-like organisms in the human gastro-intestinal tract. *Review of Infectious Diseases*. **11**:184-196.
- RUTTER, J.M. (1985). The epidemiology of enteric disease. *The Pig Veterinary Society Proceedings*. **12**:56-64.
- SAGARTZ, J.E., SWAYNE, D.E., EATON, K.A., HAYES, J.R., AMASS, K.D., WACK, R. and KRAMER, L. (1992). Necrotizing typhlocolitis associated with a spirochaete in rheas (*Rhea americana*). *Avian Diseases*. **36**:776-781.
- SCHMALL, L.M., ARGENZIO, R.A. and WHIPP, S.C. (1983). Pathophysiological features of swine dysentery: cyclic nucleotide-independent production of diarrhoea. *American Journal of Veterinary Research*. **44**:1309-1316.
- SELLWOOD, R., THOMAS, W., CHESHAM, J., BURROWS, M.R. and WALTON, F. (1992). The development of an enzyme-linked immunosorbent assay (ELISA) for the diagnosis of swine dysentery. *Proceedings of the 12th International Pig Veterinary Society Congress*, The Hague, The Netherlands. p. 264.
- SELLWOOD, R., WALTON, F., THOMAS, W., BURROWS, M.R. and CHESHAM, J. (1995). Expression of the SmpA outer membrane lipoprotein of *Serpulina hyodysenteriae* strain P18A *in vivo*. *Veterinary Microbiology*. **44**:25-35.
- SIBA, P.M., PETHICK, D.W. and HAMPSON, D.J. (1994). Dietary control of swine dysentery. *Proceedings of the 13th International Pig Veterinary Society Congress*, Bangkok, Thailand. p. 149.
- SMITH, S.C., BARRETT, L.M., MUIR, T., CHRISTOPHER, W.L. and COLOE, P.J. (1991a). Application and evaluation of enzyme-linked immunosorbent assay and immunoblotting for detection of antibodies to *Treponema hyodysenteriae* in swine. *Epidemiology and Infection*. **107**:285-296.
- SMITH, S.C., MUIR, T., HOLMES, M. and COLOE, P.J. (1991b). *In-vitro* antimicrobial sensitivity of Australian isolates of *Treponema hyodysenteriae*. *Australian Veterinary Journal*. **68**:408-409.
- SMITH, S.C., RODDICK, F., LING, S., GERRATY, N.L. and COLOE, P.J. (1990). Biochemical and immunochemical characterisation of strains of *Treponema hyodysenteriae*. *Veterinary Microbiology*. **24**:29-41.
- SMITH, W.J. and NELSON, E.P. (1987). Grower scour/non-specific colitis. *The Veterinary Record*. **121**:334.
- SONGER, J.G., GLOCK, R.D., SCHWARZ, K.J. and HARRIS, D.L. (1978). Isolation of *Treponema hyodysenteriae* from sources other than swine. *Journal of the American Veterinary Medicine Association*. **172**:464-466.
- SONGER, J.G. and HARRIS, D.L. (1978). Transmission of swine dysentery by carrier pigs. *American Journal of Veterinary Research*. **39**:913-915.
- SOTIROPOULOS, C., SMITH, S.C. and COLOE, P.J. (1993). Characterization of two DNA probes specific for *Serpulina hyodysenteriae*. *Journal of Clinical Microbiology*. **31**:1746-1752.
- SPEARMAN, J.G., NAYAR, G. and SHERIDAN, M. (1988). Colitis associated with *Treponema innocens* in pigs. *Canadian Veterinary Journal*. **29**:747.
- STANTON, T.B. (1989). Glucose metabolism and NADH recycling by *Treponema hyodysenteriae*, the agent of swine dysentery. *Applied and Environmental Microbiology*. **55**:2365-2371.
- STANTON, T.B. (1992). Proposal to change the genus designation *Serpula* to *Serpulina* gen. nov. containing the species *Serpulina hyodysenteriae* comb. nov. and *Serpulina innocens* comb. nov. *International Journal of Systematic Bacteriology*. **42**:189-192.
- STANTON, T.B., JENSEN, N.S., CASEY, T.S., TORDOFF, L.A., DEWHIRST, F.E. and PASTER, B.J. (1991). Reclassification of *Treponema hyodysenteriae* and *Treponema innocens* in a new genus, *Serpula* gen. nov., as *Serpula hyodysenteriae* comb. nov. and *Serpula innocens* comb. nov. *International Journal of Systematic Bacteriology*. **41**:50-58.
- STANTON, T.B. and LEBO, D.F. (1988). *Treponema hyodysenteriae* growth under various culture conditions. *Veterinary Microbiology*. **18**:177-190.
- STANTON, T.B., TROTT, D.J., LEE, J.I., McLAREN, A.J., HAMPSON, D.J. and PASTER, B.J. (1995). Differentiation of intestinal spirochaetes by multilocus enzyme electrophoresis and 16S rRNA sequence comparisons. *FEMS Microbiology Letters*. *In press*.
- SUENAGA, I. and YAMAZAKI, T. (1984). Experimental *Treponema hyodysenteriae* infection in mice. *Zentralblatt für Bakteriologie Mikrobiologie und Hygiene, Series A*. **257**:348-356.
- SUEYOSHI, M. and ADACHI, Y. (1990). Diarrhea induced by *Treponema hyodysenteriae*: a young chick cecal model for swine dysentery. *Infection and Immunity*. **58**:3348-3362.
- TAKEUCHI, A., JERVIS, H.R., NAKAZAWA, H. and ROBINSON, D.M. (1974). Spiral-shaped organisms on the surface colonic epithelium of the monkey and man. *The American Journal of Clinical Nutrition*. **27**:1287-1296.
- TAYLOR, D.J. (1980). Spirochaetal diarrhoea. *Proceedings of the 4th International Pig Veterinary Society Congress*, Copenhagen, Denmark. p. 235.
- TAYLOR, D.J. (1992). Spirochaetal Diarrhoea. In "Diseases of Swine." 7th edn, pp. 599-616. eds, A.D. Leman, B.E. Straw, W.L. Mengeling, S. D'Allaire and D.J. Taylor. (Iowa State University Press: Ames, Iowa).
- TAYLOR, D.J. and ALEXANDER, T.J.L. (1971). The production of dysentery in swine by feeding cultures containing a spirochaete. *British Veterinary Journal*. **127**:58-61.
- TAYLOR, D.J., ESTRADA-CORREA, A. and PRÁDAL ROA, P. (1987). Grower scour and non-specific colitis. *The Veterinary Record*. **121**:479-480.

- TAYLOR, D.J., SIMMONS, J.R. and LAIRD, H.M. (1980). Production of diarrhoea and dysentery in pigs by feeding pure cultures of a spirochaete differing from *Treponema hyodysenteriae*. *The Veterinary Record*. **106**:326-332.
- TEIGE, J. (1984). Swine dysentery: The influence of dietary selenium on clinical and pathological effects of *Treponema hyodysenteriae* infection. *Acta Veterinaria Scandinavica*. **25**:1-9.
- ter HUURNE, A.H.M., van HOUTEN, M., KOOPMAN, M.B.H., van der ZEIJST, B.A.M. and GAASTRA, W. (1992). Characterization of Dutch porcine *Serpulina* (*Treponema*) isolates by restriction endonuclease analysis and DNA hybridization. *Journal of General Microbiology*. **138**:1929-1934.
- ter HUURNE, A.H.M., MUIR, S., van HOUTEN, M., KOOPMAN, M.B.H., KUSTERS, J.G., van der ZEIJST, B.A.M. and GAASTRA, W. (1993). The role of hemolysin(s) in the pathogenesis of *Serpulina hyodysenteriae*. *Zentralblatt für Bakteriologie*. **278**:316-325.
- THOMSON, J.G. and THOMSON, D. (1914). Some researches on spirochaetes occurring in the alimentary tract of man and some of the lower animals. *Proceedings of the Royal Society of Medicine*. **7**:47-70.
- TOMPKINS, D.S., FOULKES, S.J., GODWIN, P.G.R. and WEST, A.P. (1986). Isolation and characterisation of intestinal spirochaetes. *Journal of Clinical Pathology*. **39**:535-541.
- TORP, M. and THORENSEN, O.F. (1992). Phenotypic and genotypic characterisation of Norwegian field strains of *Serpulina* spp. *Proceedings of the 12th International Pig Veterinary Society Congress*, The Hague, The Netherlands. p. 270.
- TRAMPEL, D.W., JENSEN, N.S. and HOFFMAN, L.J. (1994). Cecal spirochetosis in commercial laying hens. *Avian Diseases*. **38**:895-898.
- TROTT, D.J., McLAREN, A.J. and HAMPSON, D.J. (1995). Pathogenicity of human and porcine intestinal spirochaetes in day-old specific pathogen free chicks: an animal model of intestinal spirochetosis. *Infection and Immunity*. **63**: In press.
- TURNER, A.K., ATYEO, R.F., SELLWOOD, R. and HAMPSON, D.J. (1995). Distribution and heterogeneity of the *smgA* gene from *Serpulina hyodysenteriae* among intestinal spirochaetes. *Microbiology*. In press.
- VAREL, V.H. (1987). Activity of fiber-degrading microorganisms in the pig large intestine. *Journal of Animal Science*. **65**:488-496.
- WANNEMUEHLER, M.J., OSTLE, A.G., NIBBELINK, S.K., COYLE, D.C. and WELTER, C.J. (1990). Pathogenesis of swine dysentery: preparation of a protective vaccine. *Proceedings of the 11th International Pig Veterinary Society Congress*, Lausanne, Switzerland. p. 124.
- WHIPP, S.C., POHLENZ, J., HARRIS, D.L., ROBINSON, I.M., GLOCK, R.D. and KUNKLE, R. (1982). Pathogenicity of *Treponema hyodysenteriae* in uncontaminated gnotobiotic pigs. *Proceedings of the 6th International Pig Veterinary Society Congress*, Mexico City, Mexico. p. 31.
- WHIPP, S.C., ROBINSON, I.M., HARRIS, D.L., GLOCK, R.D., MATTHEWS, P.J. and ALEXANDER, T.J.L. (1979). Pathogenic synergism between *Treponema hyodysenteriae* and other selected anaerobes in gnotobiotic pigs. *Infection and Immunity*. **26**:1042-1047.
- WHITING, R.A., DOYLE, L.P. and SPRAY, R.S. (1921). Swine Dysentery. *Purdue University Agriculture Experiment Station Bulletin*. **257**:3-15.
- WILKINSON, J.D. and WOOD, E.N. (1987). Grower scour/non-specific colitis. *The Veterinary Record*. **121**:406.
- WINDSOR, R.S. and SIMMONS, J.R. (1981). Investigation into the spread of swine dysentery in 25 herds in East Anglia and assessment of its economic significance in five herds. *The Veterinary Record*. **109**:482-484.
- WOOD, E.N. and LYSONS, R.J. (1988). The financial benefit from the eradication of swine dysentery. *The Veterinary Record*. **121**:277-279.
- WRIGHT, J.C., WILT, G.R., REED, R.B. and POWE, T.A. (1989). Use of an enzyme-linked immunosorbent assay for detection of *Treponema hyodysenteriae* infection in swine. *Journal of Clinical Microbiology*. **27**:411-416.
- ZHANG, P., DUHAMEL, G.E., MYSORE, J.V., CARLSON, M.P. and SCHNEIDER, N.R. (1995). Prophylactic effect of dietary zinc in a laboratory mouse model of swine dysentery. *American Journal of Veterinary Research*. **56**:334-339.



## FERMENTATION IN THE LARGE GUT AND SWINE DYSENTERY

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Previous research (Siba *et al.*, 1993a, 1993b) suggests that swine dysentery may be prevented in growing pigs that are infected with *Serpulina hyodysenteriae* by feeding a diet based on cooked white rice and animal protein. This highly-digestible diet resulted in low rates of microbial fermentation in the large intestine, and it is suggested that the nature of the diet may play a role in the control of swine dysentery (SD). However, rice is uneconomical to feed to pigs commercially. In this experiment, the efficacy of a variety of grains to control SD was tested. Grains were fed either "raw" or after steam-flaking for 20 min at 150°C, "cooked". Steam-flaking is likely to increase digestibility in the small intestine causing a lower volume of nutrients to be available in the large intestine for microbial fermentation. This environment may be less favourable for the colonisation and proliferation of *S. hyodysenteriae*.

Pigs weighing  $8.7 \pm 0.09$  kg (mean  $\pm$  SEM) were weaned at 28 days of age, allocated to groups of 9-10/pen, and fed diets containing either wheat, barley, groats, maize and sorghum in either "raw" or "cooked" form. Another group was fed a commercial diet. Each diet contained the grain supplemented with sources of animal protein, minerals, and vitamins, and contained approximately 14.7 MJ DE/kg, 18.5% CP and 1.1% available lysine. No antibiotic was added to the diets. Pigs were fed *ad libitum* for an average of seven weeks, at which time half were killed at a commercial abattoir. The remaining pigs were challenged with a virulent culture of *S. hyodysenteriae* and clinical signs of the disease noted. Diarrhoeic pigs were killed and a post-mortem examination conducted.

Table 1. Incidence of disease, and gut characteristics of slaughtered pigs.

Grain	Disease incidence <sup>1</sup>		pH in proximal colon <sup>2</sup>		pH in distal colon <sup>2</sup>		Weight of large intestine (% BW) <sup>2</sup>	
	Raw	Cooked	Raw	Cooked	Raw	Cooked	Raw	Cooked
Wheat	100	100	5.5	5.7*	6.2	6.4*	3.2	3.0*
Barley	100	100	5.3	5.7*	5.9	6.1*	4.2	3.5*
Groats	100	100	5.6	5.7	6.0	6.2	3.1	3.1
Maize	75	0	5.5	5.6	5.9	6.4*	3.0	2.8*
Sorghum	50	0	5.5	5.5	6.1	5.9	3.4	3.7*
Commercial	100		5.5		6.1		4.5	

<sup>1</sup>Percentage of pigs infected showing clinical signs of disease. <sup>2</sup>From pigs killed prior to infection with *S. hyodysenteriae*. \*Indicates significance at  $P < 0.05$ .

Results from an overall ANOVA indicate that steam-flaking increased the pH of the proximal and distal colon ( $P < 0.001$ ), and decreased the weight of the large intestine when expressed relative to body-weight ( $P < 0.05$ ). All pigs fed "raw" and "cooked" wheat, barley and groats succumbed to SD. A reduced incidence of disease was noted in pigs fed maize and sorghum, and no disease was observed in pigs fed these grains in a 'cooked' form. As maize and sorghum have the lowest levels of dietary fibre, and cooking reduces hind-gut fermentation, the results support the hypothesis that a lower level of fermentation in the hind-gut prevents SD. However, the diet containing 'cooked' sorghum caused an increased rate of fermentation (based on pH and gut size) compared to sorghum fed 'raw'. This suggests alternative modes of action for sorghum, and is being studied further.

### References

- SIBA, P.M., PETHICK, D.W. and HAMPSON, D.J. (1993a). *Proceedings of the XVth International Congress of Nutrition*, Adelaide, Australia, p.878.
- SIBA, P.M., PETHICK, D.W., NAIRN, K.A. and HAMPSON, D.J. (1993b). In "Manipulating Pig Production IV", p.254, ed. E.S. Batterham. (Australasian Pig Science Association: Attwood).

## APPLICATION OF A POLYMERASE CHAIN REACTION ASSAY TO DIAGNOSE PROLIFERATIVE ENTERITIS IN PIG HERDS

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Porcine proliferative enteritis (PE) is an intestinal disease of growing pigs, characterised by proliferation of enterocytes, and the presence of ileal symbiont intracellularis (ISI) bacteria within infected epithelial cells (Barker and Van Dreumel, 1985; Gebhart *et al.*, 1993). The disease occurs subclinically, or pigs may show poor growth, diarrhoea, dysentery or sudden death (Love *et al.*, 1977; Gogolewski *et al.*, 1991). Assessment of the prevalence and economic impact of PE is difficult, as the disease cannot be reliably diagnosed in live pigs. A polymerase chain reaction (PCR) assay was developed to assist PE diagnosis, based on amplification of a 319-bp DNA fragment of ISI in faeces (Jones *et al.*, 1993). The PCR has the sensitivity to detect  $10^3$  ISI per g of faeces, and its specificity has been verified by Southern transfer, hybridisation with digoxigenin-labelled p78 and detection by chemiluminescence (Jones *et al.*, 1993).

Faeces were collected from PE-affected herds in the midwest of the United States (Farms A, B and C), and central Victoria (Farm D) to: (1) confirm the presence of ISI, (2) determine the age range of ISI shedding, (3) assess the ability to detect PE in herds with pooled faecal samples, and (4) determine the effect of medication on ISI shedding. Two faecal samples, each comprising five individual stools, were collected from at least two pen floors housing weaners, growers, finishers and breeding stock from each farm, to screen for ISI and assess the age range of shedding. Faeces were collected from individual pigs on Farms A and B to assess the association between pooled samples from pens and ISI shedding by pigs. Samples of DNA were extracted from faeces, and PCR conducted (Boom *et al.*, 1990; Jones *et al.*, 1993).

The presence of ISI was detected in the faeces of apparently healthy 10 to 25-week-old pigs only, indicating that these pigs were the main source of infection for younger pigs. Shedding was detected in 4% to 32% of pigs within "infected" (at least one positive sample) pens on Farm A, and 0% to 29% of pigs within "infected" pens on Farm B. These pigs shed ISI, despite the inclusion of 40 to 100 g/tonne of tylosin (Farms A, B and C), 10 to 200 g/tonne of chlortetracycline (Farms B and D), and 25 g/tonne of lincomycin (Farm D) to the pigs' feed. No ISI bacteria were detected in the faeces of pigs treated with a combination of 4 g/l of soluble neomycin sulphate and 100 g/tonne feed-grade tylosin (Farm A), or oxytetracycline at 100 g/tonne and 200 g/tonne of feed (Farm D). The ISI bacteria were detected in a pooled sample collected from the floor of one pen on Farm B but not from individual pigs within this pen. One pig housed in a "negative" pen on Farm B also shed ISI. These irregularities can be explained by the frequent movement of pigs among pens on this farm and the failure to clean and disinfect between batches of pigs.

These results suggest that PCR testing of pooled faecal samples can identify groups of pigs infected with ISI and can thus be used as a tool for epidemiological studies of PE.

### References

- BARKER, I.K. and van DREUMEL, A.A. (1985). In "Pathology of Domestic Animals", 3rd edn Vol 2. pp. 143-146, eds, K.V.F. Jubb, P.C. Kennedy and N. Palmer. (Academic Press: USA).
- BOOM, R., SOL, C.J.A., SALIMANS, M.M., JANSEN, C.L., WERTHEIM-VAN DILLEN, P.M.E. and van DER NOORDAA, J. (1990). *Journal of Clinical Microbiology*. 28:495-503.
- GEBHART, C.J., BARNS, S.M., McORIST, S., LIN, G.F. and LAWSON, G.H.K. (1993). *International Journal of Systematic Bacteriology*. 43:533-538.
- GOGOLEWSKI, R.P., COOK, R.W. and BATTERHAM, E.S. (1991). *Australian Veterinary Journal*. 12:406-408.
- JONES, G.F., WARD, G.E., MURTAUGH, M.P., LIN, G. and GEBHART, C.J. (1993). *Journal of Clinical Microbiology*. 31:2611-2615.
- LOVE, R.J., LOVE, D.N. and EDWARDS, M.J. (1977). *Veterinary Record*. 100:65-68.

## BACTERIAL COLONIZATION OF THE PIGLET GUT

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Investigation into the establishment of the intestinal flora of the newborn piglet and the role of fermentation during the suckling period, has been hindered by the low sensitivity and the invasiveness of the available methods. Measurement of the products of bacterial fermentation in blood is often confounded by the production of these metabolites during the normal metabolism of the animal. However, D-lactate is not normally produced in vertebrate metabolism (Drury and Wick, 1965), and the D-lactate, formed during fermentation by certain species of *Lactobacillus* is absorbed from the gut into the peripheral circulation and either slowly metabolized or excreted in the urine.

Blood samples (~60  $\mu$ L) were taken from eleven piglets (Landrace x Large White), at frequent intervals over the first 12 h after birth, and then at 18, 24, 48, 72, 96 and 120 h. A bioluminescent assay was developed to measure the concentration of D-lactate in the plasma. Piglets weighed  $1.4 \pm 0.2$  kg at birth and  $2.2 \pm 0.3$  kg at 120 h. Piglets were not cross-fostered and received no antibiotics during the experiment.

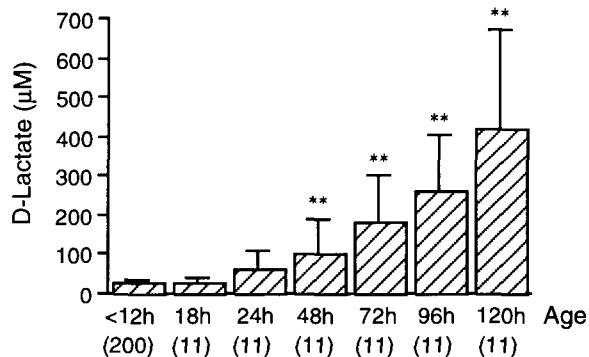


Figure 1. Changes in the concentration of D-lactate (mean  $\pm$  SD) in the peripheral blood of piglets from 0 to 120 h after birth. \*\*Concentrations significantly greater than at <12 h ( $P < 0.01$ ). The number of samples is given in parenthesis.

There was little change in the low concentrations of D-lactate during the first 18 hours with relatively low variation among piglets ( $CV=38\%$ ). Variability increased markedly by 24 h ( $CV=76\%$ ), with D-lactate increasing earlier in some piglets than in others. By 48 h the mean concentration was significantly higher than that seen during the first 12 h (Figure 1). Furthermore, D-lactate continued to increase from 48 to 120 h.

The results indicate that colonization of the intestinal tract of the piglet occurs within the first 48 h after birth and are consistent with the microbial examination of the gut contents from sacrificed piglets (Wilbur *et al.*, 1960; Pederson and Tannock, 1989). Thus, fermentation was occurring in the healthy, sucking piglet at a time when the lactase activity is maximal and the diet was highly digestible. Since it can be assumed that the progressive increase in the concentration of D-lactate was derived from fermentation in the gastro-intestinal tract, the measurement of blood D-lactate may provide a non-invasive, qualitative method of investigating variation in colonization of the intestinal tract of the newborn piglet.

### References

- DRURY, D.R. and WICK, A.N. (1965). *Annals of the New York Academy of Science*. **119**:1061-1069.  
 PEDERSON, K. and TANNOCK, G.W. (1989). *Applied Environmental Microbiology*. **55**:279-283.  
 WILBUR, R.D., CATRON, D.V., QUINN, L.Y., SPEER, V.C. and HAYS, V.W. (1960). *Journal of Nutrition*. **71**:168-175.

## THE POTATO FIBRE PREPARATION POVEX ENHANCES PANCREATIC SECRETION IN THE PIG

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In animals and man dietary fibre can affect the function of the exocrine pancreas and the gastro-intestinal (GI) tract (Dukehart *et al.*, 1989; Zebrowska and Low, 1987). When the potato fiber preparation POVEX (containing 70 % dietary fibre, Lyckeby Stärkelsen AB, Sweden) is added to an ordinary pig diet (at 1-2 %) it has been found to increase body-weight gain, improve feed utilisation efficiency and improve health. To gain some understanding of the mode of action of POVEX in pigs a study was conducted to examine its short and long term effects on exocrine pancreatic and GI-tract function.

Three Swedish Landrace pigs were weaned at 43 days of age and received a standard weaner diet (Växfor, Lantmännen, Sweden) containing 15.5 % protein and 12.2 MJ ME/kg *ad libitum*. The pigs were surgically prepared with pancreatic duct catheters, duodenal re-entrant T cannulas and jugular vein catheters at 56 days of age when they were 12.4 ± 0.2 kg body-weight (Pierzynowski, 1991). During three 2-week consecutive periods the pigs were fed: I. the standard weaner diet; II. the standard weaner diet supplemented with 2 % POVEX; III. the standard weaner diet. At the end of the experimental period the pigs were 26.1 ± 5.3 kg body-weight. Following an overnight fast, measurements of pancreatic juice secretion were made for 2.5 h every fourth day during the 6 week experimental period. Pancreatic secretion was collected for a 1 h basal period followed by a 1.5 h period of stimulation which commenced with a 30 min intra-duodenal infusion. The infusate consisted of an homogenate of the food for the period in saline (20% dry matter), and was delivered at the rate of 5 ml/kg/h. Protein and digestive enzymes in pancreatic juice were measured utilizing methods described by Pierzynowski (1991).

Table 1. Volume, and amounts (per unit body-weight<sup>0.75</sup>) of soluble protein and digestive enzymes secreted in pancreatic juice during basal and stimulated periods of secretion (mean ± SEM).

Period: (per kg <sup>0.75</sup> /h)	I		II		III	
	Basal	Stimulated	Basal	Stimulated	Basal	Stimulated
Volume (ml)	2.1 ± 0.3 <sup>a</sup>	2.1 ± 0.6 <sup>ab</sup>	5.2 ± 1.2 <sup>bc</sup>	4.9 ± 0.6 <sup>bc</sup>	6.3 ± 1.2 <sup>c</sup>	5.7 ± 0.5 <sup>bc</sup>
Protein (mg)	5.1 ± 1.4 <sup>a</sup>	6.1 ± 1.8 <sup>a</sup>	7.9 ± 2.3 <sup>ab</sup>	12.3 ± 1.5 <sup>b</sup>	14.9 ± 3.2 <sup>bc</sup>	18.3 ± 2.3 <sup>c</sup>
Trypsin (U)	4.1 ± 1.0 <sup>a</sup>	5.2 ± 1.2 <sup>a</sup>	5.9 ± 1.7 <sup>ab</sup>	9.1 ± 1.2 <sup>b</sup>	10.5 ± 2.9 <sup>bc</sup>	13.0 ± 1.8 <sup>c</sup>
Lipase (kU)	0.7 ± 0.2 <sup>a</sup>	0.5 ± 0.2 <sup>a</sup>	2.5 ± 0.6 <sup>b</sup>	1.7 ± 0.4 <sup>b</sup>	3.2 ± 0.7 <sup>b</sup>	3.1 ± 0.2 <sup>b</sup>
Amylase (MU)	0.4 ± 0.1 <sup>a</sup>	0.3 ± 0.1 <sup>a</sup>	0.7 ± 0.3 <sup>ab</sup>	1.0 ± 0.3 <sup>b</sup>	0.2 ± 0.1 <sup>a</sup>	0.2 ± 0.1 <sup>a</sup>

<sup>abc</sup>Means in the same row with different superscripts are significantly different (P < 0.05).

The general trend was that the basal and stimulated outputs of all the measured components of pancreatic secretion increased after the introduction of POVEX and, with the exception of amylase, they remained at these high levels after POVEX was removed from the diet.

It is suggested that by increasing pancreatic exocrine function POVEX enhances the digestive processes in the small intestine. However, the mechanisms whereby POVEX brings about these changes and what other effects it may have on GI processes remain to be elucidated.

### References

- DUKEHART, M.R., DUTTA, S.K. and VEATH, J. (1989). *American Journal of Clinical Nutrition*. 50:1023-1028.  
 PIERZYNOWSKI, S.G. (1991). Development and regulation of porcine pancreatic function with special reference to the exocrine pancreas. PhD Thesis. University of Lund.  
 ZEBROWSKA, T., and LOW, A.G. (1987). *Journal of Nutrition*. 117:1212-1216.

## WEIGHT AT WEANING , CAUSES AND CONSEQUENCES

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One of the major limitations to satisfactory post-weaning growth performance is low live-weight at weaning. Campbell (1989), reported that a difference of 1.8 kg (6.1 vs 7.9 kg) at weaning at 25-29 d increased to 5 kg at 78 d and to 10 kg at 150 d. Also, Mahan and Lepine (1991) found that pigs with weaning weights of 4.1 to 5.0 kg required 11 to 20 d longer to reach a slaughter weight of 105 kg than pigs with weaning weights of 7.3 to 8.6 kg. In this experiment the variation in weaning weight, both within and among litters, and the pattern of growth rate from birth to 154 days of age were examined.

The progeny (111 pigs) of 13 Large White  $\times$  Landrace fifth parity sows were used in the experiment. Following weaning at 27 days of age all pigs were fed the same starter (4 to 7 weeks), weaner (7 to 10 weeks), grower (10 to 17 weeks) and finisher (17 to 23 weeks) diets. All pigs were weighed individually at recorded intervals (Figure 1).

Live-weight of pigs at weaning was characterised by large variations both within and among litters (range 4.4 to 11.0 kg). In litters of 9 pigs total litter-weight at weaning ranged from 58 to 83 kg. Live-weights (mean  $\pm$  SD) of pigs within litters ranged from 6.4  $\pm$  0.8 kg to 9.2  $\pm$  1.1 kg; some litters had a much higher variation, eg., 7.0  $\pm$  2.4 kg. Of the 111 pigs, 14% were  $\leq$  6.0 kg and 17% were  $\geq$  9.0 kg live-weight at weaning. There were significant correlations between birthweight and weight at weaning, and between birth weight and weight at 154 d of age but the coefficients of determination were very small ( $r^2=0.10$ ,  $r^2=0.12$  respectively).

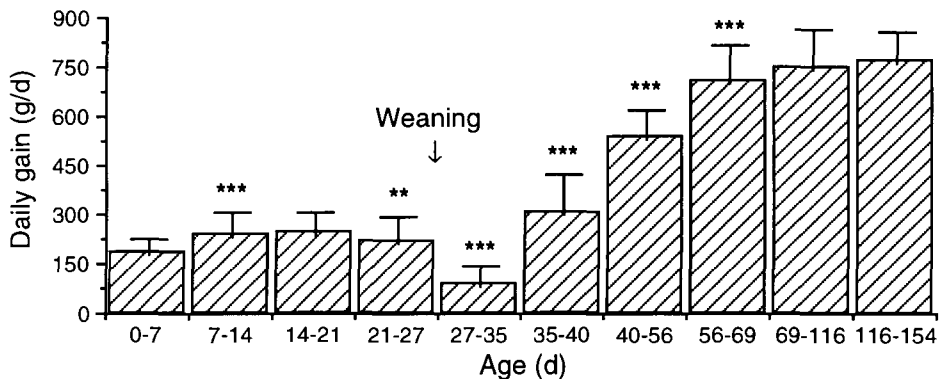


Figure 1. Growth rate of pigs from birth to 154 days of age (mean  $\pm$  SD). Daily gain was significantly different from that in the previous age group (\*\* $P < 0.01$ ; \*\*\* $P < 0.001$ ).

The amount of sows' milk which pigs receive during lactation is mirrored by their daily gain from 0 to 27 d of age (Figure 1). Maximum growth rate (248  $\pm$  63 g/d) occurred at 14 to 21 d and would correspond to the peak in sows' milk production. In the following week growth rate was significantly lower (216  $\pm$  87 g/d). It is concluded that sows milk production can be a major constraint to weight at weaning. Also, the large variation among litters in live-weight at weaning is probably due to a corresponding variation in total milk production among sows. The large number of small-for-age pigs, which occur in some litters, is possibly the result of a variable supply of milk from the individual mammary glands in the sows. Alternatively, it could be the result of variation in the ability of the piglets to suck from, or stimulate milk production by, the sow.

### References

- CAMPBELL, R.G. (1989). In "Manipulating Pig Production II", pp. 170-175, eds J.L. Barnett and D.P. Hennessy. (Australasian Pig Science Association: Werrisbee).
- MAHAN, D.C. and LEPINE, A.J. (1991). *Journal of Animal Science*. 69:1370-1378.

## GUT DEVELOPMENT FROM 4 TO 23 WEEKS OF AGE

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A major limitation to satisfactory post-weaning growth performance is low body-weight at weaning (Campbell, 1989). In this study a comparison was made between the gastro-intestinal tracts in small-for-age and large-for-age pigs, at three ages, to determine if gut development could be a contributing factor to pre- and post-weaning growth rates.

Observations were made in the lightest (L) and heaviest (H) pigs in each litter from 13 litters of Large White  $\times$  Landrace pigs at weaning (4 weeks; L pigs  $5.62 \pm 0.24$  kg body-weight and H pigs  $9.15 \pm 0.31$  kg; mean  $\pm$  SEM) and 2 weeks after weaning (L pigs  $8.34 \pm 0.26$  kg and H pigs  $11.16 \pm 0.33$  kg), and from 9 of these litters at slaughter (23 weeks; L pigs  $82.20 \pm 1.73$  kg and H pigs  $98.27 \pm 2.54$  kg). Empty stomach weight, pancreas weight, and weight of the empty small intestine were recorded.

At 4 weeks of age the total weight of the stomach, pancreas and small intestine (gut) was greater in the H pigs than the L pigs ( $P < 0.001$ ). However, relative gut weight, expressed as % body-weight, was greater for L pigs than H pigs ( $4.1 \pm 0.2\%$  vs  $3.4 \pm 0.1\%$ ). The total amount of gut tissue in 6 week-old pigs ( $573 \pm 19$  g) was 2.2-fold greater than that in 4 week-old pigs ( $261 \pm 11$  g), an increase that represented 14% of the body-weight gain during the 2 week post-weaning period. Relative gut weight in 6 week-old pigs ( $6.3 \pm 0.2\%$ ) was greater than in 4 week-old pigs ( $P < 0.001$ ), but the difference between H and L pigs was not significant, except for the stomach (Figure 1). By 23 weeks relative gut weight ( $2.6 \pm 0.1\%$ ) was significantly lower than at 4 or 6 weeks of age ( $P < 0.001$ ) and was greater in L pigs than H pigs ( $P < 0.05$ ), with the major difference being in the small intestine (Figure 1).

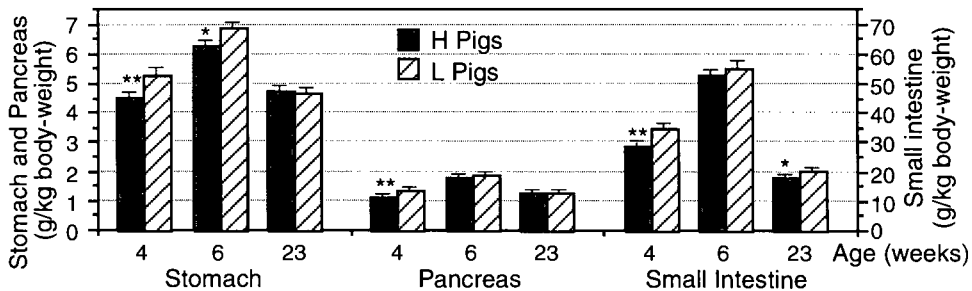


Figure 1. Relative weight of the stomach, pancreas and small intestine per unit body-weight (g/kg). Differences between H and L pigs at the same age were significant, \*  $P < 0.05$ ; \*\*  $P < 0.01$ .

The results show that during lactation gut development in the L pigs was in proportion to body size and was unlikely to be a major factor contributing to their poor pre-weaning growth. The most probable reason for the slow growth rate of L pigs during lactation was a lower nutrient intake than that of H pigs. This suggestion is consistent with the findings of Ebner *et al.* (1994) that undernourished pigs will preferentially maintain gut size and function at the expense of other sites of protein deposition, eg., skeletal muscle. The results also indicate that the gut of the pig undergoes a period of strongly positive allometric growth following weaning which either precedes or coincides with the increase in growth rate in the second week after weaning (Cranwell *et al.*, 1995).

## References

- CAMPBELL, R.G. (1989). In "Manipulating Pig Production II", pp. 170-175, eds J.L. Barnett and D.P. Hennessy. (Australasian Pig Science Association: Werribee).
- CRANWELL, P.D., TARVID, I., MA, L., HARRISON, D.T. and CAMPBELL, R.G. (1995). In "Manipulating Pig Production V", p. 174, eds D.P. Hennessy and P.D. Cranwell. (Australasian Pig Science Association: Werribee).
- EBNER, S., SCHOKNECHT, P., REEDS, P. and BURRIN, D. (1994). *American Journal of Physiology*. 266:R1736-R1743.

## GASTRIC PROTEASES IN LIGHT AND HEAVY PIGS AT 4, 6 AND 23 WEEKS OF AGE

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The transition from mainly milk-clotting activity (MCA) to general proteolytic activity (GPA) in porcine gastric proteases takes place during the first 6 weeks of life (Sangild *et al.*, 1991). As part of a study to identify factors which may influence pre- and post-weaning growth rate, the development of MCA and GPA in small-for-age pigs was compared with that in large-for-age pigs.

Stomachs from the light (L) and heavy (H) pigs described by Cranwell *et al.* (1995) were dissected. Tissue preparation and the methods used to measure MCA and GPA in the cardiac, fundic and pyloric mucosa have been described by Sangild *et al.* (1991).

Stomach weight per unit body-weight data were provided by Cranwell *et al.* (1995). Total gastric mucosa comprised  $53.6 \pm 0.5\%$  (mean  $\pm$  SEM) of stomach weight in 4 and 23 week-old pigs compared to  $57.3 \pm 0.4\%$  in 6 week-old pigs ( $P < 0.001$ ). In 4 week-old pigs fundic mucosa comprised  $65.7 \pm 1.0\%$  of total gastric mucosa which was in turn greater than that in 6 week-old ( $59.1 \pm 0.8\%$ ;  $P < 0.001$ ) and 23 week-old pigs ( $54.4 \pm 0.6\%$ ;  $P < 0.001$ ). The amount of fundic tissue per unit body-weight in L pigs, at 4 and 6 weeks, was 14% greater than that in H pigs ( $P < 0.05$ ). In 4 and 6 week-old pigs 90-94% of total MCA and 92-97% of total GPA were located in the fundic mucosa. The remainder of these activities was located in the pyloric mucosa (3-10%) with  $< 1\%$  in the cardiac mucosa. A similar pattern of distribution of GPA was found in 23 week-old pigs. Total amounts of MCA and GPA per unit body-weight were greater in L pigs than H pigs at 4 weeks (Figure 1). By 6 weeks there was a three-fold increase in total GPA per unit body-weight in both L and H pigs to levels which were not significantly different from those at 23 weeks.

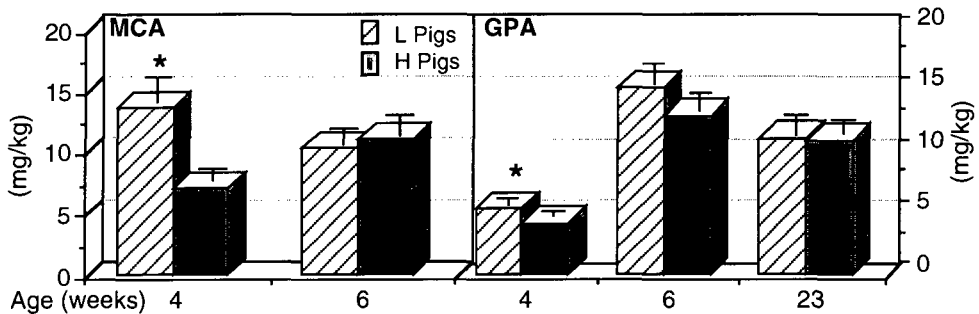


Figure 1. Total MCA and GPA in the gastric mucosa per unit body-weight (mg/kg). Differences between L pigs and H pigs at the same age were significant, \*  $P < 0.05$ .

The results, which show that the relative development of gastric proteases in L pigs in the pre- and post-weaning periods is equal to or greater than that in H pigs, are consistent with those for the development of relative stomach weight (Cranwell *et al.*, 1995). Thus, gastric development in L pigs appears to be adequate and is unlikely to be the major factor limiting their growth rate. The development of the mainly proteolytic gastric proteases, i.e., pepsin A and gastricsin (Sangild *et al.*, 1991), which are the main contributors to GPA, appears to be complete in 6 week-old pigs.

### References

- CRANWELL, P.D., TARVID, I., MA, L., HARRISON, D.T. and CAMPBELL, R.G. (1995). In "Manipulating Pig Production V", p. 175, eds D.P. Hennessy and P.D. Cranwell. (Australasian Pig Science Association: Werribee).
- SANGILD, P.T., FOLTMANN, B. and CRANWELL, P.D. (1991). *Journal of Developmental Physiology*. 16:229-238.

## PANCREATIC PROTEASES IN LIGHT AND HEAVY PIGS AT 4, 6 AND 23 WEEKS OF AGE

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The pancreatic proteolytic and peptidase enzymes play an important role in protein digestion and absorption in the pig. To ascertain if gut development and maturation are important determinants of pre- and post-weaning growth performance a comparison was made of the development of pancreatic proteases in small- and large-for-age pigs.

Pancreatic glands were obtained from the lightest (L) and heaviest (H) pigs in each litter from 13 litters of Large White × Landrace pigs at weaning (4 weeks) and 2 weeks after weaning, and from 9 of these litters at slaughter (23 weeks) (Cranwell *et al.*, 1995). General proteolytic activity (GPA), chymotrypsin, trypsin and carboxypeptidase A (CPA) were measured according to methods described by Tarvid *et al.* (1994) in homogenates of pancreatic tissue activated with enterokinase.

**Table 1. Total carboxypeptidase A (CPA), general proteolytic activity (GPA), chymotrypsin and trypsin in pancreatic tissue per unit body-weight in light (L) and heavy (H) pigs (mean ± SEM).**

	Pigs	Age		
		4 weeks	6 weeks	23 weeks
CPA (µmol leucine/min per kg)	Light	16.4 ± 1.1 <sup>a</sup>	21.3 ± 1.5 <sup>b</sup>	15.5 ± 1.0 <sup>a</sup>
	Heavy	13.8 ± 0.4 <sup>a</sup>	24.8 ± 2.2 <sup>b</sup>	16.2 ± 1.0 <sup>c</sup>
GPA (µmol tyrosine/min per kg)	Light	24.5 ± 2.2 <sup>a</sup>	8.6 ± 0.8 <sup>b</sup>	8.3 ± 0.6 <sup>b</sup>
	Heavy	16.1 ± 1.5 <sup>a</sup>	8.9 ± 1.4 <sup>b</sup>	9.0 ± 0.9 <sup>b</sup>
Chymotrypsin (mg/kg)	Light	10.1 ± 1.3 <sup>a</sup>	6.2 ± 0.7 <sup>b</sup>	15.7 ± 1.8 <sup>c</sup>
	Heavy	8.1 ± 0.7 <sup>a</sup>	7.4 ± 0.9 <sup>a</sup>	17.1 ± 1.3 <sup>b</sup>
Trypsin (mg/kg)	Light	1.4 ± 0.1 <sup>a</sup>	4.7 ± 0.5 <sup>b</sup>	2.4 ± 0.3 <sup>c</sup>
	Heavy	1.2 ± 0.1 <sup>a</sup>	5.3 ± 0.6 <sup>b</sup>	2.0 ± 0.2 <sup>c</sup>

\*Differences between L pigs and H pigs at the same age are significant ( $P < 0.05$ ). <sup>abc</sup>Means in the same row with different superscripts are significantly different ( $P < 0.05$ ).

Each enzyme underwent its own characteristic development from weaning until slaughter. In Table 1 total amounts of enzymes are expressed in relation to body-weight. Trypsin and CPA showed similar patterns of development which were opposite to that for chymotrypsin. The pattern for GPA reflected the combined changes in the other enzymes. At weaning, the relative amounts of each enzyme were greater in L pigs than H pigs; the differences for CPA and GPA were significant. At 6 and 23 weeks the amounts of all enzymes, except trypsin at 23 weeks, were greater in H than in L pigs, but the differences were not significant. The results indicate that functional development of pancreatic enzymes in relation to body size was similar for L and H pigs. It is concluded that factors other than the development of the pancreas (Cranwell *et al.*, 1995) and its enzyme systems are responsible for the poor pre- and post-weaning growth rate of small-for-age pigs.

### References

- CRANWELL, P.D., TARVID, I., MA, L., HARRISON, D.T. and CAMPBELL, R.G. (1995). In "Manipulating Pig Production V", p. 175, eds D.P. Hennessy and P.D. Cranwell. (Australasian Pig Science Association: Werrisbee).
- TARVID, I., CRANWELL, P.D., MA, L. and VAVALA, R. (1994). In "Proceedings of the VIth International Symposium on Digestion in Pigs", pp. 199-202, eds W-B. Souffrant and H. Hagemester (EAAP-publication No. 80: Dummerstorf).



## SMALL INTESTINAL PEPTIDASES IN LIGHT AND HEAVY PIGS AT 4 AND 6 WEEKS OF AGE

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The peptidase enzymes produced by the enterocytes in the mucosa of the small intestine play an important role in the final stages of protein digestion and in the absorption of peptides and amino acids in the pig. As part of a study of the development of the digestive capacity of the gut, and to determine its influence on pre- and post-weaning growth rate, comparisons were made of the small intestinal peptidases in small-for-age and large-for-age pigs at weaning (4 weeks) and 2 weeks after weaning.

Small intestines (SI) from the 4 and 6 week-old, light (L) and heavy (H) pigs described by Cranwell *et al.* (1995) were dissected, emptied, weighed and divided into 5 equal lengths. Tissue preparation and the methods used to measure total soluble protein, and aminopeptidase (AP) and dipeptidase (DP) activities in homogenates of the small intestinal mucosa have been described by Tarvid *et al.* (1994).

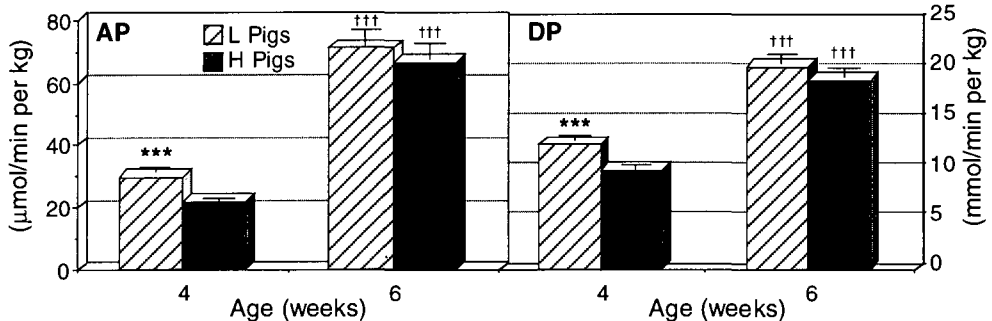


Figure 1. Total AP and DP in the small intestine per unit body-weight (mean  $\pm$  SEM). Differences between light (L) pigs and heavy (H) pigs at the same age were significant, \*\*\* $P < 0.001$ ; and differences between 4 and 6 week-old pigs (L vs L; H vs H) were significant, ††† $P < 0.001$ .

The findings that the total amounts of AP and DP in the SI per unit body-weight at weaning (4 weeks) were significantly greater in the L pigs than the H pigs (Figure 1) are consistent with the trend for small intestinal weight reported by Cranwell *et al.* (1995). Development of the SI during the 2 weeks after weaning was characterised by 2.7- and 1.7-fold increases in AP and DP activities per unit body-weight respectively (Figure 1), and by a corresponding 1.7-fold increase in small intestinal weight (Cranwell *et al.*, 1995).

Although the H pigs were 64% and 32% heavier than the L pigs at weaning and 2 weeks after weaning respectively (Cranwell *et al.*, 1995) the H pigs had only 19% and 22% more total AP and 29% and 23% more total DP than the L pigs at these times respectively. Comparison of the absolute and relative (Figure 1) amounts of these two SI enzymes in L and H pigs would suggest that the digestive capacity of the small intestine in L pigs was unlikely to have been the major factor which limited their pre- and post-weaning growth. However, despite the similar 3.5- and 2.4-fold increases in total AP and DP which occurred in L and H pigs in the two weeks after weaning, pigs which are small-for-age at weaning do not undergo compensatory growth during the period up to slaughter weight which would enable them to perform as well as their heavier counterparts.

### References

- CRANWELL, P.D., TARVID, I., MA, L., HARRISON, D.T. and CAMPBELL, R.G. (1995). In "Manipulating Pig Production V", p. 175, eds D.P. Hennessy and P.D. Cranwell. (Australasian Pig Science Association: Werribee).
- TARVID, I., CRANWELL, P.D., MA, L. and VAVALA, R. (1994). In "Proceedings of the VIth International Symposium on Digestion in Pigs", pp. 181-184, eds W-B. Souffrant and H. Hagemester (EAAP-publication No. 80: Dummerstorf).

## REDUCED PLASMA CONCENTRATIONS OF GLUTAMINE AND ITS METABOLITES IN WEANED PIGS

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Atrophy of the gut is common in weaned piglets and is often associated with an absence of gut substrates and/or catabolic stress. This atrophy may be the result of a lack of glutamine because this amino acid is not only the primary respiratory fuel for gut enterocytes but also provides amide nitrogen that may support nucleotide biosynthesis (Windmueller, 1984). Despite the abundance of glutamine in plasma and muscle, there are conditions of catabolic stress where the intra-cellular concentration of glutamine may decline by as much as 50% and plasma concentrations by as much as 30%. Elevation of glucocorticoid concentrations in blood, as experienced during stress, may increase oxidation of glutamine by increasing glutaminase activity.

At weaning exogenous supplies of glutamine generally disappear because sows' milk, a major source of glutamine, is no longer available and piglets often fast or eat very small amounts of dry food. The sudden withdrawal of exogenous glutamine should stimulate its mobilisation from endogenous sources. If endogenous sources are inadequate it might be expected that, instead of the usual rise in plasma amino acids associated with a fast, the concentrations of glutamine and its breakdown products, glutamate and alanine, would fall.

Twenty piglets were selected randomly from five sows (4 piglets from each sow) and allocated at random to one of two treatments (n=10); a weaned group and a suckled group. At 21 days of age the piglets were either weaned or left to be suckled by the sow. Blood was collected by cardiopuncture from all pigs at 24 and 48 h after the time of weaning to determine the plasma concentrations of glutamine and its breakdown products, glutamate and alanine. Plasma glutamine was 50% lower ( $P<0.05$ ) in weaned than in suckled piglets at both 24 and 48 h after weaning. Significant decreases ( $P<0.05$ ) were also found for plasma glutamate and alanine (Figure 1). Weaned piglets consumed almost no feed ( $5 \pm 0.7$  g) on the first day and only  $75 \pm 26$  g on the second day after weaning.

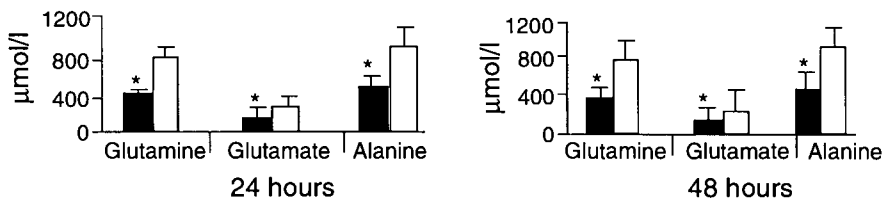


Figure 1. Plasma concentrations of glutamine, glutamate and alanine in weaned pigs (■) at 24 and 48 h after weaning and in sucking (□) piglets at the same ages (mean  $\pm$  SEM). \*Differences between sucking and weaned pigs were significant,  $P<0.05$ .

The decline in plasma glutamine and its breakdown products after weaning indicates that endogenous glutamine sources are incapable of maintaining plasma levels at this time. Thus glutamine is in short supply in the post-weaning period and, if it is essential for maintaining the integrity of the enterocytes, the decline in its availability may well be associated with the gut atrophy which occurs in newly weaned piglets. It is proposed that glutamine might be a conditionally-essential amino acid for weaned piglets.

### References

- WINDMUELLER, H.G. (1984). In "Glutamine metabolism in mammalian tissues", eds D. Haussinger and H. Sies. (Springer: Heidelberg).

## GLUTAMINE STIMULATES INTESTINAL HYPERPLASIA IN WEANED PIGLETS

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Gut atrophy is common in weaned piglets. The atrophy might be due to a deficiency of glutamine, the main respiratory fuel for enterocytes. It has been found that weaning is associated with a 50% decline in plasma glutamine concentrations (Ayonrinde *et al.*, 1995). Glutamine supplementation to parenteral diets of rats has been effective in reversing the gut atrophy normally associated with this form of nutrition (Platell *et al.*, 1993). Enriching weaner diets with glutamine might help to ameliorate the structural integrity of the gut.

Twenty piglets were selected at random from five sows (4 piglets from each sow) and, at 21 days of age, were weaned and offered a cereal-based diet with either 4% glutamine or 4% glycine. Five days later piglets were slaughtered. Two standardised segments of jejunum and ileum were taken from each piglet. One segment was used to measure villous height and crypt depth. The mucosa was scraped off the other segment with a glass slide and analysed for mucosal protein, DNA and glutaminase activity. These values are expressed per unit gut length (cm). Apart from gut protein, glutamine significantly ( $P < 0.05$ ) improved all indices of jejunal and ileal integrity measured. Mean live-weights at slaughter of the piglets fed glutamine ( $6.6 \pm 0.48$  kg) were slightly higher than those fed glycine ( $6.4 \pm 0.56$  kg) but the difference was not significant.

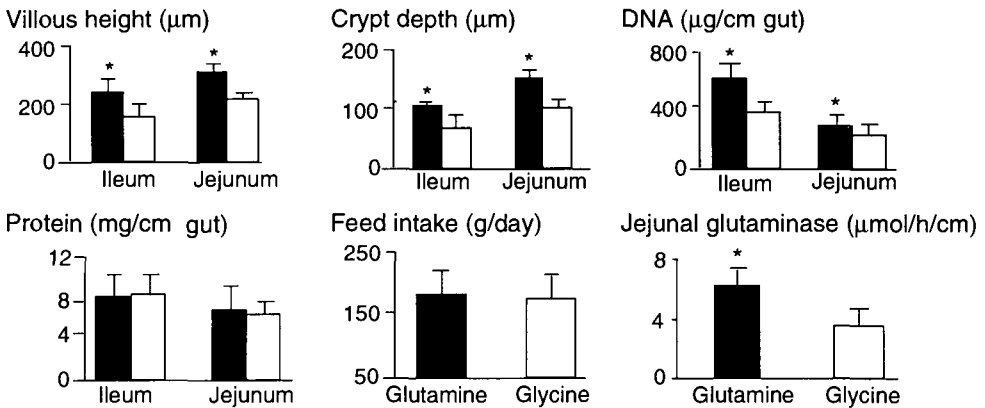


Figure 1. Villous height, crypt depth, DNA, protein, glutaminase activity and feed intake of piglets fed glutamine (■) and glycine (□) diets for 5 d after weaning (mean ± SEM). \*Differences between treatments were significant,  $P < 0.05$ .

This study demonstrates that a glutamine-enriched diet helps to maintain the integrity of the gut of weaned piglets and suggests that glutamine may be an essential amino acid under some circumstances such as in the immediate post-weaning period.

### References

- AYONRINDE, A.I., WILLIAMS, I.H., McCAULEY, R. and MULLAN, B.P. (1995). In "Manipulating Pig Production V", p. 179, eds D.P. Hennessy and P.D. Cranwell. (Australasian Pig Science Association: Werribee).
- PLATELL, C., McCAULEY, R., McCULLOCH, R. and HALL, J. (1993). *Journal of Parenteral and Enteral Nutrition*. 17:348-354.

## DIGESTIBILITY OF BOVINE IMMUNOGLOBULIN IN THE PIGLET

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Bovine immunoglobulin preparations may be protective against diseases of the gastro-intestinal tract when fed to humans or animals (Mietens *et al.*, 1983). However, to be effective, the immunoglobulins must resist digestion and remain active in the gastro-intestinal tract. Therefore, the aim of this study was to measure the survival of orally fed bovine Immunoglobulin G (IgG) along the gastro-intestinal tract.

Six Large White  $\times$  Landrace piglets, which had been weaned at 4 weeks when they were  $7.8 \pm 0.71$  kg live-weight (mean  $\pm$  SD), were used in the experiment. At five weeks of age piglets were fed a diet containing 4.3 g bovine IgG/kg air dry feed for 14 d. On day 15, the piglets ( $14.97 \pm 0.57$  kg live-weight) were fed at hourly intervals for 6 h ( $6 \times 100$  g of feed) and then slaughtered 1 h after the last feed. Digesta were collected from the proximal (S1), medial (S2) and terminal (S3) parts of the small intestine, the caecum and the colon. Diet and digesta were analysed for bovine IgG using polyclonal-based radial immunodiffusion kits (Binding Site Ltd, Institute of Research and Development, Birmingham, UK). The amount of IgG in the diet and digesta were expressed as g IgG /g indigestible chromium marker. The diet contained 1.486 g IgG /g chromium. Resistance to digestion was measured as the percentage of undigested IgG (% IgG), calculated from the ratio of IgG in the digesta to IgG in the diet. A linear model was used to estimate the effect of location in the digestive tract on the amount of IgG present and the % IgG surviving digestion (SAS, 1985). Least-square means for IgG and % IgG for each location are given in Table 1.

**Table 1.** Least-square means and standard error of the mean (SEM) for the amount of IgG per unit chromium (IgG) and the percentage of undigested IgG (%IgG) in digesta collected from the proximal- (S1), medial- (S2) and terminal- (S3) thirds of the small intestine, the caecum and the colon of piglets.

Location	Least-squares means					SEM
	S1	S2	S3	Caecum	Colon	
IgG	0.754	0.700	0.150	0.013	0.001	0.22
% IgG	50.7	47.1	10.1	0.9	0.1	14.9

There was a significant ( $P < 0.001$ ) effect of location on both IgG and % IgG. The percentage of undigested bovine immunoglobulins in the digesta is higher in the proximal and medial parts of the small intestine than in the terminal part. No undigested IgG was found in the caecum and colon. It is concluded that orally fed bovine immunoglobulin (IgG) can be found in the proximal and medial small intestine in amounts which could be sufficient to prevent or treat upper gastro-intestinal tract diseases.

### References

- MIETENS, C., HILPERT, H. and WERCHAU H. (1983). In "Acute diarrhea: its nutritional consequences in children", pp. 111-122, ed. J.A. Bellanti. (Raven Press: New York).
- SAS. (1985). "User's Guide: Statistics." 5th edn. (Statistical Analysis System Institute: Cary, North Carolina).

## PORCINE PANCREATIC EXOCRINE FUNCTION DURING THE FIRST FIVE DAYS AFTER WEANING

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It is well established that at weaning the change from milk to solid food plays an important role in the maturation of the gastro-intestinal (GI) tract and the exocrine pancreas (Pierzynowski, 1991; Cranwell, 1995). The aim of the present study was to monitor the changes in pancreatic function from the day before until the fifth day after weaning in pigs.

Four, 23 day-old, Swedish Landrace sucking piglets were surgically prepared with pancreatic duct catheters, duodenal re-entrant T cannulas and jugular vein catheters (Pierzynowski, 1991). One day before weaning (30 days of age) pancreatic juice was collected for 1 h before and 1 h after a morning sucking (at 0800 h approximately) and an evening sucking (at 2000 h approximately). At weaning the pigs received a standard weaner diet (Växfor, Lantmännen, Sweden) containing 15.5 % crude protein and 12.2 MJ ME/kg, *ad libitum*. At 1, 2, 3 and 5 d after weaning pancreatic juice was collected continuously for 24 h. The volume of pancreatic secretion was measured at hourly intervals; 1.0 ml was taken for analysis and the remaining juice was returned to the animal by way of the duodenal cannula, by continuous infusion, at the same rate as it had been collected during the previous hour. Protein and digestive enzymes in pancreatic juice were measured utilizing methods described by Pierzynowski (1991).

**Table 1. Body-weight, feed intake, volume of pancreatic juice (PJ) and amounts of soluble protein and trypsin secreted in PJ before and after weaning (mean  $\pm$  SD).**

	Day -1	Day 1	Day 2	Day 3	Day 5
Body-weight (kg)	5.2 $\pm$ 0.5	5.5 $\pm$ 0.4	-	5.2 $\pm$ 0.4	5.3 $\pm$ 0.4
Feed intake (g)	-	0.0 $\pm$ 0.0	110 $\pm$ 90 <sup>a</sup>	130 $\pm$ 50 <sup>a</sup>	220 $\pm$ 100 <sup>b</sup>
Volume (ml/h)	3.6 $\pm$ 2.1 <sup>a</sup>	4.4 $\pm$ 3.3 <sup>ab</sup>	5.9 $\pm$ 2.9 <sup>ab</sup>	6.2 $\pm$ 1.9 <sup>ab</sup>	9.5 $\pm$ 3.2 <sup>b</sup>
(ml/g food)	-	-	3.7 $\pm$ 5.4	1.1 $\pm$ 0.4	1.3 $\pm$ 0.7
Protein (mg/h)	8 $\pm$ 8 <sup>a</sup>	12 $\pm$ 12 <sup>ab</sup>	8 $\pm$ 19 <sup>ab</sup>	5 $\pm$ 10 <sup>ab</sup>	25 $\pm$ 17 <sup>b</sup>
(mg/g food)	-	-	9 $\pm$ 13	3 $\pm$ 3	4 $\pm$ 3
Trypsin (U/h)	3 $\pm$ 3 <sup>a</sup>	5 $\pm$ 6 <sup>ab</sup>	8 $\pm$ 8 <sup>ab</sup>	6 $\pm$ 3 <sup>ab</sup>	16 $\pm$ 17 <sup>b</sup>
(U/g food)	-	-	4 $\pm$ 5	1 $\pm$ 1	3 $\pm$ 3

<sup>ab</sup>Means in the same row with different superscripts are significantly different ( $P < 0.05$ ).

An increase in the secretion of PJ, and in the output of protein and trypsin, was observed during the first 5 days after weaning and coincided with the increase in the consumption of solid food. The factors responsible for the increase in PJ secretion include the presence of solid food in the GI tract. However, the cessation of milk intake may also be important since milk  $\beta$ -casomorphins are known to inhibit pancreatic secretion via the stimulation of somatostatin secretion (Schusdziarra *et al.*, 1983).

### References

- CRANWELL, P.D. (1995). In: "The Neonatal Pig: Development and Survival", pp. 99-154, ed. M.A. Varley. (CABI: Wallingford).
- PIERZYNOWSKI, S.G. (1991). Development and regulation of porcine pancreatic function with special reference to the exocrine pancreas. PhD Thesis. University of Lund.
- SCHUSDZIARRA, V., SCHICK, R., de la FUENTE, A., HOLLAND, A., BRANTL, V. and PFEIFFER, E.F. (1983). *Endocrinology*. 112:1948-1951.

## COLOSTRUM FEEDING STIMULATES PANCREATIC GROWTH IN NEWBORN PIGS

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In newborn pigs the gastro-intestinal tract and the pancreas undergo intense growth and functional maturation during the immediate postnatal period (Xu and Cranwell, 1990; Xu *et al.*, 1992; Tarvid *et al.*, 1994). The mechanisms regulating these changes are not fully understood. This paper reports the finding that colostrum feeding stimulates pancreatic growth in newborn pigs.

A total of 16 newborn unsuckled piglets (12 males and four females) were obtained from four litters of Landrace pigs. Four piglets obtained from each litter were randomly assigned into four treatment groups. Animals in group 1 were euthanased within 4 h of birth and without any feeding. Animals in groups 2 to 4 were bottle fed for 3 d with equal volumes of 5% lactose solution or sow colostrum or trypsin-hydrolysed sow colostrum respectively, and then euthanased. Two hours prior to euthanasia each animal received four intra-peritoneal injections of bromodeoxyuridine (5 mg/kg body-weight for each injection) at 30 min intervals. Immediately after euthanasia the pancreas from each piglet was dissected and weighed. A block of pancreatic tissue from the head region was fixed in Bouin's fluid and processed for histological and morphological analyses. The remaining pancreatic tissue was stored at -20°C for chemical analyses. The incorporation rate of bromodeoxyuridine into the pancreatic cells (an index of cell mitosis) was measured by an immuno-staining technique (Xu *et al.*, 1994). Data were analysed by one-way analysis of variance followed by the least significant difference test.

**Table 1. Body-weight, pancreatic weight and pancreatic cell mitotic index (mean  $\pm$  SEM) in newborn pigs, and in pigs fed for 3 d with 5% lactose solution or colostrum or trypsin-hydrolysed colostrum (Tr. Col.).**

	Newborn (n=4)	Lactose (n=4)	Colostrum (n=4)	Tr. Col. (n=4)
Body-weight (kg)	1.41 $\pm$ 0.06	1.28 $\pm$ 0.12	1.54 $\pm$ 0.13	1.48 $\pm$ 0.16
Pancreas weight (g)	1.75 $\pm$ 0.12 <sup>a</sup>	1.55 $\pm$ 0.26 <sup>a</sup>	2.68 $\pm$ 0.24 <sup>b</sup>	2.55 $\pm$ 0.29 <sup>b</sup>
Pancreas/body-weight (g/kg)	1.24 $\pm$ 0.4 <sup>a</sup>	1.20 $\pm$ 0.12 <sup>a</sup>	1.74 $\pm$ 0.08 <sup>b</sup>	1.73 $\pm$ 0.09 <sup>b</sup>
Mitotic index (cell/mm <sup>2</sup> )	49 $\pm$ 10 <sup>a</sup>	103 $\pm$ 64 <sup>a</sup>	392 $\pm$ 28 <sup>b</sup>	734 $\pm$ 80 <sup>c</sup>

<sup>abc</sup>Mean values in the same row with different superscripts are significantly different ( $P < 0.05$ ).

The preliminary results (Table 1) show that feeding with colostrum or trypsin-hydrolysed colostrum, but not lactose solution, stimulated pancreatic growth in newborn pigs. Of particular interest is the observation that trypsin-hydrolysed colostrum had a greater mitogenic activity compared to untreated colostrum. The substances in the colostrum responsible for such trophic effects remain to be determined.

### References

- TARVID, I., CRANWELL, P.D., MA, L. and VAVALA, R. (1994). In "Proceedings of the VIth International Symposium on Digestion in Pigs", pp. 199-202, eds W-B. Souffrant and H. Hagemester (EAAP-publication No. 80: Dummerstorf).
- XU, R.J. and CRANWELL, P.D. (1990). *Journal of Developmental Physiology*. 13:315-326.
- XU, R.J., MELLOR, D.J., BIRTLES, M.J., BREIER, B.H. and GLUCKMÁN, P.D. (1994). *Biology of the Neonate*. 66:280-287.
- XU, R.J., MELLOR, D.J., TUNGTHANATHANICH, P., BIRTLES, M.J., REYNOLDS, G.W. and SIMPSON, H.V. (1992). *Journal of Developmental Physiology*. 18:161-172.

## STABILITY OF GASTRIN IN THE GASTRO-INTESTINAL LUMEN OF THE PIG

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Gastrin is a gastro-intestinal (GI) regulatory peptide produced by the G cells in the pyloric region of the stomach. It has stimulatory effects on gastric acid secretion and GI mucosal growth. Under electrical vagal stimulation gastrin is released from the G cells into the blood stream as well as into the gastric lumen (Uvnas-Wallensten, 1977). It has been suggested that luminal gastrin may play a role in stimulating GI mucosal growth if the peptide can survive luminal digestion (Xu and Cranwell, 1991). The objectives of this study were to examine the stability of gastrin in the GI lumen of pigs of different ages and the effects of porcine milk on the stability of gastrin in the GI tract.

Contents from the stomach, and proximal, medial and distal small intestine were collected from nine 3-day-old sucking and three 5-week-old weaner Large White  $\times$  Landrace pigs, and three adult pigs of undetermined breed. The sucking and weaner pigs were fed 2 h before they were killed and the adult pigs were fasted for >24 h. The contents were diluted with an equal volume of saline, and food particles were removed by centrifugation. Iodine ( $^{125}$ I) labelled human little gastrin (G17) was incubated *in vitro* with the GI contents at 37°C for 20 min. Hydrolysis of G17 was determined by measuring radioactivity in the soluble fraction following treatment with trichloroacetic acid. The inhibitory activity of porcine milk on luminal G17 hydrolysis was examined by the addition of defatted porcine milk or its casein or acid soluble fractions into the incubation medium containing the intestinal fluid. The casein fraction was separated from the acid soluble fraction by adjusting milk pH to 4.6, followed by centrifugation.

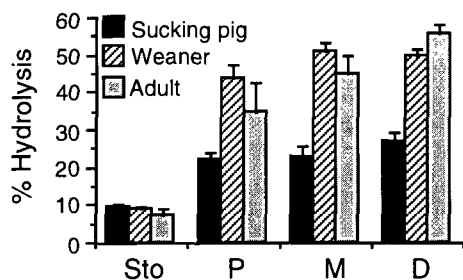


Figure 1. Hydrolysis of G17 by the luminal fluids of the stomach (Sto), the proximal (P), medial (M) and distal (D) small intestine (mean  $\pm$  SEM).

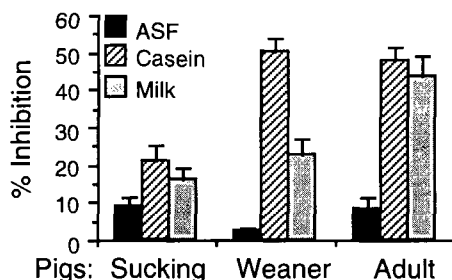


Figure 2. Inhibition of G17 hydrolysis in proximal small intestinal fluid by the acid soluble fraction of porcine milk (ASF), casein and defatted porcine milk (mean  $\pm$  SEM).

Hydrolysis of G17 in gastric fluid was low (<10%) for all age groups (Figure 1). The hydrolytic activity of intestinal fluids was high, but the activity was significantly lower ( $P < 0.05$ ) in sucking pigs than in weaner and adult pigs. Defatted porcine milk inhibited G17 hydrolysis by intestinal fluid, with the casein component being more effective than the acid soluble fraction (Figure 2).

The results show that gastrin is more stable in the GI fluids of sucking pigs than in older animals, and porcine milk can protect gastrin from luminal digestion. These observations suggest that luminal gastrin may survive GI digestion and play a role in regulating GI development in sucking pigs.

### References

- UVNAS-WALLENSTEN, K. (1977). *Gastroenterology*. 73:487-491.  
 XU, R.J. and CRANWELL, P.D. (1991). *Comparative Biochemistry and Physiology*. 98B:615-621.

## PHYSIOLOGICAL RESPONSE OF LACTATING SOWS TO FEEDING RAPESEED MEAL "00" AND MICROBIAL PHYTASE

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In many countries rapeseed meal is available in increasing amounts. Its potential as a feed and its influence on the physiology of lactating sows remain to be investigated. The utilisation of phytase-bound phosphorous (P) in rapeseed meal may be enhanced by adding microbial phytase. The objective of this study was to measure exocrine pancreatic outflow; some characteristics of blood and milk; apparent digestibility of total P; maternal weight loss and piglet weight gain during lactation when sows were fed cereal-based diets with either 0 or 10% of rapeseed meal which contained a very low glucosinolate and erucic acid content "00"; supplemented without or with microbial phytase (0 or 800 phytase units [PTU]/kg). For this purpose sixteen primiparous sows were fitted with a pancreatic duct catheter, a duodenal silicone T-cannula, and a permanent catheter in the femoral artery. At farrowing, the sows weighing approximately 158 kg were assigned randomly to four treatments (a 2 x 2 factorial arrangement), each with four replicates. Treatment 1 (control) consisted of maize and extracted soya bean meal (SBM) as major components. In Treatment 2, SBM was substituted for rapeseed meal "00" (RSM) on an isonitrogenous basis. Treatments 3 and 4 were formulated by adding microbial phytase (800 PTU/kg) to Treatments 1 and 2, respectively. These treatments contained approximately 150 g of crude protein, 8 g of lysine, 4.2 g of total P and 7.3 g of phytic acid per kg. Milk samples after injecting oxytocin were obtained four times, during a 4-wk lactation. The lactating sows were given *ad libitum* access to feed and water and had an average of 10.1 piglets per litter. The responses to added RSM and microbial phytase are presented in Table 1.

**Table 1. Effects of dietary supplementation with rapeseed meal and phytase on pancreatic function, phosphorus and nitrogen utilisation and sow and piglet performance during lactation.**

Main effects	Rapeseed meal "00"		Microbial phytase		SED
	0	10%	0	800 PTU/kg	
Pancreatic juice outflow (ml/h/kg, live-weight)	3.65	2.88	3.20	3.33	0.49
Trypsin activity (U/h/kg, live-weight)	4.91	4.60	4.51	5.00*	0.20
Plasma inorg. P (mmol/L)	1.42	1.59	0.75	1.35***	0.12
Milk inorg. P (mmol/L)	586.12	505.44	484.33	607.23*	45.17
Urine P (mmol/L)	1.70	2.00	1.03	2.67*	0.68
Digestible P (%)	40.1	33.6*	28.4	45.2***	2.63
Digestible N (%)	78.8	77.0	76.5	79.3*	1.25
Sow feed intake (kg/d)	4.56	4.86	4.77	4.65	0.56
Sow weight loss (kg)	16.0	18.1	17.3	16.8	2.2
Piglet weight gain (kg)	6.8	6.6	6.7	6.7	0.4

\* and \*\*\* indicate significant differences at  $P < 0.05$  and  $P < 0.001$  respectively.

Overall, rapeseed meal "00" can be used at levels of about 10% as a substitute for SBM in lactation diets. Addition of microbial phytase improved trypsin activity and apparent digestibility of total phosphorus and nitrogen. The use of microbial phytase improves the degradation of phytase-bound phosphorus in rapeseed meal thus reducing the need for P supplements. A consequence of reducing the need for P supplementation will be a reduction in excreted P in faeces by about 20 %.



## DIAGNOSIS OF SWINE DYSENTERY AND INTESTINAL SPIROCHAETOSIS BY THE USE OF POLYMERASE CHAIN REACTION TESTS ON FAECAL SAMPLES

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The polymerase chain reaction (PCR) is a technique based on amplification of specific DNA sequences, the products of which may be detected using electrophoresis (Saiki *et al.*, 1985). The DNA sequences from *Serpulina hyodysenteriae*, the causative agent of swine dysentery (SD) (Combs and Hampson, 1992), and from the spirochaete previously called "*Anguillina coli*" (Lee *et al.*, 1993), which is associated with intestinal spirochaetosis (IS) (Park *et al.*, 1995), were used to design PCR tests, and these were applied to the detection of these bacteria in pigs.

The PCRs were optimised by applying the tests to purified DNA extracted from 49 strains of *S. hyodysenteriae*, 12 strains of the spirochaete associated with IS, and 46 strains from other closely related genetic groups, such as *S. innocens*, *S. intermedius*, *S. murdochii* and *Brachyspira aalborgi*, all of which had been previously differentiated using the technique of multilocus enzyme electrophoresis (Lee *et al.*, 1993; Lee and Hampson, 1994). These included strains from Australia, the UK, the USA, and Canada. These were isolated mainly from pigs, but strains from humans, chickens, rheas, and an isolate from a swan were included. Both PCRs were specific for the organism they were designed to detect, and an internal oligonucleotide probe was used to confirm the identity of the *S. hyodysenteriae* DNA product. Variations on the PCR protocols to increase their sensitivity, including the use of hot starts and nested reactions, were evaluated.

The treatment of faeces prior to PCR is an important part of clinical diagnosis, because of the large number of PCR inhibitors which are present in faeces (Newton and Graham, 1994). A number of different regimes for preparing the faeces were examined, including the use of immunomagnetic separation of spirochaetes, commercial DNA extraction kits, Instagene, phenol/chloroform DNA extraction, diatomaceous earth DNA extraction, and culture resuspension prior to diatomaceous earth extraction (CRDEX). In the latter technique the total growth on blood agar plates from samples which had been cultured was collected and the DNA harvested. The CRDEX technique was the best method of treatment, since  $10^2$ - $10^4$  spirochaetes could be detected in 0.2 g of faeces. All other techniques could only detect  $\geq 10^6$  spirochaetes in 0.2 g of faeces. The use of CRDEX would be likely to correctly diagnose pigs with clinical SD or IS, but might not detect carrier pigs, which would probably be shedding fewer organisms than this (Kinyon *et al.*, 1977).

Combinations of CRDEX and PCR tests were applied to 22 porcine clinical faecal samples, and to the faeces of seven experimentally infected pigs. The tests successfully detected the two spirochaetes respectively in all samples from which they could be cultured and isolated, and from four samples from which spirochaetes could not be isolated. The CRDEX technique reduced the time required for diagnosis from three weeks to less than ten days. The use of sequential PCRs on single DNA extracts is becoming an important tool for the rapid diagnosis of both SD and IS.

### References

- COMBS, B.G. and HAMPSON, D.J. (1992). *Proceedings of the 12th International Pig Veterinary Society Congress*, The Hague, Netherlands, p. 150.
- KINYON, J.M., HARRIS, D.L. and GLOCK, R.D. (1977). *Infection and Immunity*. 15:638-646.
- LEE, J.I. and HAMPSON, D.J. (1994). *Journal of Medical Microbiology*. 40:365-371.
- LEE, J.I., HAMPSON, D.J., LYMBERY, A.J. and HARDERS, S.J. (1993). *Veterinary Microbiology*. 34:273-285.
- NEWTON, C.R. and GRAHAM, A. (1994). "PCR" (BIOS Scientific Publishers Limited: Oxford).
- PARK, N.Y., CHUNG, C.Y., McLAREN, A.J., ATYEO, R.F. and HAMPSON, D.J. (1995). *FEMS Microbiology Letters*. 125:225-230.
- SAIKI, R.K., SCHARF, S., FALOONA, F., MULLIS, K.B., HORN, G.T., ERLICH, H.A. and ARNHEIM, N. (1985). *Science*. 230:1350-1354.

## THE EFFECT OF BODY-WEIGHT ON THE MARGINAL EFFICIENCY OF NITROGEN UTILIZATION IN PIGS

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Knowledge of factors influencing the marginal efficiency of protein (amino acid) utilization is crucial for a factorial calculation of amino acid requirements and the development of pig growth simulation models. Several authors concluded that the efficiency of protein utilization decreased with increasing body-weight (BW), eg., Rao and McCracken (1991). However, in most of these studies a decrease in efficiency may have been caused by an increase in the proportion of amino acids used for maintenance with increasing BW, and/or by a decrease in the required dietary protein:energy ratio, and consequently an over-supply of dietary amino acids. Therefore, this study was conducted to determine the effect of BW on the marginal efficiency of nitrogen retention.

Forty-eight entire male pigs (commercial hybrids, VOC Nieuw Dalland, The Netherlands) were divided into three groups each of 16 pigs. The nitrogen retention (NR) of pigs of these three groups was measured by the balance technique (urine and faeces collection) at 30, 60 or 90 kg BW. In the growth period from 24 kg BW until the start of the balance period the pigs received sufficient nitrogen (N) for maintenance plus 25 g N (ileal digestible ideal protein basis) per d above maintenance. In each of the three balance periods the 16 pigs were allocated to four levels of N intake above maintenance: 10, 15, 20, and 25 g N/d and after 7 d of adaptation NR was measured over 5 d. Throughout the experiment energy was supplied at a level of  $3 \times$  maintenance ( $ME_m = 458 \text{ kJ/kg}^{0.75}$ ). Lysine requirements for maintenance were taken as  $36 \text{ mg/kg}^{0.75}$  (Fuller *et al.*, 1989). The dietary amino acid pattern was consistent with lysine being the first limiting amino acid. Thus, the efficiency of N utilization was determined by the utilization of dietary lysine.

Nitrogen retention increased linearly ( $P < 0.001$ ) with increasing N intake (Figure 1). The relationships between NR (Y) and digestible N intake (X) expressed in  $\text{g/kg}^{0.75}$  ( $\pm$  SE of estimate) were:

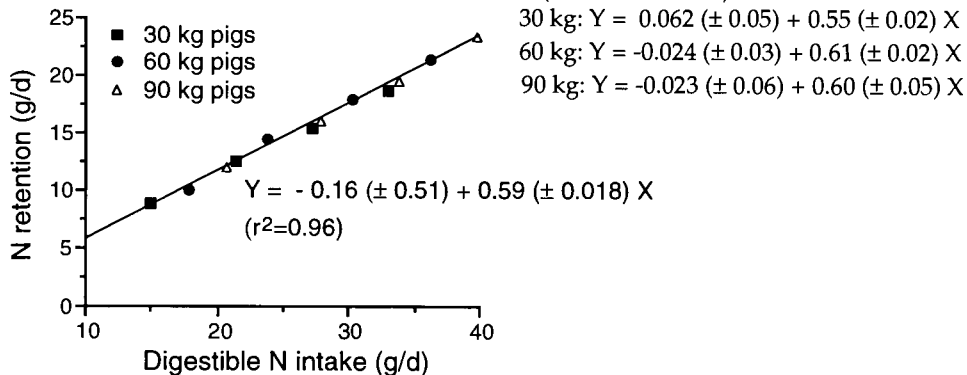


Figure 1. Relationship between N intake and N retention in pigs of 30, 60 and 90 kg BW.

No significant differences in intercept were found among the three balance periods. Using one intercept, the respective slopes, representing the marginal efficiency, were 0.57, 0.59, and 0.57. No effect of BW on this efficiency was found. It is concluded that these results do not support the assumption of a decreasing marginal efficiency of protein utilization with increasing BW. A possible influence of BW when amino acids other than lysine are limiting is still to be studied.

### References

- RAO, D.S. and McCracken, K.J. (1991). *Animal Production*. 52:499-507.  
 FULLER, M.F., McWILLIAM, R., WANG, T.C. and GILES, L.R. (1989). *British Journal of Nutrition*. 62:255-267.

## AUSPIG INCREASES PROFITS BY IMPROVED FEED FORMULATION

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AUSPIG uses many physical and managerial factors to simulate the growth of pigs (Black *et al.*, 1986) and, when applied to commercial piggeries, it can lead to considerable savings in feed costs and increased profits (Mullan *et al.*, 1993). Although AUSPIG shows great potential for use in determining amino acid specifications for pigs within individual herds, the majority of pig producers lack the confidence to use the model to re-specify diets. This experiment was designed to test the hypothesis that AUSPIG can simulate the growth of entire male finisher pigs, from farm records, precisely enough to be of value in making decisions that will increase profit.

A simulation data set was established in AUSPIG from farm records of a 1200-sow piggery. The genotype was defined within AUSPIG so that the growth of the pigs fed the existing diets accurately simulated the average liveweight (92.8 kg) and backfat (12.4 mm) at sale (151 d). Two AUSPIG finisher diets were formulated using the same ingredients and level of digestible energy (13.5 MJ DE/kg) as the existing finisher diet (control, C; \$245/t). AUSPIG diets were formulated to supply either 90% (\$229/t) or 105% (\$234/t) of the most limiting amino acid requirement for the average entire male at 101 days of age (45 kg). The control diet (C) diet was predicted to supply 115% of amino acid requirements. One hundred and eighty entire male Large White × Landrace pigs were group-housed (0.7 m<sup>2</sup>/pig) in twelve pens and fed one of the three treatment diets *ad libitum* from 101 days of age for nine weeks until slaughter (164 d). Individual carcass weight and backfat at the P2 position were collected from the processors' records.

**Table 1. Finisher performance and net income of pigs fed either a control diet or diets formulated by using AUSPIG to meet 90% (A90) or 105% (A105) of the amino acid requirement for maximum growth of the entire male pig at a live-weight of 45 kg.**

Diet	Liveweight at:		Hot carcass weight (kg)	P2 (mm)	Feed used <sup>1</sup> (kg)	Profit <sup>2</sup> (\$/pig)
	Start	Slaughter				
Control	38.6	93.9	63.8	13.0	122.7	-
A105	38.7	94.8	64.4	13.7	122.5	1.62
A90	38.0	92.2	62.7	13.5	136.1	-3.22

Column means not significant ( $P > 0.05$ ). <sup>1</sup>Calculated on a pen-basis. <sup>2</sup>Relative to control.

Although not significant, pigs fed the A90 diet tended to have lighter carcasses of lower value and ate more than those fed the C and A105 diets. A saving in feed cost of \$1.44/pig, for pigs fed the lower specified A105 diet improved net income, supporting our hypothesis that AUSPIG can benefit commercial producers in making decisions that will increase profits. All pigs started the experiment at a lighter weight than the average simulated entire male at 45 kg, and as a consequence, amino acid requirements were higher than assumed. Using actual start weights, AUSPIG predicted that the A105 diet would supply only 92% and the A90 diet only 84% of the amino acid requirement for maximum growth. This might explain the fatter carcasses of pigs fed the AUSPIG diets compared to those fed the C diet. Another possible explanation is the supply of amino acids from the diets might have been less than assumed. However, growth rates were similar between the C and A105 diet, which suggests that AUSPIG might be over-estimating amino acid requirements in certain genotypes.

### References

- BLACK, J.L., CAMPBELL, R.G., WILLIAMS, I.H., JAMES, K.J. and DAVIES, G.T. (1986). *Research and Development in Agriculture*. 3:121-145.  
 MULLAN, B.P., DAVIES, G.T. and CHARLES, M. (1993). In "Manipulating Pig Production IV", p. 220, ed. E. S. Batterham. (Australasian Pig Science Association: Attwood).

## THE EFFECT OF AUSPIG DETERMINED DIETARY LYSINE LEVELS ON THE GROWTH PERFORMANCE OF PIGS BETWEEN 63 AND 112 DAYS OF AGE

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Recent AUSPIG (Black *et al.*, 1986) simulations at Bunge Meat Industries (BMI) indicate that the dietary lysine concentration of current grower diets may be substantially higher than the pigs actual tissue requirements. The present experiment was designed to examine the relationship between AUSPIG determined dietary lysine levels and growth performance of pigs between 63 and 112 days of age.

Four AUSPIG diets of a similar energy density (14 MJ DE/kg) were formulated to provide 100, 115, 125 and 135% of simulated amino acid requirements for the BMI genotype at 25 kg live-weight. Eight hundred and eighty pigs comprising equal numbers of males and females were allocated in pens of 20 amongst the four diets at 63 days of age (25 kg live-weight). The diets were offered *ad libitum* from 63 to 112 days of age. Pig performance was measured between 63 and 84, and 85 and 112 days of age.

The results (Table 1) showed that growth performance improved in a linear fashion with increasing lysine to the highest levels tested in the period 63 to 84 days of age. In contrast, neither growth rate nor feed to gain were significantly affected by lysine in the period 85 to 112 days of age.

**Table 1. The effect of dietary available lysine content on the growth performance pigs between 63 and 112 days of age.**

Lysine (g/MJ DE)	63-84 d		85-112 d		63-112 d	
	Daily gain (g)	Feed:gain	Daily gain (g)	Feed:gain	Daily gain (g)	Feed:gain
0.54	688 <sup>a</sup>	2.05 <sup>a</sup>	746	2.33	719 <sup>a</sup>	2.22 <sup>a</sup>
0.62	724 <sup>ab</sup>	1.91 <sup>ab</sup>	750	2.32	743 <sup>ab</sup>	2.14 <sup>ab</sup>
0.70	742 <sup>ab</sup>	1.88 <sup>ab</sup>	727	2.37	742 <sup>ab</sup>	2.13 <sup>b</sup>
0.77	769 <sup>b</sup>	1.81 <sup>b</sup>	731	2.35	760 <sup>b</sup>	2.07 <sup>b</sup>
LSD <sub>(P=0.05)</sub>	68	0.15	67	0.17	38	0.09

Within columns a,b, differ at P<0.05

The linear daily gain response obtained between 63 and 84 days indicates that lysine concentration may be limiting maximum growth potential in the BMI genotype when fed at AUSPIG recommended levels. The results from 63 to 84 and 85 to 112 days suggest that the pigs tissue and dietary amino acid "requirements" change rapidly over this period. These changes may be a result of the selection for efficient lean growth, typifying modern commercial genotypes, altering the response of growing pigs to lysine.

The data supports the concept of phase feeding as a means of further improving the efficiency and profitability of pig production. However, further research is required to more accurately establish the responses to lysine in the period 85 to 112 days of age following the feeding of a common diet to this age.

### References

BLACK, J.L., CAMPBELL, R.G., WILLIAMS, I.H., JAMES, K.J. and DAVIES, G.T. (1986). *Research and Development in Agriculture*. 3:121-145.

## $\beta$ -GLUCANASE SUPPLEMENTATION OF BARLEY-BASED PIG FEEDS REDUCES DIGESTA VISCOSITY AND SHORT CHAIN FATTY ACID CONCENTRATION

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Recent work has shown that enzyme supplementation of barley-based feeds for early weaned pigs improves ileal starch and non-starch polysaccharide (NSP) digestibility (Inbarr *et al.*, 1993). This in turn would explain the improved performance and health status of pigs fed diets fortified with carbohydrate-degrading enzymes (Böhme, 1990). This investigation was undertaken to study the effect of adding  $\beta$ -glucanase to diets based on barley on pig performance and some gastro-intestinal parameters relating to the digestive processes.

A total of 40 pigs (Danish Landrace  $\times$  Large White) weaned at about four weeks of age with a mean initial live-weight of 9.5 kg were divided into five groups of eight and placed in metabolism cages and assigned to one of four dietary treatments based on live-weight, gender and litter. The experimental diets were fed for 21 d. The diets were based on one of two barley cultivars; var. Arra, with low (26 g/kg) and var. Condor with high (48 g/kg)  $\beta$ -glucan content; either with (indicated +) or without (-) added  $\beta$ -glucanase (*Trichoderma longibrachiatum*) to give a 2  $\times$  2 factorial design. The enzyme was added when mixing the feed which was given as a mash. It was considered that pelleting may cause avoid heat damage to the enzyme. Digesta viscosity, short chain fatty acid (SCFA) concentrations in digesta, animal performance and  $\beta$ -glucan digestibility were determined.

Feed intake was unaffected, while the live-weight gain and feed conversion ratio tended to be improved in the presence of the enzyme, although not significantly. The viscosities of samples obtained from the stomach and the three proximal quarters of the small intestine (SI 1-3) were significantly ( $P < 0.05$ ) reduced when the enzyme was added. The SCFA (the sum of acetic, propionic and butyric acid) concentrations in the caecum, colon and rectum were significantly ( $P = 0.04$ ) reduced in the presence of the enzyme (average values over the three sections were 123 mmol/L without and 113 mmol/L with the enzyme added to the diet). The ileal digestibility of  $\beta$ -glucan was 0.32 in diets without added enzyme, and 0.63 in the diets supplemented with the enzyme ( $P < 0.001$ ).

**Table 1.** Viscosity (cPs) of digesta obtained from the stomach and each quarter of the small intestine (SI 1-4), for pigs fed diets based on either Arra or Condor barley cultivars supplemented with (+) or without (-)  $\beta$ -glucanase.

Treatment	Arra -	Arra +	Condor -	Condor +	P-value (enzyme)
Stomach	1.38	1.33	1.71	1.31	0.029
SI 1	1.48	1.43	1.93	1.32	0.021
SI 2	1.93	1.74	2.94	1.77	0.004
SI 3	2.77	2.01	3.82	2.33	0.007
SI 4	2.55	2.44	2.94	2.52	0.306

Supplementation of the diet with  $\beta$ -glucanase changed the conditions in the digestive tract by reducing digesta viscosity and the concentration of SCFA. There was a tendency towards improved pig performance in the presence of the enzymes. In poultry, a high negative correlation between reduced digesta viscosity and bird performance has been reported (Bedford and Classen, 1992). Whether a similar relationship exists in the pig needs further investigation.

### References

- BÖHME, H. (1990). *Landbauforschung Völkenrode*. 40:213-217.  
 INBARR, J., SCHMITZ, M. and AHRENS, F. (1993). *Animal Feed Science and Technology*. 44:113-127.  
 BEDFORD, M.R. and CLASSEN, H. (1992). *Journal of Nutrition*. 122:560-569.

## EFFECT OF PHYTATE, PHYTASE AND LACTIC ACID ON FAECAL DIGESTIBILITY OF ASH AND SOME MINERALS IN PIGS

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Phytic acid, a strong chelating agent in vegetable based feeds, may form insoluble complexes with di- and tri-valent cations, which are poorly digested by pigs. Phytase (Natuphos®) can hydrolyze the phytate complexes, thus increasing the digestibility of phosphorus (P) and minerals which are complexed to phytate. Organic acids may improve the efficacy of phytase by decreasing the gut pH. The effects of phytase and lactic acid on the digestibility of diets with 2 levels of phytate were tested. The design of the experiment, the diet and methods are described by Kemme *et al.* (1995) on page 195 of these proceedings. The basal diet contained 0.55% Ca and 0.30% P.

Mono-calcium phosphate (MCP) addition improved the digestibility of P 43.2 vs 36.1%, ( $P < 0.05$ ), but had no effect on the digestibility of ash, Ca or Mg. The main effects of phytase, Na-phytate and lactic acid on the faecal digestibility of ash, Ca and Mg are given in Table 1.

**Table 1. Apparent total tract digestibility of ash, Ca, and Mg (in %) as affected by Natuphos®, Na-phytate and lactic acid.**

	Natuphos®		Na-Phytate		Lactic Acid		SED
	-	+	-	+	-	+	
Ash	52.2	59.5***	56.9	54.7*	53.5	59.1***	1.03
Ca	44.8	54.5***	49.8	49.5	45.3	54.1***	1.48
Mg	22.0	28.3**	22.3	28.1**	22.7	27.7*	1.71

\*  $P < 0.05$ ; \*\*  $P < 0.01$ ; \*\*\*  $P < 0.001$ ; SED = standard error of difference.

Addition of phytase or lactic acid enhanced the digestion of ash, Ca and Mg. The lactic acid effect was probably as a result of a lower affinity of phytate to cations at the lower gut pH. Na-phytate decreased the digestion of ash, but increased that of Mg, while Ca-digestibility was unaffected. Digestibility of P was affected ( $P < 0.05$ ; SED = 2.69) by the interactions Natuphos® × Na-phytate and Natuphos® × lactic acid (Table 2).

**Table 2. Effect of the interactions Natuphos® × Na-phytate and Natuphos® × lactic acid on P digestibility (%).**

		Na-phytate		Lactic acid	
		-	+	-	+
Natuphos®	-	21.5	30.4	24.7	27.3
Natuphos®	+	47.2	45.2	40.9	51.6

The addition of Na-phytate alone (without phytase) increased P digestibility. When only phytase was added, P-digestibility was increased substantially and was as high as in the diets supplemented with both phytase and Na-phytate. The addition of lactic acid alone had no significant effect on P digestion. Phytase addition to the diets without lactic acid improved P digestibility by 15 percentage units, while with the addition of both Natuphos® and lactic acid the digestibility was increased by 27 percentage units.

It is concluded that Natuphos®-phytase or lactic acid supplementation to a corn soya bean meal based diet had a positive effect on the total tract digestibility of ash, Ca and Mg. Digestibility of P was improved by Natuphos® alone, while there was a synergistic effect of the addition of both phytase and lactic acid.

### References

KEMME, P.A., JONGBLOED, A.W., MROZ, Z., MÄKINEN, M. and KIES, A.K. (1995). In "Manipulating Pig Production V", p. 195, eds D.P. Hennessy and P.D. Cranwell. (Australasian Pig Science Association: Werribee).

## VARIATION IN THE PROTEIN QUALITY OF BLOOD MEALS

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Blood meal is commonly used as a dietary ingredient for pigs. There appears to be considerable variability, however, in the quality of blood meals (Batterham *et al.*, 1986). In the study reported here 12 samples of commercially produced blood meal were analyzed for crude protein (CP, total nitrogen  $\times$  6.25) and 1-fluoro-2,4-dinitrobenzene-reactive lysine (FDNB; Booth, 1971). Apparent ileal protein digestibility values (ADCP) were also determined at slaughter, in 6-week-old, 160 g body-weight Sprague-Dawley male rats ( $n=6$  per blood meal). Chromic oxide was used as an indigestible marker.

Drying, the major process in blood meal production, may be carried out in a batch dryer or by continually moving the material through either a heated rotating drum or a ring shaped dryer. Alternatively the liquid product may be sprayed on to a heated fluid bed.

**Table 1. Method of drying, drying time (min), crude protein (g/100 g DM), apparent ileal digestibility of CP (%) and FDNB reactive lysine (g/kg DM) for 12 New Zealand blood meals.**

Dryer type	Drying time	CP	ADCP	FDNB lysine	Dryer type	Drying time	CP	ADCP	FDNB lysine
Spray	0.5	89.9	94.6	98.8	Ring	5	82.5	87.2	86.0
Ring	1.5	91.6	87.8	88.4	Ring	10	87.9	90.1	98.8
Ring	2.0	89.9	87.4	91.5	Rotary	15	89.7	84.5	97.1
Ring	4.0	88.6	92.0	100.0	Batch	180	87.2	65.1	63.7
Ring	5.0	87.6	88.3	94.9	Batch	180	87.0	63.0	95.1
Ring	5.0	86.4	80.9	93.8	Batch	210	90.7	17.0	60.3

The CP content of the 12 blood meal samples were similar (Table 1), ranging from 82.5-91.6 g/100 g dry matter (DM). In contrast, the mean ADCP was highly variable ranging from 17-94.6%. It is notable that for all drying times less than or equal to 15 min, regardless of manufacturing process, the ADCP was relatively high ranging from 80.9-94.6%. For longer drying times considerably lower ADCP's were observed. The reactive FDNB lysine content was also variable, ranging from 60.3-100 g/kg DM. As for the ADCP, FDNB-reactive lysine contents were similar for blood meals that had undergone minimal heat processing ( $\leq 15$  min) ranging from 86-100 g/kg DM, but for two of the three more severely heated blood meals (heating times, 180 min), FDNB-reactive lysine was considerably lower (60.3 and 63.7 g/kg DM). Apparent ileal protein digestibility appeared to be affected more by the processing than was the FDNB lysine. Apparent CP digestibility offers a more sensitive measure of protein quality than CP content.

The results of this study show that protein digestibility and reactive lysine content are highly variable among blood meal samples. It appears that the batch drying process leads to significantly lower quality blood meals, as meals produced by this method had consistently lower ADCP and generally lower FDNB lysine concentrations compared with samples from other drying procedures. Processing can lead to extremely low quality blood meals as evidenced by the protein digestibility and FDNB values.

### References

- BATTERHAM, E.S., DARNELL, R.E., HERBERT, L.S. and MAJOR, E. (1986). *British Journal of Nutrition*. 55:427-440.
- BOOTH, V.H. (1971). *Journal of the Science of Food and Agriculture*. 22:658-666.

## EFFECTS OF DIETARY AVAILABLE PHOSPHORUS AND PHYTASE (NATUPHOS) ON THE PERFORMANCE OF PIGS FROM 19 TO 40 DAYS POST-WEANING

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Phytase has been reported to increase the availability of P in cereal based diets (Mroz *et al.*, 1994). However, the extent to which phytase might influence pig growth performance independent of its effect of P availability remains unclear. The present experiment which involved 32 pens each of 20 pigs was conducted to investigate the interrelationship between dietary available P (0.15, 0.25, 0.35 and 0.45%) and phytase (0 and 100 g/tonne) on the performance of pigs from 19 (11.1 kg live-weight, 42 days old) to 40 d post-weaning. The enzyme was added to the mixer and the diets were pelleted at 70°C to prevent denaturation of the enzyme. All diets contained 14.8 MJ DE/kg, 1.2% lysine and 1.0% Ca and were offered *ad libitum*. Pigs offered the diets supplemented with phytase exhibited superior performance on all diets and equal or better performance when offered the diet containing 0.25% available P than those offered the unsupplemented diets containing the higher available P contents.

**Table 1.** Effects of dietary available P and phytase on the performance of pigs from 19 to 40 d post-weaning.

Phytase (g/tonne)	Available P (%)	Daily gain (g)	Feed intake (kg/d)	Feed:gain
0	0.15	403	0.64	1.58
	0.25	481	0.72	1.48
	0.35	530	0.80	1.51
	0.45	540	0.82	1.55
100	0.15	472	0.74	1.56
	0.25	540	0.77	1.42
	0.35	629	0.85	1.40
	0.45	595	0.82	1.38
<b>Significance P=</b>				
Phytase		0.004	0.175	0.093
Av. P		0.001	0.133	0.307
Phytase × Av. P		0.939	0.790	0.739

The results suggest that phytase improves growth performance independent of its effects on the availability of P. These findings have important implications because they suggest that feed intake and the overall performance of pigs in the period 19 to 40 days post-weaning may be constrained by phytate-type complexes. However, dietary Ca is known to have adverse effects on the feed intake of growing pigs and the responses observed in the present experiment may have been at least in part as a result of changes in the Ca:P ratio. The interrelationship between Ca, P and phytase on the performance of weaner pigs warrants further research.

### References

MROZ, Z., JONGBLOED, A.W. and KEMME, P.A. (1994). *Journal of Animal Science*. 72:126-132.



## INTERRELATIONSHIPS BETWEEN PROTEIN SOURCE AND BIOFEED PLUS ON THE PERFORMANCE OF WEANED PIGS FROM 23 TO 42 DAYS OF AGE

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Eighty individually housed pigs weaned at 23 days of age (5.73 kg) were used to investigate the effects of supplementing diets, containing 30% soya bean meal or 30% lupin kernels with a carbohydrase mixture of fungal xylanases, pentosanases and beta glucanases (Biofeed Plus) on growth performance to 42 days of age. The two basal diets contained 50% wheat, 14.2 MJ DE/kg and 1.33% lysine. The diets were supplemented with zero and 400 g/tonne Biofeed Plus and pelleted at 70°C to prevent deterioration of enzyme activity. The results (Table 1) showed that pigs offered the diet based on lupin kernels grew faster and had a lower feed:gain than those offered the diet based on soya bean meal. Enzyme supplementation of the diets improved growth rate and tended to improve feed intake and feed:gain. Although there was no significant interaction between the two factors the improvement in growth rate elicited by the enzyme was 9.3 and 21.3% for pigs offered the diet based on lupins and soya bean respectively. The corresponding improvements in feed:gain and feed intake were 4.9 and 2.6% for pigs offered the lupin based diet and 4.7 and 16.6% for pigs offered the soya bean based diet.

**Table 1. Interrelationships between dietary protein source and enzyme supplementation on the performance of newly weaned pigs.**

Protein source	Biofeed Plus (g/t)	Daily gain (g)	Feed:gain	Feed intake (kg/d)
Lupin	0	355	1.21	0.420
	400	388	1.15	0.431
Soya bean	0	291	1.29	0.368
	400	353	1.23	0.429
<b>Significance P=</b>				
Protein source (P)		0.001	0.001	0.116
Biofeed Plus (B)		0.006	0.080	0.059
P × B		0.274	0.788	0.356

The results show that despite its established superiority in terms of nutrient availability for older pigs soya bean meal has a greater inhibitory effect on the voluntary feed intake and performance of newly weaned pigs than lupin kernels. The improvement in performance exhibited by pigs offered the lupin based diet supplemented with Biofeed Plus was associated with an improvement in nutrient availability. In contrast the results for pigs offered the soya bean meal diets suggest the improvement in growth performance elicited by the enzyme was associated more with improvement in rate of passage and hence feed intake than nutrient availability *per se*. The constraints placed on growth performance by both protein sources can be overcome by dietary enzyme supplementation. Enzymes of the type used in the present experiment have the potential to improve the cost effectiveness of diets for weaned pigs by enabling the more expensive and traditional ingredients to be replaced with soya bean meal or lupins.

## EFFECT OF PHYTATE, PHYTASE AND LACTIC ACID ON THE ILEAL AMINO ACID DIGESTIBILITY IN PIGS

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Phytic acid, a strong chelating agent in vegetable based feeds, may form insoluble complexes, with cations and proteins, which are poorly digested by pigs. Phytase (Natuphos®) can hydrolyze the phytate complexes, thus increasing protein and mineral digestibility. Organic acids may improve the efficacy of phytase, by decreasing gut pH. The effect of phytase and lactic acid on the digestibility of diets with 2 levels of phytate was tested.

Six castrates (37-95 kg), fitted with steered ileo-caecal valve cannulae (Mroz *et al.*, 1994), were used in a 2<sup>3</sup> factorial design carried out in a 6 × 6 Latin square, with 4 replications per treatment, plus an extra control group with 1.0 g phosphorus/kg, from mono-calcium phosphate, added to the diet (MCP). The diets were supplemented with phytase, at 0 or 900 FTU/kg (1 FTU liberates 1 µmol of orthophosphate from 1.5 mmol Na-phytate in 1 min at 37°C and pH 5.5); Na-phytate at 0 or 1.5 g P/kg, and lactic acid at 0 or 30 g/kg. A corn-soya bean meal diet (13% crude protein (CP), 0.68% lysine) was fed at 2.3 times maintenance requirement for ME. The digestibility of CP and amino acids (AA) were assessed using Cr<sub>2</sub>O<sub>3</sub> as a marker.

Supplementing the diet with MCP had no effect on CP or AA digestibility. The interactive effect of Na-phytate × phytase and the main effect of lactic acid (no significant interactions) are presented in Table 1.

**Table 1. Apparent ileal digestibility coefficients (%) for crude protein (CP) and some amino acids.**

	Na-phytate - 0		Na-phytate -1.5		SED	Lactic Acid		SED
	Natuphos®		Natuphos®			0	30	
	0	900	0	900				
CP	74.2	75.8	77.1	76.1	0.89	75.4	76.3	0.63
Lysine	77.5 <sup>a</sup>	79.9 <sup>b</sup>	80.4 <sup>b</sup>	79.7 <sup>b</sup>	0.85	78.4	80.3 <sup>**</sup>	0.60
Methionine	80.6	81.7	81.4	80.3	0.83	80.3	81.7 <sup>*</sup>	0.58
Tryptophan	68.3 <sup>a</sup>	72.7 <sup>b</sup>	73.3 <sup>b</sup>	72.7 <sup>b</sup>	1.53	71.2	72.3	1.08
Threonine	68.3 <sup>a</sup>	71.2 <sup>b</sup>	73.0 <sup>b</sup>	70.4 <sup>ab</sup>	1.25	69.8	71.7 <sup>*</sup>	0.89
Isoleucine	77.9 <sup>a</sup>	80.0 <sup>b</sup>	80.9 <sup>b</sup>	79.5 <sup>ab</sup>	0.95	78.8	80.3 <sup>*</sup>	0.67

<sup>ab</sup> Mean values within a row differ at P<0.05. SED = Standard error of difference. Effect of lactic acid <sup>\*</sup>P<0.05; <sup>\*\*</sup>P<0.01.

None of the dietary treatments influenced the digestibility of CP. The addition of dietary phytase alone (without Na-phytate) increased the digestibility of 4 of the 5 AA's. Similar improvements were obtained by the addition of Na-phytate alone (without phytase). The addition of both phytase and Na-phytate marginally, but not significantly, decreased AA digestion compared to Natuphos® or Na-phytate alone, indicating that the phytase preferably hydrolyzed free added Na-phytate rather than the endogenous dietary phytate. Lactic acid alone also increased the digestion of most AA. No evidence was found that lactic acid enhanced the effect of phytase on ileal AA-digestibility.

### References

MROZ, Z., BAKKER, G.C.M., DEKKER, R.A., JONGBLOED, R. and JONGBLOED, A.W. (1994). In "Proceedings of the Vth International Symposium on Digestive Physiology in Pigs", pp. 57-59, eds W.B. Souffrant and H. Hagemester. (EAAP-publication 80: Dummerstorf).

## ZINC OXIDE SUPPLEMENTATION FOR WEANER PIGS

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Zinc oxide (ZnO) has traditionally been added to pig diets for the prevention of zinc deficiency. More recently there have been reports from on-farm studies that adding up to 3 kg ZnO/tonne to weaner diets results in a reduction in *E. coli* induced post-weaning diarrhoea (PWD) and mortality, and an increase in average daily gain (ADG) (Holm, 1990). The aim of this experiment was to study the effect of level and period of ZnO supplementation on the performance of weaner pigs and to monitor zinc residues in liver, kidney, skeletal muscle and pancreatic tissue at slaughter at 90 kg live-weight (LW).

In a 3 × 3 factorial experiment, ZnO (1.5, 3.0 and 4.5 kg/tonne) was fed to Large White × Landrace pigs (8.3 ± 0.11 kg LW) for 7, 14 or 21 d post-weaning, after which time they received the control diet (0.1 kg ZnO/tonne) until 49 days of age. The tenth treatment received the control diet for the duration of the experiment. The control diet was formulated to contain 14.5 MJ DE/kg, 0.85 g available lysine/MJ DE and 1.1% calcium. Forty-four pigs per treatment were housed in groups of 12 to 15. The pigs did not have access to creep food prior to weaning, and they were fed *ad libitum* throughout the experimental period.

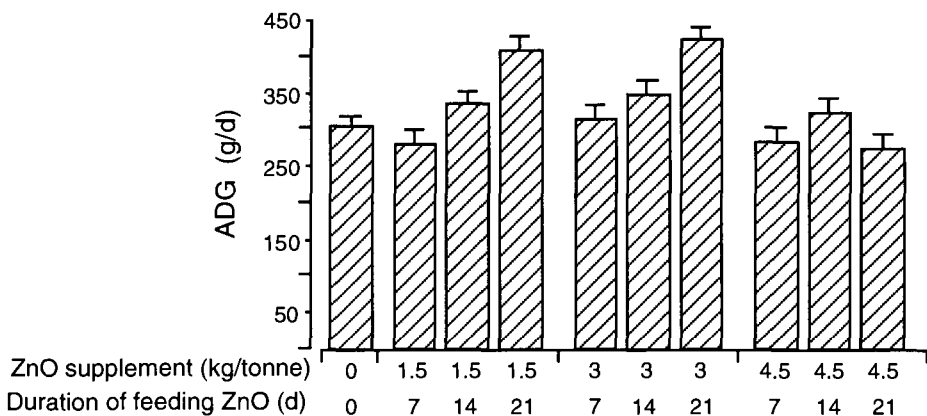


Figure 1. The effect of level and duration of ZnO supplementation on the ADG of pigs post-weaning (21 days).

Post-weaning diarrhoea was not observed during this experiment, therefore the effect of ZnO on PWD could not be determined. There was a significant linear relationship ( $P < 0.001$ ) between ADG and duration of supplementation for piglets fed diets containing either 1.5 or 3.0 kg ZnO/tonne. Compared to the pigs on the control treatment there was a significant ( $P < 0.001$ ) increase in ADG when diets containing either 1.5 or 3.0 kg ZnO/tonne were fed for 21 d. This equated to an increase in LW after 21 d of 2.1 kg (14.9 vs 17.1 kg). There was no response in ADG at the higher level of supplementation. There were no significant treatment effects on the concentrations of Zn, Cu, Fe or Mn in the various tissues at slaughter. Supplementing weaner diets with ZnO up to 3.0 kg/tonne increased ADG, and this was independent of any potential reduction in PWD.

### References

HOLM, A. (1990). *Proceedings of the 11th International Pig Veterinary Society Congress*, Lausanne, Switzerland, p. 154.

## BIOTIN AND PRE-WEANING MORTALITY IN PIGS

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Piglet survival is an important factor in increasing the efficiency of pig production. The majority of pig deaths occur in the pre-weaning period, especially during the first few days after birth. Hartmann *et al.* (1989) noted that depriving pigs of colostrum and milk, with the consequential reduction in the supply of energy and immunoglobulins, were the major factors influencing neonatal piglet mortality. In the sucking pig, colostrum and milk provide 35-50% of the daily requirement for glucose. Endogenous glucose from liver glycogen and gluconeogenesis provides up to 65% of the glucose needs of the neonatal pig.

Biotin has an important role in gluconeogenesis as a coenzyme, and biotin deficiency is a contributing factor in hypoglycaemic deaths in the Fatty Liver Kidney Syndrome of chickens (Whitehead and Blair, 1976). A similar scenario may occur in the neonatal pig, where stress from starvation or cold combined with inadequate biotin could induce hypoglycaemia-associated pre-weaning mortality.

In a series of 4 field trials the effect of biotin supplementation (10 mg/piglet) on piglet mortality was examined. Biotin was administered, either orally or by injection, within 6 or 24 h following birth. Each litter was randomly divided into two groups of equal numbers of pigs. Each group of pigs was treated with either a biotin supplement or a placebo and then returned to the sow. Mortalities were recorded (Table 1) during the first four days post-partum.

**Table 1. Mortalities over the first 4 days post-partum of piglets given biotin (10 mg/pig) or a placebo.**

Trial No.	Biotin Mode	Total No.piglets on trial	Mortality (0-4 d)		Significance
			Placebo	Biotin	
1	Injection	3,453	75	34	P<0.01
2	Oral	2,780	61	67	NS
3	Oral	770	20	10	NS
4	Oral	512	20	16	NS

In the first trial where biotin was administered by injection, biotin reduced mortalities, over the first four days post-partum, by 50% of that in the placebo group. The biotin treated group had 54%, 33%, 73% and 86% fewer mortalities in the overlays, runts, weak/defect and miscellaneous piglet death categories respectively. In the three trials in which biotin was administered orally there appeared to be little effect of biotin on 0-4 d piglet mortalities. These results suggest that the mode of biotin administration may play a role in its efficacy. Further work is necessary to confirm the value of biotin in ameliorating pre-weaning mortality and to optimise the protocol for its effective use.

### References

- HARTMANN, P.E., BIRD, P.H. and HOMES, M. A. (1989). In "Manipulating Pig Production II", pp. 116-121, eds J.L. Barnett and D.P. Hennessy. (Australasian Pig Science Association: Attwood).  
 WHITEHEAD, C.C. and BLAIR, R. (1976). *Research in Veterinary Science*. 21:141-145.

## PERFORMANCE OF WEANER PIGS FED DIETS WITH TWO LEVELS OF ANIMAL PROTEIN AND SIX LEVELS OF CITRIC ACID

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It has been hypothesised that supplementing weaner pigs with organic acids may improve growth performance and feed conversion efficiency (Batterham, 1990). Previous research (eg., Falowski and Aherne, 1984) indicated that acidifying weaner feeds which contain a high proportion of animal based proteins offered little advantage in regard to improved growth rate or feed conversion. Better performances were observed when organic acids were added to diets low in animal proteins (Radecki *et al.*, 1988).

Two experiments were designed to determine the effectiveness of citric acid (CA) supplementation on weaner pig performance. In experiment one (E1), 68% of the total dietary protein (TDP) content was derived from animal protein meals. In experiment two (E2) only 18% of TDP was derived from animal protein meals.

Four hundred and eighty Large White weaner pigs (240 of each gender) were used in each experiment. The pigs were selected at  $28 \pm 0.5$  days and remained in the trial for 28 days. Six diets, containing CA levels of 0, 1, 2, 3, 4 and 5% w/w were used in each experiment. All experimental diets were isoenergetic and isonitrogenous. A randomized complete block design of 20 blocks was used with each block consisting of two male and two females. The mean starting weight of each block in E1 was  $9.39 \pm 0.14$  kg and for E2 was  $8.63 \pm 0.09$  kg. Each experimental block was housed in a pen at a stocking rate of  $0.275 \text{ m}^2/\text{pig}$  and fed *ad-libitum*.

There was a significant linear ( $P < 0.001$ ) decrease for feed intake (FI) in E2 when CA was included in the diet (Table 1). Mean FI in E1 also decreased linearly but was not significant. The response of feed:gain ratio to CA inclusion in both experiments was curvilinear, but was not significant.

Table 1. Influence of citric acid supplementation on individual mean weight gain, feed intake and feed:gain ratio from 28 and 56 days of age.

	Citric acid concentration in feed (%)						SEM
	0%	1%	2%	3%	4%	5%	
<b>Experiment 1</b>							
Weight gain (kg)	8.33	8.96	8.46	7.89	7.88	7.66	0.21
Feed intake (kg)	13.63	13.62	13.29	13.42	12.99	12.85	0.26
Feed:gain ratio	1.77	1.63	1.69	1.89	1.73	1.85	0.06
<b>Experiment 2</b>							
Weight gain (kg)	8.89	9.46	9.27	9.06	8.53	8.16	0.21
Feed intake (kg)	18.04 <sup>a</sup>	17.62 <sup>ba</sup>	15.93 <sup>cb</sup>	16.21 <sup>dbc</sup>	15.40 <sup>ebcd</sup>	14.98 <sup>cde</sup>	0.28
Feed:gain ratio	2.12	1.93	1.78	1.88	1.89	2.04	0.05

Means within a row with different superscripts differ significantly ( $P < 0.05$ )

The high weaning weight observed on this farm was a function of small litter size, while the post-weaning growth rate was restricted by a poor environment. These factors may have confounded the results obtained in this study and could explain the lower than expected responses to citric acid supplementation.

### References

- BATTERHAM, E.S. (1990). In "Pig Rations Assessment and Formulation", pp. 135-140. (Post Graduate Committee in Veterinary Science, University of Sydney).
- FALOWSKI, J.F. and AHERNE, F.X. (1984). *Journal of Animal Science*. 58:935-938.
- RADECKI, S.V., JUHL, M.R. and MILLER, E.R. (1988). *Journal of Animal Science*. 66:2598-2650.

## HIGH AMBIENT TEMPERATURE DECREASES VOLUNTARY FEED INTAKE BUT DOES NOT INCREASE BACKFAT THICKNESS IN ENTIRE MALE FINISHING PIGS

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Pigs grown in Australia are often subjected to high ambient temperatures during the summer months. High temperatures cause voluntary feed intake (VFI) to decrease as the pig attempts to maintain its body temperature and this often results in a decreased growth rate (Giles *et al.*, 1988). High ambient temperatures may also cause backfat thickness to increase as a result of a decrease in the rate of protein accretion relative to fat accretion (Holmes, 1971). Diets high in fibre may exacerbate the problem because hindgut fermentation of fibre may add to the heat load of the pig. This experiment tested the hypotheses that (i) high ambient temperatures will reduce VFI and that the decrease will be larger with pigs fed a high-fibre diet than pigs fed a high-fat diet; and (ii) backfat thickness increases in pigs grown at high ambient temperatures, and that the increase can be reduced by substituting a diet high in fibre with an isoenergetic diet high in fat.

Sixty four entire male finishing pigs were used in a 2 × 2 factorial experiment. The pigs were housed in temperature-controlled rooms in which the temperature was varied in a sinusoidal pattern reaching a maximum at 1500 h and a minimum at 0400 h each day. Two temperature treatments were used; a thermoneutral temperature regime (19-24°C) and a hot temperature regime (25-32°C). Pigs were fed one of two isoenergetic (13.3 MJ DE/kg) and isonitrogenous (16.5% CP) diets; one diet high in fat (5.13% fat and 5.52% fibre) and one diet high in fibre (3.5% fat and 7.68% fibre). The pigs were grown from 50 to 85 kg live-weight at a stocking density of 0.66 m<sup>2</sup> of floor space per pig, and in a relative humidity of between 40 and 50%. Bite drinkers were installed to minimize skin wetness. Feed and water were available *ad libitum*. Live-weight gain, backfat thickness (at the P2 position) by ultrasound, and VFI were measured during the experiment.

**Table 1. Temperature effects on voluntary feed intake (VFI), liveweight gain (LWG) and feed conversion ratio (FCR) of entire male pigs from 50 to 85 kg, and backfat at 85 kg live-weight.**

	Thermoneutral room			Hot room			SEM <sup>1</sup>
	High fibre	High fat	Mean	High fibre	High fat	Mean	
VFI (kg/d)	2.45	2.42	2.43 <sup>a</sup>	2.09	2.06	2.07 <sup>b</sup>	0.071
LWG (g/d)	849	849	849 <sup>a</sup>	756	750	753 <sup>a</sup>	46.3
FCR	2.88	2.85	2.86 <sup>a</sup>	2.76	2.75	2.75 <sup>a</sup>	0.238
Backfat (mm)	12.2	12.6	12.4 <sup>a</sup>	11.7	12.2	11.9 <sup>a</sup>	0.42

<sup>ab</sup> main effect means within a row with different superscripts differ significantly ( $P < 0.05$ ). <sup>1</sup>SEM = pooled standard error between the means.

Voluntary feed intake decreased, as expected, by 17% ( $P < 0.05$ ) in the hot room compared to the thermoneutral room (Table 1). However, reducing the fibre content of the diet by substituting energy as fat did not reduce the depression in VFI, possibly because the difference in fibre content was not large enough to elicit an effect on VFI. The hot temperature regime used in this experiment did not cause an increase in backfat as anticipated. The temperature regime in the hot room provided the pigs with 8 h each day in which the ambient temperature was at or below their evaporative critical temperature (about 26°C), which may have meant that the pigs were not sufficiently heat stressed to cause a change in the relative rates of protein and fat accretion.

### References

- GILES, L.R., BELINDA-DETMANN, E. and LOWE, R.F. (1988). *Animal Production*. 47:467-474.  
 HOLMES, C.W. (1971). *Animal Production*. 13:521-527.

## A PROTOCOL FOR EVALUATING PIG FEEDERS

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There are many feeders currently available on the commercial market. Some piggeries are even making their own. All of these feeders are of different designs with different qualities, many of them claiming to be better than any other. In order to assess the performance of a feeder it is necessary to conduct comparative trials. During a study tour Payne (1991) collected data from comparative trials carried out at several farms. In most instances he found that essential information had not been collected, or that insufficient replicates had been performed. The various test conditions from farm to farm meant that a feeder tested at one site could not be compared to a different feeder tested at a different location. This problem was also identified at a feeder workshop convened by the Pig Research and Development Corporation (1991).

In an effort to address this problem the Pig Feeder Evaluation Test Protocol was developed for testing single space pig feeders. By specifying particular test conditions (Williams and Moore, 1995) it limits the variation of test results across time, thus enabling two different feeders to be tested at different times and still allowing the results to be reliably compared. The protocol also introduces a new measure for the performance of a pig feeder.

The Pig Feeding Efficiency (PFE) was devised as a simple means to express how well the feeder tested gets feed into the pig.

$$\text{PFE} = \frac{\text{actual consumption}}{\text{feed removed from feeder}} \times \frac{\text{actual consumption}}{\text{potential consumption}}$$

The potential consumption is the amount the pigs will eat if there are no restrictions to their eating. Restrictions may be insufficient eating time, inadequate space in the feeder for the pig to put its head in, or some other constraint. For a particular genotype potential consumption can be determined by unrestricted feeding in a single pig trial. The PFE equation can be rewritten as:

$$\text{PFE} = \frac{[\text{FR} - \text{FS}]^2}{\text{FR} \times \text{PI}} \quad \text{where:} \quad \begin{array}{l} \text{FR} = \text{the feed removed from the feeder.} \\ \text{FS} = \text{the feed spilt.} \\ \text{PI} = \text{the potential intake for the group.} \end{array}$$

The amount of spillage was estimated by sweeping the front half of the pen daily and using the total ash content of the sweepings to estimate the amount of feed and manure in the sweepings. The amount of spillage was found to be small and typically was less than 2% of intake.

The PFE embodies two measures of feeder performance, spillage and access restriction, and expresses them as a single number that may be used to compare the performance of feeders in a quick, simple manner.

The results indicate that despite tight control of environmental variables there was variation in the performance of identical feeders at different sites. The PFE ranged from 0.83 to 1.01. At two of three sites with replicate trials the PFE varied by less than 0.02. It was concluded that the Protocol in its present form can be used for comparative trials of feeders at a single site. It could also be used to compare various feeders against a chosen standard feeder; if used in this manner it was recommended that a single research institution be chosen to undertake such trials.

### References

- PAYNE, H. (1991). Study tour report: Single space wet and dry feeders. Pig Research and Development Corporation.  
 PIG RESEARCH and DEVELOPMENT CORPORATION (1991). Design criteria for pig feeding systems.  
 WILLIAMS, R. and MOORE, G. (1995). Pig Research and Development Corporation Final Report, UM64P. Australia.

## DISTURBANCE-FREE PIG WEIGHING

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Electronic measurement of the growth performance of pigs housed under commercial conditions is an important research tool. Several researchers have tested automatic weighing systems. Slader and Gregory (1988) used a large crate mounted on load cells but it also modified the pig's feeding behaviour. Turner *et al.* (1985) used a small platform that only weighed half the pig while it was drinking. The whole weight was determined by a calibration factor, determined by weighing the whole pig on one occasion, but the data produced by that system was not accurate enough for research purposes. This paper reports on a method of automatically weighing pigs.

A portable weighing device (Salter #235/9) was used to study the viability of weighing half-pigs to determine their entire weight. The effect of stance was tested using 23 pigs. It was found that when the pigs' front feet were elevated 145 mm they carried less weight than when both front and rear legs were at the same level. It was also found that if the front legs of a pig were weighed three times in succession that an unacceptably high variation occurred because of differences in stance.

A mesh platform, 300 by 500 mm, placed in front of a single space feeder and supported by two load cells (A&D Mercury LC4103-K060), was used as part of a system to automatically weigh pigs. Individual pigs in the pen were identified by implantable electronic transponders. The effect of stance was further highlighted by the electronic weigh platform. The proportion of the total weight carried by the front legs was about 60%, rather than the 50% found using the manual scales (Williams *et al.*, 1994).

Not every value from the weigh platform represents the weight on the front legs of a single pig. Pigs slept on the platform, stood on it with only one foot, or several pigs may have occupied the space simultaneously. Several methods were used to filter out the spurious weights, but most of them were only useful once the data was collected.

Real time processing was achieved using a Kalman filter, a method of smoothing noisy data from a digital signal. In a typical day more than 100 estimates of pig weight were obtained for each pig and the standard deviation for this data was about 1.5 kg ( $\mu=50$  kg). By choosing suitable values for the filter coefficient and the largest allowable difference between the instantaneous measured weight and the expected value, it was possible to sieve the incoming data and only use reliable values in the estimation of the weights of the pigs.

There are several important considerations to be made when using an electronic platform similar to that used here. Correct interpretation of the weight data is vital when forming calibration equations or serious errors may result. A synchronized time lapse video was used in this work to aid the understanding of data received from the weigh platform. Also of importance is the identification of individual pigs. If the pigs are treated as a group, then the estimated average weight will be biased towards those pigs that make the greatest number of visits. By identifying individuals this problem can be eliminated through estimating the weight of each pig and then taking the mean of the results.

With this system it is still necessary to weigh the pigs manually to provide initial and final weights. However, it is now possible on a daily basis to accurately estimate the whole weight of individual pigs housed as a group, and yet only periodically weigh them manually. This can be of great benefit to those involved in behavioural and/or productivity studies where disruption to the pigs' routine is to be avoided as much as possible.

### References

- SLADER, R.W. and GREGORY, A.M.S. (1988). *Computers and Electronics in Agriculture*. 3:171-175.  
TURNER, M.B.J., BENSON, J.A., HANLEY, M. and HARTWELL, E.S. (1985). National Institute of Agricultural Engineering, Divisional Note DN 1266. United Kingdom.  
WILLIAMS, S.R.O., MOORE, G.A. and CURRIE, E. (1994). AgEng 94 International Conference on Agricultural Engineering, Milano. European Society of Agricultural Engineers.



## CHARACTERIZATION OF PIGGERY ANAEROBIC LAGOONS IN SOUTHERN QUEENSLAND

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The supernatant and sludge characteristics of piggery anaerobic lagoons are dependant on the volume and composition of incoming effluent and the ability of the lagoon to degrade organic material, settle solids and volatilize nitrogen. Limited information is available on the characteristics of effluent stored in piggery lagoons in Australia. Fulhage (1980) and Barth and Kroes (1985) measured average concentrations of Total Kjeldahl Nitrogen (TKN) in the supernatant of piggery lagoons in the USA. They reported a range of average concentrations of TKN for all the lagoons of 285 to 430 mg/L.

Eleven piggeries in Southern Queensland were selected to measure the waste composition of different effluent collection and treatment systems. Supernatant and sludge samples were taken from the lagoons using the techniques described by Barth and Kroes (1985). Representative sump and screen samples were collected from those piggeries that used these collection and treatment systems. Each of the sumps, screened samples and lagoons were analysed for a variety of parameters. Selected parameters [TKN, total phosphorus (TP), potassium (K), electrical conductivity (EC), chloride (Cl), total solids (TS) and volatile solids (VS)] of supernatant samples from the first (anaerobic) lagoon at each piggery are presented in Table 1.

**Table 1. Analyses of supernatant samples from anaerobic lagoons.**

Site	TKN (mg/L)	TP (mg/L)	K (mg/L)	EC (dS/m)	Cl (mg/L)	TS (mg/L)	VS (mg/L)
1	2285	750	21	11.1	706	45400	29000
2	792	275	38	4.4	167	2000	1000
3	1731	132	40	12.4	994	7000	3800
4	544	74	24	4.8	279	2400	1200
5	709	39	14	6.3	325	2200	960
6	633	108	51	6.0	288	3000	1200
7	470	117	83	15.8	2415	13000	2800
8	479	36	6	5.0	492	2400	1200
9	1361	69	30	14.4	1607	8600	3400
10	871	62	31	8.3	743	3840	1600
11	1107	111	28	9.2	697	4960	2360
Mean	998	161	33	8.9	792	8618	4411
± SD	580	206	21	4.0	676	12668	8217

In Table 1 there are large standard deviations (SD) for all the parameters; large variations were also typical for these parameters in the sludge samples. Values of TKN were all greater than those recorded from Fulhage (1980) and Barth and Kroes (1985). The size of the SD of the tabulated parameters verifies that the use of standard or text-book mean concentrations is not always an accurate method of prediction. Rather, physical measurement or accurate predictive tools such as dynamic models are required to forecast lagoon concentrations. These concentrations can then be used to calculate the loading rates of effluent for distribution on land.

### References

- BARTH, C.L. and KROES, J. (1985). In "Agricultural Waste Utilisation and Management", pp. 660-671, Proceedings of the Fifth International Symposium on Livestock Wastes. (ASAE: St Joseph, Michigan).  
 FULHAGE, C.D. (1980). In "Livestock Waste: A Renewable Resource", pp. 225-227, Proceedings of the Fourth International Symposium on Livestock Wastes. (ASAE: St Joseph, Michigan).

## A REVIEW - THE EFFECTS OF ENVIRONMENTAL CONDITIONS INSIDE SWINE HOUSING ON WORKER AND PIG HEALTH

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### Summary

This article reviews the respiratory, physical, and infectious health hazards of the environment on people employed in buildings associated with the intensive pig industry. Similar health responses do occur among workers in the intensive poultry industry and can occur (although less commonly and severely) among workers in other types of confinement operations, eg., beef cattle, dairy cattle, or sheep. Throughout this paper the terms worker, and pig industry employee are frequently used. These terms refer specifically to people employed in intensive or confinement pig production as opposed to those employed in non-intensive (non-confinement) pig production.

In typical modern livestock housing, where animals are densely confined, dusts and gases generated from the operation commonly reach harmful concentrations relative to conventional structures. The sources of these harmful dusts and gases include the animals, their faeces, and their feed. Dusts are more appropriately termed bioaerosols, as the dust is primarily made up of particles of organic origin which are biologically active in their effects. Ammonia ( $\text{NH}_3$ ) comes primarily from the animals' urine and faeces, while hydrogen sulfide ( $\text{H}_2\text{S}$ ) comes from manure pits. When manure pits are agitated in the process of emptying, the concentration of  $\text{H}_2\text{S}$  in confined spaces can rise to lethal levels within seconds; causing sudden death or severe lung injury. Bioaerosol and gas levels are highest in winter, although bioaerosol levels increase whenever animals are fed, handled, moved, or when the building is cleaned with a high pressure sprayer.

Bioaerosols and gases generated in confinement systems can acutely affect exposed persons over a short exposure period. Chronic health effects are common, and typically develop after about six years of exposure. These health problems have forced owners, employees, and veterinarians to stay out of confinement buildings or seek other employment. Adverse responses vary from person to person, may affect any part of the respiratory tract, and may include inflammatory, toxic, or allergic (rarely) processes. Exposure to bioaerosols generated in confinement systems can cause effects to health similar to other agricultural bioaerosols. Health effects include acute or chronic bronchitis (the most common health effect), increased airways reactivity (non-allergic or occupational asthma), a systemic influenza-like response called organic dust toxic syndrome (ODTS), and a general mucus membrane irritation syndrome (MMI), including chronic sinusitis. Research results suggests that chronic obstructive pulmonary disease may occur among confinement workers with long-term exposure, although that has not yet been clearly shown.

When diagnosing and treating respiratory illness in pig industry employees, physicians should make a conscientious attempt to discover links between exposure to bioaerosols and gases in the work environment and the illness. This will avoid the overuse of symptomatic treatment that is ineffective in the long run. Workers must be protected by reducing bioaerosol and gas levels in the piggery buildings through management practices and engineering, and/or by the use of respirators. Pre-employment considerations should include a screening and warning to smokers, and to persons with a history of concurrent respiratory or cardiac conditions, as they tend to have more frequent and more severe problems. Employees should be monitored for development of respiratory disease. Effective prevention must include the integration of 1) medical surveillance, 2) owner-operator, and worker education, and 3) application of industrial hygiene modalities of environmental assessment and control.

In addition, physical injuries appear to be quite important for pig industry workers relative to other livestock production employees. Cuts, lacerations, noise-induced hearing loss and injuries from needles are some of the most common hazards.

There are at least six infectious diseases of pigs to which humans are susceptible. Swine influenza, leptospirosis, brucellosis, erysipeloid, *Streptococcus suis*, and salmonella are all recognized as potential occupational infections of pig industry workers.

### Introduction and Background

Since the 1950s global economic and agricultural policies have driven agricultural enterprises in most western countries to become larger, more intensive, more specialized, more capital intensive (Donham, 1995), and less labour intensive. In livestock and poultry production, systems were developed to raise large numbers of animals in a relatively small space, with relatively little labour (Strange, 1984). These intensive housing systems (also called confinement systems) were first applied to poultry production in the 1950's in the USA. Intensive swine housing began in Europe in the early 1960's, and in North America in the late 1960s and early 1970s. Since that time, similar concepts have been applied in a limited fashion to dairy, as well as beef and sheep production. The use of intensive swine and poultry production facilities have now also begun to appear in developing countries, including Mexico, South America and the Pacific rim countries including Taiwan and the Philippines.

Reports in 1977 and 1982, first indicated there may be some health hazards to persons working in intensive swine housing environments (Donham *et al.*, 1977; Donham and Gustafson, 1982). During recent years, additional research reports from several countries have corroborated these findings. This paper will review and summarize the data from published literature, focusing on exposures, symptoms and pulmonary function studies and comparing these parameters to those of workers exposed to agricultural bioaerosols in other processes. Additionally, environmental analysis and control procedures will be reviewed. Finally, a recommendation for a comprehensive worker protection program will be described.

### Hazardous agents

The bioaerosols in livestock facilities are a complex mixture of potential agents (Table 1) generated primarily from the animals, dried faeces, feed, and saprophytic microbes that grow in the environment (Nilsson, 1984; Donham *et al.*, 1985a; Kiekhaefer *et al.*, 1995). Gases are generated from animal urine and faeces (Donham *et al.*, 1982b; Donham *et al.*, 1985a; Donham *et al.*, 1985b). These bioaerosols and gases accumulate to concentrations that may be hazardous to human and animal health (Donham and Gustafson, 1982).

Each building used to house pigs will contain its own complex mixture and concentration of particles and gases, which is dependent on numerous factors including: Management practices; ventilation of the building; the production stage and species of animal; how they are fed; how their wastes are handled; and generally how well the facility is managed.

Bioaerosol and gas concentrations and composition change within a single building over time relative to the season and the age of the animals. The types of confinement operations and the corresponding bioaerosol and gas exposures are listed in Table 2. This report will concentrate on swine operations, where potentially hazardous bioaerosols and gases and resulting health problems have been studied best. Of the other intensive animal industries, similar responses would occur most commonly among poultry confinement workers (Olenchock *et al.*, 1982; Bar-Sela *et al.*, 1984; Jones *et al.*, 1984; Lenhart, 1984; Thelin *et al.*, 1984; Donham, 1986; Leistikow *et al.*, 1989).

Bioaerosol particles contain approximately 25% protein and range in size from less than 2 to 50 $\mu$ m in diameter (Donham *et al.*, 1985a). One-third of the particles are within the respirable size range (less than 10 $\mu$ m in diameter) (Nilsson, 1984; Donham *et al.*, 1985a). Bioaerosols of faecal origin include high concentrations of gut-flora bacteria and exfoliated gut epithelium. As these particles are quite small relative to other bioaerosol components, they constitute the major burden to small airways and alveoli. The larger

particles are mainly of feed and grain origin, and form the major upper airways burden. Also present are animal dander, broken bits of hair, bacteria, bacterial endotoxins, (1→3)-β-D glucan, pollen grains, insect parts, and fungal spores (Lenhart, 1984; Donham *et al.*, 1985a). The bioaerosol particles can absorb NH<sub>3</sub> and possibly other toxic or irritating gases adding to the potential hazards of the inhaled particles (Donham and Gustafson, 1982; Donham *et al.*, 1985a).

**Table 1. Potentially hazardous agents found in bioaerosols from livestock buildings. Taken from Donham (1989).**

Mould (spores, sporangia, hyphae)
Bacteria
Microbial metabolites/components
Endotoxin
(1→3)-β-D glucan
Mycotoxins (eg., aflatoxin, fumonasin)
Microbial proteases
Tannins
Feed particles, including grain dust, antibiotics, growth promotants
Dried livestock or poultry proteins (urine, dander, serum)
Swine faeces, including gut microbial flora, gut epithelium, undigested feed
Grain mites, insect parts
Mineral ash
Ammonia adsorbed to particles
Pollen
Infectious agents

**Table 2. Relative implications for human health of dust bioaerosols and gases found in various confinement operations.**

Type of confinement operation	Dusts	Ammonia	Hydrogen sulfide <sup>1</sup>
Swine	Major	Moderate	Major
Poultry	Moderate	Major	None <sup>2</sup>
Sheep	Moderate	Moderate	None <sup>2</sup>
Veal	Moderate	Major	Moderate
Dairy	Moderate	Minimal	Moderate
Beef	Minimal	Minimal	Moderate

<sup>1</sup> Following manure agitation. <sup>2</sup> Manure stored in solid form, does not allow for H<sub>2</sub>S production

Toxic, irritating, and asphyxiating gases are continuously generated in the manure pit. If the pit is located under the buildings these gases rise into the air within the building. Of the 40-plus gases generated in anaerobically degenerating manure, hydrogen sulfide (H<sub>2</sub>S), ammonia (NH<sub>3</sub>), carbon dioxide (CO<sub>2</sub>), methane (CH<sub>4</sub>), and carbon monoxide (CO) are the most common potentially hazardous gases present (Donham and Gustafson, 1982; Donham *et al.*, 1982a; Donham *et al.*, 1985a, 1985b). Ammonia is also released into the atmosphere in the building by bacterial action on pig urine and faeces found on the building floor (Donham *et al.*, 1985a). Carbon monoxide and CO<sub>2</sub> are generally not produced in hazardous concentrations from the manure pit. They are produced in much higher concentrations by fossil-fuelled heating systems especially in winter, as well as by the animals' respiration (CO<sub>2</sub> only) (Donham and Gustafson, 1982). These latter gases are usually only hazardous when the heaters or ventilation system

malfunction. Additionally there have been illnesses from CO exposure resulting from operating gasoline engine-powered high pressure sprayers inside these buildings (Anon, 1993). Methane is not a respiratory hazard in these buildings, but occasionally it is a fire or explosive hazard in some buildings (only of concern in buildings with manure stored underneath).

The microbial flora consists of high levels of Gram positive and Gram negative bacteria (mostly of an enteric origin) and yeasts and moulds that are environmental saprophytes (Kiekhaefer *et al.*, 1995). Total organisms range from  $10^4$ - $10^9$  organism/ $m^3$  of air, with Gram positive bacterial species in the majority.

### Populations at risk

A primary risk factor for respiratory illness is related to the length of time the person has worked in these buildings. Those who have worked more than two hours daily and for six or more years are at greatest risk (Donham *et al.*, 1977; Donham and Gustafson, 1982; Donham *et al.*, 1984b; Donham *et al.*, 1989; Donham *et al.*, 1995). Piggery owners and managers, employees and farm family members may work in the pig buildings anywhere from a few hours a week to eight or more hours per day. Women are now commonly employed in these livestock facilities, often working in the farrowing component of the operation. This raises particular concern for risk to pregnant women. For example, CO is occasionally known to exist at levels (50-150 ppm) that could harm human foetuses but may not be acutely toxic to adults. In several areas of the US where larger corporate operations are becoming common, non-traditional labour (from Hispanic and Asian migrant backgrounds) is being employed. This presents language and social barriers which have an impact on health and health care and can complicate other inherent conditions of this socio-economic group such as tuberculosis (Slesinger and Ofstead, 1993). It has been estimated that 300,000 persons in the US are exposed to potentially hazardous environments in intensive pig production operations.

In North America, intensive pig production and resulting health problems are concentrated in the "corn belt" of the Mid-west. However a rapid increase in pig production has been seen in the south-eastern states in recent years, particularly in North Carolina. Newer operations are also found in western Nebraska, Kansas, Oklahoma, Texas, Utah, and Colorado. Intensive poultry production operations in the US are concentrated in the North-east, South-east, Mid-west, and Far-west. Other types of confinement operations are primarily located in the Mid-west's corn belt. In Canada, confinement operations are found in the prairie provinces, as well as the eastern provinces.

Considering an international perspective, intensive pig production in Europe has been present and growing since the 1960's in most northern countries, but particularly so in Denmark, The Netherlands, Sweden, Germany, and the United Kingdom. The former East Germany has had extremely large confinement operations which were initiated on their cooperative farms.

### Situations of work place exposure to bioaerosols and gases

Workers are exposed to high risk atmospheres as they are preparing feed, feeding animals, cleaning the buildings, sorting and moving animals from one building or part of a building to another, and performing routine vaccinations, treatments, or other management and maintenance procedures. The turnover rate of staff employed in intensive pig production can be quite high. Indeed some owners have had to sell their operations because they could not work in their own units, reportedly because of respiratory problems. One survey revealed that over 60% of veterinarians who provide services for these operations report adverse respiratory symptoms (Donham *et al.*, 1977). The severity of their respiratory reactions may force them to stay out of these buildings.

Bioaerosol and gas concentrations increase in winter when the houses are tightly closed and ventilation rates reduced to conserve heat. Also, CO and CO<sub>2</sub> are released from poorly vented or improperly functioning heaters (Donham and Gustafson, 1982). Bioaerosol concentrations also increase when animals are being moved, handled, and fed (Nilsson, 1984). Ventilation systems are designed to control heat and humidity in the

building and often will not reduce bioaerosol or gas levels adequately to ensure a safe environment for either humans or the livestock. During the cold weather, should the ventilation systems fail for several hours, CO<sub>2</sub> from animal respiration, and CO<sub>2</sub> and CO from heaters and manure pits can rise to asphyxiating levels. In the warm weather, the greater risk from ventilation failure is heat stress from high temperature and humidity. Although massive animal losses have been attributed to these latter situations, they are probably not a human health threat.

Hydrogen sulfide from manure pits is most hazardous when the pits are beneath the buildings. However, if gases from outside pits can backflow into a building, an acutely toxic environment may result. Manure pit gases pose an acute hazard when the liquid manure slurry is agitated, an operation commonly performed to suspend solids so that pits can be pumped empty (Osborn and Crapo, 1981; Donham *et al.*, 1982b). During agitation, H<sub>2</sub>S can be released rapidly, soaring from usual ambient levels of less than 5 ppm to lethal levels of over 500 ppm within seconds (Donham *et al.*, 1982b; Donham *et al.*, 1988). Animals and workers have died or become seriously ill when H<sub>2</sub>S has risen during agitation of pits underneath pig buildings. Several workers have died when entering a pit during or soon after the emptying process to repair pumping equipment or clean out solids (Donham *et al.*, 1982b). Persons attempting to rescue these workers also have died. Workers may be exposed to high H<sub>2</sub>S levels when they enter the pit to retrieve animals or tools, or to repair ventilation systems or cracks in the cement.

### Symptoms of exposure to bioaerosols and gases

Two recent reports reviewed 14 different epidemiological studies of workers employed in the intensive pig industry (Rylander *et al.*, 1989; Donham, 1990). Two additional studies have recently been reported (Cormier *et al.*, 1991; Zuskin *et al.*, 1991). Twelve of these studies also included pulmonary function testing. Approximately 3,000 people were studied in these 16 reports. Most of the questionnaires used to assess symptoms were based either on the British Medical Research Council questionnaire, the American Thoracic Society questionnaire, or the organic dust questionnaire (Rylander *et al.*, 1990b). Most of the studies assessed chronic symptoms. Only a few of the studies assessed acute symptoms related to the workplace exposure.

Figure 1 shows a comparison of the major chronic respiratory symptoms among pig industry employees in different countries, including the United States, The Netherlands, Sweden, Canada, France, Denmark, and the United Kingdom (Donham *et al.*, 1977; Donham and Gustafson, 1982; Donham *et al.*, 1984a; Brouwer *et al.*, 1986; Donham *et al.*, 1986; Bongers *et al.*, 1987; Haglind and Rylander, 1987; Holness *et al.*, 1987; Dosman *et al.*, 1988; Cormier *et al.*, 1991; Donham, 1991; Heederik *et al.*, 1991).

The prevalence of chronic symptoms in these populations was from two to four times that found in the reference control populations. Cough and phlegm were the two most prevalent symptoms with cough ranging from 20% to about 55% and phlegm from 12% to about 55% in the population. The extent of these cough and phlegm symptoms was comparable in the US, Sweden, France, the United Kingdom, Denmark, and Canada. In The Netherlands, cough and phlegm were reported about 50% less frequently compared to other countries. However, the prevalence of asthmatic symptoms (wheezing and tightness of chest) in The Netherlands was more similar to the other countries, ranging between 12% and 33%. Distribution of the shortness of breath symptom was similar to that of tightness of chest.

Organic dust toxic syndrome (ODTS) is a condition noted by development of an acute influenza-like illness following exposure to bioaerosols. It is characterized by malaise, muscle aches, fever and headache. The episodes may last from 12-48 hours. (Note: ODTS has also been referred to as toxic alveolitis in recent literature.) The ODT syndrome was studied in the US, Canada, and Sweden and the figures were reasonably comparable, ranging from 10% to 30%. Several studies have noted workers commonly report chronic fatigue, which is an emerging condition being more commonly seen (Donham *et al.*, 1989; Auger, 1992).

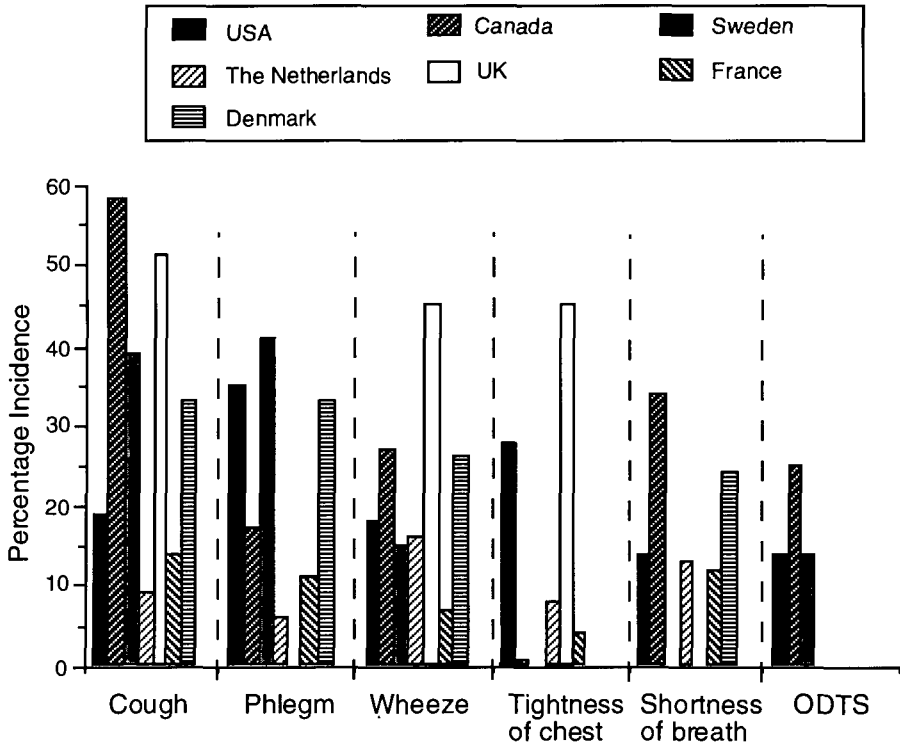


Figure 1. Percentage incidence of various chronic symptoms of respiratory disease found in employees working in the intensive pig industry in various countries. Data collected from various surveys between 1985 and 1993. ODTS = organic dust toxic syndrome. Missing bars represent symptom not reported.

Acute symptoms, defined as those which the worker directly associated with the working environment, were studied in the US, Sweden, Denmark, and The Netherlands (Brouwer *et al.*, 1986; Donham *et al.*, 1990). Table 3 lists the typical prevalence of acute symptoms in these studies. Workers in the US, Sweden and Canada had a similar prevalence of acute cough (38-75%), which was about twice as prevalent as chronic cough. Workers in The Netherlands reported little difference between acute cough (18%) and chronic cough (10-16%).

The prevalence of the acute symptoms of wheeze and tightness of chest was 20-55%, or approximately 1.5 - 2 times that seen for the chronic symptoms. However, in The Netherlands, the prevalence of these acute symptoms was comparable to the chronic symptoms.

The reference, or control, populations used in the studies mentioned above were of two types: 1) farmers not raising pigs or at least not raising pigs in intensive facilities; and 2) workers in a "clean" air environment, e.g., postal workers. The symptoms of those working in the intensive pig industry were at least twice as prevalent as the non-farming controls, and almost 50% higher than pig producers not using confinement facilities.

**Table 3. Prevalence of various acute symptoms of respiratory disease among pig industry workers. Taken from Donham *et al.* (1977); Donham *et al.* (1989); and Iverson *et al.* (1988).**

Symptoms	Prevalence
Cough	67%
Sputum or phlegm	56%
Scratchy throat	54%
Runny nose	45%
Burning or watering eyes	39%
Headaches	37%
Tightness of chest	36%
Shortness of breath	30%
Wheezing	27%
Muscle aches and pains	25%

### Effects of exposure on pulmonary function

The baseline values for pulmonary function in cross-sectional studies of swine confinement workers were generally within normal limits when compared to standard non-farming urban control populations (Table 4). However, studies in the Netherlands revealed small decreases in the forced expiratory volume-in-one-second (FEV<sub>1</sub>), the FEV<sub>1</sub> to forced vital capacity (FVC) ratio, and flow rates (Bongers *et al.*, 1987). There were also decreases in FEV<sub>1</sub>/FVC and flow rates in studies from Sweden and the US, although the FEV<sub>1</sub> was not decreased (Dosman *et al.*, 1988; Donham *et al.*, 1989). Decreases in FEV<sub>1</sub>/FVC ratio and flow rates were also shown in a study of 488 swine producers in Quebec, Canada (Cormier *et al.*, 1991). This picture of decreased ratios and flow rates suggests a pattern of obstructive disease. Studies in the US resulted in evidence of air entrapment in the lungs, further suggesting a risk for chronic obstructive pulmonary disease (Schwartz *et al.*, 1990).

In three studies, the change in pulmonary function during a period of work (work-shift change) was examined in 318 farmers (Donham *et al.*, 1984b; Haglund and Rylander, 1987; Donham *et al.*, 1989; Schwartz *et al.*, 1990). The mean decrease in FVC ranged from 1.2% to 3.3% while the FEV<sub>1</sub> decreased up to 6%. The mean FEV<sub>1</sub>/FVC ratio showed a decrease of 3%. However, the greatest change over the work-shift was in the flow rates, such as the forced expiratory flow, at 25 to 75 percent of lung volume, (FEF 25-75) which decreased by 4 to 12%. Statistically significant work-shift changes in FEV<sub>1</sub> and flow rates were present in both Swedish and US studies.

Although these mean decreases in lung function are modest, there are individuals who have a clinically significant decrease. Two studies, one in Canada and one in The Netherlands (Brouwer *et al.*, 1986; Dosman *et al.*, 1988), calculated the prevalence of lung dysfunction (defined as less than 80% of predicted value, or at least one standard deviation from the mean). The Canadian study showed that 14% of the workers had a clinically significant decrease in FVC. Significant reductions in FEV<sub>1</sub> were also seen but were less notable. Twelve percent of the pig farmers in The Netherlands had a clinically significant decrease in FEV<sub>1</sub> and 20% had reduced flow rates.

A recent study (Malmberg and Larsson, 1992) demonstrated that exposure to bioaerosols found in pig buildings resulted in prolonged broncho-constriction, hyper-responsiveness and increased inflammatory cells in bronchial alveolar lavage (BAL) fluids in five of six naive subjects. Cross-sectional studies of workers in Sweden, the US, and France have shown increased broncho-constriction in methacholine challenge studies compared to controls, and compared to other livestock workers. Further evidence of the broncho-constrictive nature of the bioaerosols found in intensive pig production, was shown by their ability to stimulate contraction of guinea pig tracheal muscle in an *in vitro* model (Zuskin *et al.*, 1991).



**Table 4. Baseline pulmonary function test measurements of the forced vital capacity (FVC); forced expiratory volume-in-one-second (FEV<sub>1</sub>); forced expiratory flow (FEF) among pig industry workers. Data is presented as the percent predicted values.**

	n <sup>1</sup>	FVC	FEV <sub>1</sub>	FEV <sub>1</sub> /FVCx100	FEF
<b>US</b>					
Donham <i>et al.</i> (1984a)	24	102	109	79	112
Donham <i>et al.</i> (1990)	108	108	75	81	80
<b>Sweden</b>					
Haglund and Rylander (1987)	29	95	92	97	nt <sup>2</sup>
Donham <i>et al.</i> (1989)	54	92	100	78	60
<b>Canada</b>					
Holness <i>et al.</i> (1987)	5	98	95	97	64
Controls	?	95	94	99	72
Dosman <i>et al.</i> (1988)	50	97	96	98	87
Cormier <i>et al.</i> (1991) <sup>3</sup>	488	4.48	3.37	0.75	3.14
Controls <sup>3</sup>	216	4.51	3.41	0.75	3.17

<sup>1</sup> n = number of persons; ? = number unknown. <sup>2</sup> nt = measurements not taken).

<sup>3</sup> Results reported in absolute volumes (litres)

In summary, studies of baseline pulmonary function have shown only a small average decrease in pulmonary volumes, as well as FEV<sub>1</sub>/FVC. Flow rates, however, were generally significantly lower than controls. Work-shift reductions in FEV<sub>1</sub> and FVC were seen in several studies. The major change was in decreased flow rates over the work-shift (work-shift = period of time a person is working on a particular day). Although it is generally recognized that there is a problem with variability in measurement of flow rates, these parameters were consistently lower in all work-shift and baseline studies and therefore, are likely to be real changes. Clinically significant suppressions (Donham and Gustafson, 1982; Lenhart, 1984; Dosman *et al.*, 1988) were seen in 12%-14% of individual workers, primarily in flow rates, volumes and capacity. Other studies have provided evidence of air trapping in the lungs. These pulmonary function studies, although performed on healthy subjects, suggest that early phases of obstructive lung disease were present

### Allergy studies

There have been seven different studies (summarized in Table 5) that assessed allergic conditions in pig industry employees (Katila *et al.*, 1981; Harries and Cromwell, 1982; Matson *et al.*, 1983; Brouwer *et al.*, 1986; Donham *et al.*, 1989; Cormier *et al.*, 1991; Crook *et al.*, 1991; Zuskin *et al.*, 1991). Five of these studies showed evidence of increased IgG antibodies to antigens that were isolated from the environment, including both animal allergens and mould spores. Four of the studies showed a low incidence of IgE response. A skin-prick-test was performed in three studies, and in one study a challenge test was done.

The results of these immunologic studies showed that IgG antibodies to environmental allergens were frequently seen, while IgE antibody responses were less common than IgG. In the studies with controls, no differences in antibody levels between pig industry workers and controls were found. In the one study that examined the relationship between the presence of IgG antibodies to environmental allergens and symptoms of respiratory disease no correlation could be found. However, in one Dutch study (Brouwer *et al.*, 1986), a relationship was seen between IgE antibodies and the number of hours worked per week in the buildings. A second study (Zuskin *et al.*, 1991), showed an increased broncho-constriction over the work-shift in three workers who also had IgE antibodies to dust from the pig sheds. However, none of these studies have shown a statistical significant causative association between IgE or IgG antibodies and symptoms of respiratory disease. In the studies where skin-prick-tests were done, there

was no relationship between a positive skin test and a disease condition. One case study in the US showed that a challenge of pig urine resulted in development of asthma in one patient. No other challenge studies could be found in the literature.

**Table 5. Antibody (IgG and IgE), skin-prick-test (SPT) and other test responses in pig industry workers and in control populations found in the various allergy studies and the causal relationship of the immune response to disease conditions. (+ = increased levels; NR = not reported).**

	IgG	IgE	SPT	Other	Controls	Disease relation
Katila <i>et al.</i> (1981)	+	neg	NR	NR	+	None
Harries and Cromwell (1982)				challenge	NR	Asthma
Matson <i>et al.</i> (1983)	+	+	+	NR	+	None
Donham <i>et al.</i> (1989)	+	NR	NR	NR	+	None
Brouwer <i>et al.</i> (1986)	+	+	NR	NR	NR	Hours/week <sup>1</sup>
Heederik <i>et al.</i> (1991)	+	NR	NR	NR	NR	None
Donham <i>et al.</i> (1990)	NR	NR	+	NR	+	None
Cormier <i>et al.</i> (1991)	NR	+	+	NR	+	None
Zuskin <i>et al.</i> (1991)	NR	+	+	NR	+	Increased broncho-constriction

<sup>1</sup>Antibody response related to number of hours worked in swine building per week

Methacholine challenge studies have shown that hyper-responsive airway disease is common in workers exposed to high bioaerosol concentrations (Rylander *et al.*, 1990a). Also, the response to challenge was quite sustained in length of time. However, there was no relationship of this hyper-responsiveness to atopy or positive skin-prick-test (not mediated by an allergic condition).

A relationship between antibody or skin-test response to disease was generally not seen. The antibody response appears to be more indicative of exposure, but not related to disease or symptoms. It is apparent that classical Type 1 allergic mediated asthma is only a minor part of the disease picture in the healthy pig industry workers. The primary mechanism of injury appears to be inflation and/or direct tissue damage, rather than classical allergy.

### Exposure response studies

Smoking and total exposure time have shown the most consistent relationship to symptomatology, as well as pulmonary dysfunction. Studies by Matson (Matson *et al.*, 1983) and Donham (Donham and Gustafson, 1982; Donham *et al.*, 1986) point out that there is an increased relative risk of 1.5 - 2.0 for coughing and wheezing for confinement workers who smoke. However, for the symptoms of phlegm, shortness of breath, and work absence the risk was not increased for workers who smoked.

Table 6 summarizes the effects of smoking and working in the intensive pig industry on pulmonary function testing. Regarding baseline pulmonary function, smokers generally had lower values for FVC (reduced by 0-7%). The FEV<sub>1</sub> for smokers was 4% to 20% lower compared to non-smokers. Pulmonary flow rates were approximately 5% lower in smokers compared to non-smokers.

Regarding the decline in pulmonary function during a work-shift, workers who smoke had from one to two times greater work-shift decrease in FVC compared to non-smokers. This same decrement was seen also in FEV<sub>1</sub>, as well as in the flow rates.

Exposure-response studies also included an assessment of the response to endotoxin, bioaerosol, ammonia and microbes. Of these hazardous substances airborne endotoxin concentrations had the strongest and most consistent relationship to symptoms of ODS and work-shift decreases in pulmonary function test (PFT) (Donham *et al.*, 1989). A significant relationship was seen between bioaerosol concentration and

bronchitic symptoms (cough and phlegm). A weaker relationship of bioaerosol concentrations to tightness of chest and febrile syndromes was found. There was no relationship of bioaerosol to pulmonary function changes. Ammonia did show some relationship to decreased baseline pulmonary function in three different studies (Donham *et al.*, 1989; Donham *et al.*, 1995; Reynolds *et al.*, 1995). In one of the studies, the levels of microbes showed a significant relationship to symptoms of hyper-reactive airways (Donham *et al.*, 1989).

**Table 6. Effects of smoking on baseline pulmonary function test measurements of the forced vital capacity (FVC); forced expiratory volume-in-one-second (FEV<sub>1</sub>); forced expiratory flow (FEF) among pig industry workers. Taken from Matson *et al.* (1983); Donham and Gustafson (1982) and Donham *et al.* (1986).**

Baseline measurement	Non-smokers compared to smokers %
FVC	0 to 7% lower
FEV <sub>1</sub>	4% to 20% lower
FEF	5% lower

A study in The Netherlands (Heederik *et al.*, 1991) suggested that both endotoxin and Gram negative bacteria were related to reductions in FEV<sub>1</sub> and FVC. Also, significant relationships were shown between symptoms of bronchitis, or ODTS to endotoxin or Gram negative bacteria exposure.

### Exposure limit studies

There is little scientific doubt that disease symptoms and work-shift declines in pulmonary function are related to several components of the bioaerosols and gases found in buildings housing pigs under intensive conditions. These components include dust, endotoxin, and ammonia. However, the most important question in this regard is how much exposure creates a health hazard? Knowledge of the appropriate exposure limits is extremely important for controlling the work environment.

Data which suggests the exposure limits in relation to adverse pulmonary function and symptoms is found in three dose-response studies. The first is a Swedish study of 54 pig industry workers (Donham *et al.*, 1989). Several significant correlations were found between health parameters and contaminants measured at stationary sites in the building, but not with contaminants measured by samplers affixed to workers.

The symptoms which were compared to exposures were primarily those suggestive of chronic airways inflammation including, bronchitis and reactive airways disease. Also commonly reported are symptoms of chronic muscle aches, fatigue, and dyspnoea. Significant increases in these symptoms are associated with exposure to concentrations higher than the following: 1) total bioaerosol 1.5 - 5 mg/m<sup>3</sup>; 2) respirable bioaerosol 0.1 - 0.3 mg/m<sup>3</sup>; 3) microbes 10<sup>3</sup> - 10<sup>7</sup> per m<sup>3</sup>; and 4) endotoxin 0.05 - 0.15 µgm/m<sup>3</sup>. Table 7 lists the recommended maximum levels of environmental exposures based on the three studies.

More recent data analyses from a US study have corroborated the previous exposure limit study (Donham *et al.*, 1995; Reynolds *et al.*, 1995). A longitudinal study of 208 swine farmers (randomly selected from a stratified sample of all pig producers in Iowa) resulted in consistent evidence of exposure to the dust and gases found in pig buildings and decreased pulmonary function. Furthermore, multiple regression analyses of the data, provided results consistent with the Swedish study previously mentioned, eg., ammonia (7 ppm) and total bioaerosols (2.5 mg/m<sup>3</sup>) (Donham *et al.*, 1995; Reynolds *et al.*, 1995). A follow-up study of the 208 US swine farmers substantiated the exposure limit recommendations from the first study.

### Pathology of bioaerosol related respiratory disease

The respiratory pathology of workers exposed to high concentrations of bioaerosols and gases are primarily located in the airways. Pulmonary function testing often reveals lowered flow rates relative to controls populations, and evidence of air trapping in the lungs (evidence of airways obstruction). Pulmonary function tests over a work-period commonly reveal moderate decreases in FEV<sub>1</sub> (evidence of reactive airways disease, or occupational asthma), flow rates, and possibly FVC. A general increase in white cells in BAL fluids is seen in these workers, with a predominance of neutrophils in the acutely exposed person, and of lymphocytes in the chronically exposed. Endotracheal examination often reveals an inflamed mucosal surface of the trachea and bronchi. Although no histological studies have been done on human airways, chronic exposure studies in animals have revealed necrosis and metaplasia of the respiratory epithelium; a diffuse mononuclear infiltration into the lung parenchyma; and a chronic pleuritis (Donham and Leininger, 1984). Acute exposure studies in animals revealed a neutrophilia of BAL fluid and histologically, a neutrophilic infiltration of the airway mucosa and sub-mucosa.

**Table 7. Human and pig exposure thresholds for various bioaerosol components found in swine buildings. Exposure to concentrations of contaminants in excess of values given are associated with a higher proportion of ill-health in workers, and with disease, or lower production parameters in pigs. Taken from Donham *et al.* (1989)<sup>1</sup>, Donham *et al.* (1995)<sup>1</sup> and Donham (1991)<sup>2</sup>.**

Bioaerosol component	Human health <sup>1</sup>	Swine health <sup>2</sup>
Total dust mg/m <sup>3</sup>	2.4	3.7
Respirable dust mg/m <sup>3</sup>	0.23	0.23
Endotoxin g/m <sup>3</sup>	0.08	0.15
Carbon dioxide (ppm)	1,540	1,540
Ammonia (ppm)	7.0	11.0
Total microbes cfu/m <sup>3</sup>	4.3×10 <sup>5</sup>	4.3×10 <sup>5</sup>

### Pathogenesis of bioaerosol related respiratory disease

The pathogenesis of bioaerosol related respiratory damage is most likely that of chronic inflammation, at least in regards to the outcome of bronchitis, and bronchial hyper-responsiveness (Donham *et al.*, 1990; Choudat *et al.*, 1994; Rylander, 1994). Allergic illnesses mediated via IgE and IgG are apparently very rare events in the pathogenesis of these conditions. Although the effects of organic dust exposure among workers in the intensive pig industry may be complicated by ammonia and hydrogen sulfide exposure, the primary chronic effect seems to be that of organic dust.

As mentioned in the section on pathology, the primary respiratory tissues affected are the airways. There are two interactive tissue systems affected that result in bronchitis and the airway reactivity observed; namely 1) the airway epithelium, and 2) the macrophage-mediator complex.

Turner and Nichols (1995) have presented an in-depth overview of the pathogenesis of the respiratory epithelium in airways disease. When the airway epithelium, which is normally a barrier, is damaged by chronic exposures it allows greater access to, and effects of, air contaminants on the underlying structures. This results in stimulation of nerve endings causing smooth muscle constriction and goblet cell hyper-secretion - thus narrowing the airway lumen. The epithelial cells normally secrete a variety of cytokines and mediators that effect the normal function of the secretory cells and smooth muscle of the mucosal and sub-mucosal layers. Damaged epithelial cells secrete abnormal quantities of cytokines, which attract neutrophils. Neutrophils release lysozymes and free oxygen radicals which are tissue damaging in themselves. They also attract macrophages

which further release a series of mediators and substances cascading into tissue inflammation and further tissue damage. Results include increased but less viscous mucus and serous secretions, smooth muscle constriction, and cilia disruption. The results are a narrowing of the bronchiolar lumen, and decreased clearance. The symptoms are cough, phlegm, wheezing, and tightness of chest.

Although most of the symptoms involve the airways, the lung parenchyma may also be affected. When inflammatory substances such as endotoxin and (1→3)- $\beta$ -D glucan enter the alveoli, neutrophils are attracted to the area, followed by macrophages as described above. Macrophages are activated resulting in the inflammation cascade. The endothelial cells become damaged, resulting in leaky alveolar capillaries. Additionally, infiltrates arrive at the inter-cellular space creating partial blockage of gas exchange through the alveoli. Symptoms may include difficult breathing, fatigue, or may progress to fever and influenza-like symptoms (systemic effects of ODTS).

The condition of ODTS is also referred to as toxic pneumonitis because of evidence of associated inflammation of the alveoli. Organic dust toxic syndrome is a systemic manifestation of an acute inflammatory response to a sudden high-level exposure.

The outcome or end-result for workers exposed to hazardous piggery environments are not well known, as few prospective studies or studies of severely affected individuals have been done. However, one 6-year follow-up in Canada revealed about 15% of one group of swine producers had dropped out of farming because of respiratory disease (Holness *et al.*, 1987).

#### Animal health; relevance to human health

The buildings used for intensive animal production may also represent health hazards for the animals. Occasionally pigs die suddenly from acute hydrogen sulfide, CO<sub>2</sub> asphyxiation or CO poisoning. Chronic exposure to excessive bioaerosols and ammonia are related to increased rates of pneumonia and lowered productivity (Donham, 1989). Monitoring animal health could principally serve as a model or sentinel for human health effects as well as leading to improvements in the health and productivity of the pig herd.

Relationships between swine health and environmental exposures have been found between exposure to dust, endotoxin, and ammonia, and an increased prevalence of pneumonia and pleuritis (Donham, 1991). Also, relationships between the concentration of these agents and slower growth rate, feed efficiency, and increased mortality were reported in the same study.

Other reports have shown decreased bacterial clearance and increased upper respiratory infections (Donham, 1991). The results from one study (Donham and Gustafson, 1982) suggest strong positive correlations between development of lung infection and atrophy of the nasal turbinates and environmental exposure to dust, ammonia, and microbes. Laboratory animals placed in these units developed chronic pneumonia and chronic degenerative airways inflammation (Donham and Leininger, 1984).

#### Physical injury hazards in intensive pig production

Respiratory disease appears to be the most important health problem for people employed in intensive pig production (Rylander *et al.*, 1989). However, physical agents also appear hazardous. Randolph and Rhodes (1993) studied one operation with 176 employees, and found an injury rate amongst employees of 65 percent per year. This compared to 9.6 and 33.4% in the dairy and meat packing industries. The most common injuries in the pig industry were bruises, sprains, and stabs with needles (45% of total injuries).

Noise was also a hazard in these facilities. Noise levels in farrowing and gestation rooms can reach 103 decibels. The US Occupational Safety and Health Administration's (OSHA) threshold limit for noise is 85 decibels. Therefore, there is good evidence for risk of noise induced hearing loss (Donham, 1995).

## Infectious disease risks in intensive pig production

Several infectious diseases of swine are known to be infectious for humans (Donham, 1985). These organisms include: 1) swine influenza virus, 2) leptospira, 3) brucellosis, 4) *Erysipelothrix rhusiopathiae*, 5) *Streptococcus suis*, and 6) salmonella. The prevalence of these diseases among pork producers is not known. These diseases are difficult to diagnose, and conditions are usually treated symptomatically without diagnoses. Also, these diseases may cause morbidity, but rarely mortality. The exception may be *Streptococcus suis*, where brain abscess and death have been reported from this organism in the United Kingdom (Stanford and Ross, 1986).

### Summary of health effects

The research regarding occupational health of working in the intensive pig industry has identified an increased risk for physical injuries, noise induced hearing loss, infectious disease, and respiratory illness. Respiratory illness has received the bulk of research attention. Summarizing the results of previous epidemiological studies of respiratory disease is difficult because of variations in use of survey instruments, controls, and pulmonary function study protocols. Of the fourteen studies examined, only seven included comparison (control) populations, and in one instance the comparison population was extremely small. The following conclusions are made with recognition of the current limitations of the data.

The various symptoms seen in workers can be grouped and then classified according to a probable disease condition. Some of the most apparent generalizations that can be made include (Donham *et al.*, 1984b; Malmberg *et al.*, 1985; Iverson *et al.*, 1988; Donham *et al.*, 1989; Malmberg and Larsson, 1992):

- 1) Symptoms of upper and lower airways inflammation are common, manifesting themselves as acute or chronic bronchitis (cough and phlegm).
- 2) Symptoms of airway hyper-reactivity (non-allergic or occupational asthma) are also common.
- 3) Mucous membrane irritation has also been noted, characterized by rhinitis, pharyngitis, and sinusitis.
- 4) Symptoms of ODDS are seen in approximately 25% of workers.
- 5) Symptoms of chronic fatigue, muscle aches and pain are also commonly reported.

The generalized symptoms (ODDS) also present in workers are noted by episodes of delayed onset of fever, malaise, muscle aches, and headaches are commonly reported (Malmberg *et al.*, 1985). Other symptoms include chronic fatigue, chronic muscle aches and pains.

The symptoms reported by workers in the pig industry are grouped and summarized in Table 8. When considered this way, there is circumstantial evidence that several conditions may be ascribed to exposure to hazardous environments found in many buildings used for intensive pig production. Several of the conditions may occur simultaneously in the same workers.

Because pulmonary function tests often demonstrate only marginal decreases in the mean values, symptoms may be a better parameter for which to study the relationship between respiratory disease and the working environment. In future studies, there is a need to use a standardized questionnaire that assesses the types of symptoms seen after exposure to organic bioaerosols. It is also necessary that standard PFT protocols and representative control populations be included in future studies.

### Diagnosis of respiratory disease resulting from exposure to bioaerosols

The use of diagnostic aids for occupational respiratory conditions is of secondary importance to a detailed clinical and occupational history. It is important to recognize that a patient's response to bioaerosols and gases is variable, and that one or more conditions may be occurring simultaneously (eg., chronic bronchitis, occupational asthma

and sinusitis). Patients should be questioned in detail about their most serious complaints, including questions on how long symptoms have been present and the time relationship of symptoms to exposure in the work-place. Exposure to bioaerosols and gases in the work-place for more than two hours per day, and six or more years total exposure are related to increased frequency and severity of symptoms. Smoking exacerbates the severity and frequency of symptoms. An improvement in the symptoms over a vacation, and the development of worse than normal symptoms upon return to work are indicators the hazardous nature of the workplace environment.

**Table 8. Summary of the symptoms reported by swine confinement workers and the suggested disease conditions.**

Symptoms	Suggested disease conditions
Sudden unconsciousness Respiratory failure Pulmonary oedema	Acute hydrogen sulfide exposure
Cough Phlegm Tightness of chest	Airway inflammation or bronchitis
Wheeze Tightness of chest Shortness of breath	Occupational asthma or hyper-reactive airway disease
Acute episodes of fever Malaise Muscle and joint pain Headache Fatigue	Organic dust toxic syndrome (ODTS)
Chronic fatigue Muscle and joint pain Shortness of breath	Chronic inflammation or chronic ODTS
Irritation of throat, eyes and nose	Mucous membrane irritation (Nasopharyngitis, Conjunctivitis)
Dizziness "Popping" ears Continuous or frequent colds Stuffy nose	Mucous membrane inflammation (Sinusitis)
Chest tightness on return to work	Byssinosis-like reaction

Patients should be questioned on the specific job they do and on the environment in the building. Moving and sorting animals and power-washing the building inside are jobs where the patient is exposed to much higher bioaerosol concentrations. Use of a proper respirator that has a good seal around the nose and mouth may decrease the symptoms. The investigating clinician should take an in-depth personal and family medical history, including questions on allergies, asthma, heart conditions and hobbies or personal habits (such as smoking) that might complicate the issue. They should ask how many hours per day or week the patient works in the confinement buildings? How long the patient has held this job? What conditions prevail within the building? Does the patient recognize the building as particularly bad? Have environmental assessments been done? Pulmonary function tests may also be useful. Lowered pulmonary flow rates are common and, while baseline values may be normal, decreases of 5% or more in lung volumes over the work-period are common in affected workers. A decreased tolerance to methacholine challenge is also common. Skin-prick-tests for suspected feed or swine allergens are usually negative.

Without a proper environmental history, the physician may fail to relate the patient's symptoms to exposure to a hazardous atmosphere. In addition, misdiagnosis and subsequent treatment of confinement-related respiratory conditions as allergic responses are common; such treatment may provide symptomatic relief through bronchodilators and inhaled steroids, but is non-specific and probably ineffective in the long run, unless the patient is protected from further environmental exposures.

### **Treatment of respiratory disease**

Medically, there are few drugs that may be prescribed for the treatment of chronic respiratory symptoms as described above. However, some of the acute illnesses (eg., asthma, pulmonary oedema from H<sub>2</sub>S intoxication) may be more amenable to drug therapy. Bronchitis and reactive airways disease may respond, temporarily, to inhaled antihistamines, broncho-dilators, and corticosteroids. However, these do not address the underlying causes of these problems.

Respiratory conditions must be controlled through protecting the patient from the environment. First, by reducing bioaerosol and gas levels and second, by the appropriate use of respirators, and/or temporary removal from the work site. In almost all cases, with appropriate use of these modalities, workers can continue to work safely. In order to reduce bioaerosols and gases, a patient may need to contact a consulting veterinarian or agricultural engineer who has knowledge of environmental control. The local veterinarian or the Cooperative Extension Service agricultural engineer should be able to recommend an appropriate expert.

Physicians may need to address the patient's anxiety as well as the patient's medical problems. Workers often are told to quit working in intensive pig production if they are having respiratory problems. Usually this recommendation is unnecessary and should only be given once the cause and prognosis of illness have been determined and other avenues of controlling the harmful exposures have been fully explored. In many instances, the farmer has no reasonable alternative-occupational choice other than to continue working in the pig industry. Such a recommendation may produce extreme mental stress since there may be no other reasonable occupational alternative, and quitting farming is leaving a life-style as well as a job.

Farmers in the intensive or confinement pig industry are becoming increasingly aware of work-place associated respiratory conditions. A physician who can explain the potential relationships of environmental exposures and respiratory conditions will instill confidence in the patient regarding maintenance of his/her health status. This will also assist in facilitating the protection of the farmer, their family and employees from health problems associated with the work environment. Monitoring the patient's respiratory status may be reassuring to many patients. An initial examination should include a thorough occupational history, spirometry, and a chest x-ray if patients are symptomatic. These can be repeated if clinically indicated at later annual check-ups.

### **Prevention of workplace related respiratory disease**

Health hazards associated with intensive pig production must be addressed by improvements in the environment. This will be achieved mainly by 1) decreased generation of bioaerosols and gases through improved management practices; 2) removal of contaminants once in the air, eg., ventilation or electrostatic precipitation and; 3) protection of the individual by proper use of respirators (Mutel *et al.*, 1992).

A model prevention program for respiratory problems in the pig industry, based on education and industrial hygiene consultation, has demonstrated its effectiveness (Donham *et al.*, 1990; Mutel *et al.*, 1992). This program is now available for distribution to help extension workers or other groups initiate prevention programs in their region. Some examples of management practices to reduce or eliminate the sources of bioaerosols and gases include; 1) delivering feed from the conveyor system by extension spouts into covered feeders, rather than letting the feed fall freely several feet into open feeders; 2) frequently (eg., every three weeks) and systematically washing the inside of buildings with power-sprayers to keep them as clean as possible (note a respirator should be worn during this high exposure task); 3) using wire-mesh floors which are more self-cleaning



compared to concrete slats and; 4) assuring that heating units are clean, vented, and functioning properly. Details of this control program have been published elsewhere (Donham *et al.*, 1990; Mutel *et al.*, 1992). The effectiveness of control techniques can be assessed by measuring bioaerosol and gas concentrations. Buildings should be routinely monitored to assure air contaminants are within safe levels.

Because it is impossible to completely eliminate the formation of bioaerosols and gases, techniques for removing contaminants from the air are critically important. Ventilation systems are usually engineered on parameters of controlling moisture and temperature in the buildings, not hazardous substances. Ventilation can however reduce gases, but not necessarily bioaerosols, to safe levels. Ventilation systems must be properly designed and maintained, and ventilation rates adjusted to include consideration of air quality. Air quality must be monitored to assure the concentrations of contaminants remains at safe levels. Operators often keep ventilation rates low in winter because of concerns for conserving heat, this may cause bioaerosol and gas concentrations to rise. A number of engineering techniques (eg., use of heat exchangers which allow increased ventilation while capturing some waste heat) have been tried with varying degrees of success.

Anyone working in a swine or poultry confinement operation would be wise to wear an approved dust mask, unless they are assured that the concentrations of bioaerosols and gases are below recommended limits. Persons exposed to building with high bioaerosol or gas concentrations, or persons with respiratory conditions, may need to use a more sophisticated respirator, such as a half-mask cartridge respirator or air-helmet.

Preventing exposure to high concentrations of H<sub>2</sub>S from manure pits requires stringent controls. General safety measures include constructing manure pits outside of the building, constructing openings so that lids or other objects cannot fall into the pit requiring a worker to enter the pit for retrieval, and erecting safety guards and warning signs. Whenever a pit which is under a building is being agitated, people should stay out of the building, ventilation inside the building should be maximized, and the animals should be removed or observed from outside the building.

Even when not being agitated, manure pits can seldom be entered safely. If entrance is imperative, only a self-contained breathing apparatus, worn by an individual trained in its use, will provide adequate protection. All operators should understand that high concentrations of H<sub>2</sub>S cannot be smelled and that H<sub>2</sub>S above 1000 ppm can produce unconsciousness in only one to three breaths. A variety of H<sub>2</sub>S gas alarms give an accurate indication of hazard.

Poor air quality in the building has also been shown to be associated with health problems in swine, as well as lowered productivity (Donham, 1989). Promoting improvement of air quality may be the most expedient way to create environmental improvement for the workers, according to this author's experience in North America and Northern Europe.

### Summary

With the advent of intensive swine production systems, over 16 studies in eight countries have documented an increased risk for respiratory disease in workers. These conditions primarily involve the airways (bronchitis, occupational asthma), and an episodic generalized condition called ODS. Additionally, episodic instances of acute hydrogen sulfide poisoning causing death occur.

Environmental control programs are necessary to help prevent occupational illness in workers and promote swine production at the maximum of their genetic potential. Although further research is needed in defining threshold concentrations of contaminants, practical and accessible monitoring systems, and comprehensive occupational health programs, there is sufficient information at the present time to begin initiation of control programs in swine production systems.

The data presented in this review suggest that poor air quality inside swine buildings is an important factor relative to the health and welfare of those working in these buildings. Medical expenses, disability and occasional deaths add significantly to the economic and social costs of production. Similarly, poor air quality is related to

lowered productivity and health loss in swine. This has a further impact on the economic and animal welfare costs of production.

Air quality issues have received relatively little attention by researchers and the industry. Research and implementation of environmental control measures for both human and animal health has significant potential to improve the sustainability of the swine industry.

## References

- Anon (1993). Unintentional Carbon Monoxide Poisoning from Indoor Use of Pressure Washers - Iowa, January 1992 - January 1993. *Morbidity and Mortality Weekly Report*. Centers for Disease Control, USPHS. 42:777-779. (The Massachusetts Medical Society: Waltham).
- AUGER, P. (1992). Clinical experience with patients suffering from a chronic fatigue-like syndrome and repetitious upper respiratory infections in relation to airborne moulds. Presentation at Skokloster 3 Workshop, Skokloster, Sweden, April 6-9.
- BAR-SELA, S., TEICHTAHL, H., and LUTSKY, I. (1984). Occupational asthma in poultry workers. *Journal of Allergy and Clinical Immunology* 73:271-275.
- BONGERS, P., HOUTHUIJS, D., REMIJN, B., BROUWER, R., and BIERSTEKER, K. (1987). Lung function and respiratory symptoms in pig farmers. *British Journal of Industrial Medicine*. 44:819-823.
- BROUWER, R., BIERSTEKER, K., BONGERS, P., REMIJN, B., and HOUTHUIJS, D. (1986). Respiratory symptoms, lung function and IgG<sub>4</sub> levels against pig antigens in a sample of Dutch pig farmers. *American Journal of Industrial Medicine*. 10:283-285.
- CHOUDAT, D., GOEHEN, M., KOROBAEFF, M., BOULET, A., DEWITTE, J.D., and MARTIN, M.H. (1994). Respiratory symptoms and bronchial reactivity among pig and dairy farmers. *Scandinavian Journal of Work Environment and Health*. 20:48-54.
- CORMIER, Y., BOULET, L., BEDARD, G., and TREMBLAY, G. (1991). Respiratory health of workers exposed to swine confinement buildings and dairy barns. *Scandinavian Journal of Work Environment and Health*. 17:269-275.
- CROOK, B., ROBERTSON, J., GLASS, S.A., BOTHEROYD, E.M., LACEY, J., and TOPPING, M.D. (1991). Airborne dust, ammonia, microorganisms, and antigens in pig confinement houses, and the respiratory health of exposed farm workers. *American Industrial Hygiene Association Journal*. 52(7):271-279.
- DONHAM, K.J. (1985). Zoonotic diseases of occupational significance: a review. *International Journal of Zoonoses*. 12:163-191.
- DONHAM, K.J. (1986). Hazardous agents in agricultural dusts and methods of evaluation. *American Journal of Industrial Medicine*. 10:205-220.
- DONHAM, K.J. (1989). Relationships of air quality and productivity in intensive swine housing. *Agri-Practice*. 10:15-26.
- DONHAM, K.J. (1990). Health effects from work in swine confinement buildings. *American Journal of Industrial Medicine*. 17:17-25.
- DONHAM, K.J. (1991). Association of environmental contaminants with disease and productivity in swine. *American Journal of Veterinary Research*. 52:1723-1730.
- DONHAM, K.J. (1995). Agricultural medicine and environmental health: the missing component of the sustainable agricultural movement. In "Agricultural Health and Safety: Workplace, Environment, Sustainability" (Lewis Publishers: Boca Raton). In press.
- DONHAM, K.J., CARSON, T.L., and ADRIAN, B.R. (1982a). Carboxyhemoglobin values in swine relative to carbon monoxide exposure: guidelines to monitor for animal and human health hazards in swine buildings. *American Journal of Veterinary Research*. 5:813-816.
- DONHAM, K.J., and GUSTAFSON, K.E. (1982). Human occupational hazards from swine confinement. *Annals of American Conference on Governmental Industrial Hygiene*. 2:137-142.
- DONHAM, K.J., HAGLIND, P., PETERSON, Y., RYLANDER, R., and BELIN, L. (1986). Environmental and health studies in swine confinement buildings. *American Journal of Industrial Medicine*. 10:289-294.
- DONHAM, K.J., 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.
- DONHAM, K.J., KNAPP, L.W., MONSON, R., and GUSTAFSON, K.E. (1982b). Acute toxic exposure to gases from liquid manure. *Journal of Occupational Medicine*. 24:142-145.
- DONHAM, K.J., and LEININGER, J.R. (1984). The use of laboratory animals to study potential chronic lung disease in swine confinement workers. *American Journal of Veterinary Research*. 45:926-931.
- DONHAM, K.J., MERCHANT, J.A., LASSISE, D., POPENDORF, W., and BURMEISTER, L. (1990). Preventing respiratory disease in swine confinement workers: Intervention through applied epidemiology, education, and consultation. *American Journal of Industrial Medicine*. 18:241-262.
- DONHAM, K.J., REYNOLDS, S.J., WHITTEN, P., MERCHANT, J.A., BURMEISTER, L.F., and POPENDORF, W.J. (1995). Respiratory dysfunction in swine production workers: Dose-response relationship of environmental exposures and pulmonary function. *American Journal of Industrial Medicine*. 27:405-418.
- DONHAM, K.J., RUBINO, M.J., THEDELL, T.D., and KAMMERMEYER, J. (1977). Potential health hazards of workers in swine confinement buildings. *Journal of Occupational Medicine*. 19:383-387.
- DONHAM, K.J., SCALLON, L., POPENDORF, W.J., TRUEHAFT, M., and ROBERTS, R. (1985a). Characterization of dusts collected from swine confinement buildings. *American Industrial Hygiene Association Journal*. 46:658-661.
- DONHAM, K.J., YEGGY, J., and DAGUE, R. (1985b). Chemical and physical parameters of liquid manure from swine confinement facilities: health implications for workers, swine, and the environment. *Agricultural Wastes*. 14:97-113.
- DONHAM, K.J., YEGGY, J., and DAGUE, R. (1988). Production rates of toxic gases from liquid manure: health implications for workers and animals in swine buildings. *Biological Wastes*. 24:161-173.

- DONHAM, K.J., ZAVALA, D.C., and MERCHANT, J.A. (1984a). Acute effects of the work environment on pulmonary functions of swine confinement workers. *American Journal of Industrial Medicine*. 5:367-376.
- DONHAM, K.J., ZAVALA, D.C., and MERCHANT, J.A. (1984b). Respiratory symptoms and lung function among workers in swine confinement buildings: A cross-sectional epidemiological study. *Archives of Environmental Health*. 39:96-100.
- DOSMAN, J.A., GRAHM, B.L., HALL, D., PAHWA, P., MCDUFFICE, H., LUCEWICZ, M., and TO, T. (1988). Respiratory symptoms and alterations in pulmonary function tests in swine producers in Saskatchewan: results of a survey of farmers. *Journal of Occupational Medicine*. 30:715-720.
- HAGLIND, P., and RYLANDER, R. (1987). Occupational exposure and lung function measurements among workers in swine confinement buildings. *Journal of Occupational Medicine*. 29:904-907.
- HARRIES, M.G., and CROMWELL, O. (1982). Occupational allergy caused by allergy to pig's urine. *British Medical Journal*. 284:867.
- HEEDERIK, D., BROUWER, R., BIERSTEKER, K., and BOLEIJ, J. (1991). Relationships of airborne endotoxin and bacteria levels in pig farms with lung function and respiratory symptoms of farmers. *International Archives of Occupational and Environmental Health*. 62:595-601.
- HOLNESS, D.L., O'GLENIS, E.L., SASS-KORTSAK, A., PILGER, C., and NETHERCOTT, J. (1987). Respiratory effects and dust exposures in hog confinement farming. *American Journal of Industrial Medicine*. 11:571-580.
- IVERSON, M., DAHL, R., KORSGAARD, J., HALLAS, T., and JENSEN, E. (1988). Respiratory symptoms in Danish farmers: an epidemiological study of risk factors. *Thorax*. 48:872-877.
- JONES, W., MORRING, K., OLENCHOCK, S., WILLIAMS, T., and HICKEY, J. (1984). Environmental study of poultry confinement buildings. *American Industrial Hygiene Association Journal*. 45:760-766.
- KATILA, M., MÄNTYJARVI, R.A., and OJANEN, T. (1981). Sensitization against environmental antigens and respiratory symptoms in swine workers. *British Journal of Industrial Medicine*. 38:334-338.
- KIEKHAFFER, M., DONHAM, K., WHITTEN, P., and THORNE, P. (1995). Cross-sectional studies of airborne microbial studies in swine buildings: implications for worker and animal health. *Annals of Agricultural and Environmental Medicine*. 2:1-8.
- LEISTIKOW, B., PETTIT, W., DONHAM, K., MERCHANT, J., and POPENDORF, W. (1989). Respiratory risks in poultry handling. In "Principles of Health and Safety in Agriculture", pp. 62-65, eds J.A. Dosman and C.O. Cockroft. (CRC Press: Boca Raton).
- LENHART, S.W. (1984). Sources of respiratory insult in the poultry processing industry. *American Journal of Industrial Medicine*. 6:89-96.
- MALMBERG, P., and LARSSON, K. (1992). Acute exposure to swine dust causes bronchial hyperresponsiveness. Presentation at Skokloster 3 Workshop, Skokloster, Sweden, April 6-9.
- MALMBERG, P., RASK-ANDERSON, A., PALMGREN, U., and HÖGLUND, S. (1985). Exposure to microorganism, febrile airway obstructive symptoms, immune status and lung function of Swedish farmers. *Scandinavian Journal of Work Environment and Health*. 11:287-293.
- MATSON, S.C., SWANSON, M.C., REED, C.E., and YUNGINGER, J.W. (1983). IgE and IgE-immune mechanisms do not mediate occupational-related respiratory or systemic symptoms in hog farmers. *Journal of Allergy and Clinical Immunology*. 72:299-304.
- MUTEL, C., DONHAM, K.J., FERGUSON, K., GJERDE, C., and HRADEK, C. (1992). "Swine Confinement and Respiratory Health: A Guide to the Prevention of Job-related Health Problems for Swine Producers Who Work in Confinement Facilities", 2nd edn, (The University of Iowa Institute of Agricultural Medicine and Occupational Health: Iowa City).
- NILSSON, C. (1984). Dust investigations in pig houses. In "Proceedings of the International Society of Animal Hygiene", pp. 31-37, ed. H.G. Hillinger. (University of Hannover: Hannover).
- OLENCHOCK, S.A., LENHART, S.W., and MULL, J.C. (1982). Occupational exposure to airborne endotoxins during poultry processing. *Journal of Toxicology and Environmental Health*. 9:339-349.
- OSBERN, L.N., and CRAPO, R.O. (1981). Dung lung: a report of toxic exposure to liquid manure. *Annals of Internal Medicine*. 95:312-314.
- RANDOLPH, G., and RHODES, S. (1993). Injuries to workers in a swine confinement facility. *Journal of Occupational Medicine*. 35:518-529.
- REYNOLDS, S.J., DONHAM, K.J., WHITTEN, P., and MERCHANT, J.A. (1995). A longitudinal evaluation of dose-response relationships for environmental exposures and pulmonary function in swine production workers. *American Journal of Industrial Medicine*. In press.
- RYLANDER, R. (1994). Symptoms and mechanisms of inflammation of the lung. *American Journal of Industrial Medicine*. 25:19-24.
- RYLANDER, R., DONHAM, K.J., HJORT, C., BROUWER, R., and HEEDERIK, D. (1989). Effects of exposure to dust in swine confinement buildings: A working group report. *Scandinavian Journal of Work Environment and Health*. 15:309-312.
- RYLANDER, R., ESSELE, N., and DONHAM, K.J. (1990a). Bronchial hyperreactivity among pig and dairy farmers. *American Journal of Industrial Medicine*. 17:66-69.
- RYLANDER, R., PETERSON, Y., and DONHAM, K.J. (1990b). Questionnaire evaluating organic dust exposure. *American Journal of Industrial Medicine*. 17:121-126.
- SCHWARTZ, D.A., DONHAM, K.J., POPENDORF, W.J., LASSISE, D.L., HUNNINGHAKE, G.W., and MERCHANT, J.A. (1990). Are work shift changes in lung function predictive of underlying lung disease? Abstract in *American Review of Respiratory Disease*.
- SLESINGER, D.P., and OFSTEAD, C. (1993). Economic and health needs of Wisconsin migrant farm workers. *Journal of Rural Health*. 9:138-148.
- STANFORD, S.E., and ROSS, R.F. (1986). Streptococcal diseases. In "Diseases of Swine", 6th edn, pp. 607-608, eds A. Leman, B. Straw, R. Glock, W.L. Mengeling, R. Penny and E. Scholl. (Iowa State University Press: Ames).
- STRANGE, M. (1984). "It's not All Sunshine and Fresh Air: Chronic Health Effects of Modern Farming Practices", (Center for Rural Affairs: Walthill).

- THELIN, A., TEGLER, O., and RYLANDER, R. (1984). Lung reactions during poultry handling related to dust and bacterial endotoxin levels. *European Journal of Respiratory Diseases*. 65:266-291.
- TURNER, F., and NICHOLS, P.J. (1995). Role of the epithelium in the response of the airways. Abstract for 19th Cotton and other Organic Dust Research Conference, San Antonio, Texas, January 6-7.
- ZUSKIN, E., KANCELJAK, B., SCHLACHTER, E., MUSTAJBEGOVIC, J., GOSWAMI, S., MAAYANI, S., MAROM, Z., and RIENZI, N. (1991). Immunological and respiratory findings in swine farmers. *Environmental Research*. 56:120-130.

## THE AUSTRALIAN PIG INDUSTRY TAKES THE LEAD IN OCCUPATIONAL HEALTH AND SAFETY DEVELOPMENT

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Occupational health and safety is a significant issue in respect to future profitability of the Australian pig industry. Industry personnel, are exposed to hazards that have the potential to affect human health. The impact of injury and illness and its associated cost, must be considered a priority for producers in planning complete farm management strategies. Recent research data provides an initial insight into the cost and number of injuries being incurred on piggeries across Australia. These costs are a feature of pig production and through effective management they can be reduced.

The survey by Ferguson (1994), found that pig producers had an acute injury and illness rate over one year of 36.9 per 100 farms. Estimates from the study place the cost of acute injury and illness for the pig industry at \$187,504 per 100 farms or approximately nine million dollars per year if applied to the entire Australian pig industry. Although these estimates must be viewed with caution because of high relative standard errors, they do show the magnitude of the situation. Injuries are borne as direct and indirect costs by way of medical treatment, replacement labour, lost work time and reduction of animal productivity. Recent increases in workers compensation premiums for many Australian producers also exemplify the cost of injury in the industry. This trend for rising premium levels is expected to continue over the medium term.

These figures represent only part of the total cost that workplace injury and illness inflict on the Australian pig industry. Besides the cost of acute injuries, there are potential long term health problems. Chronic health conditions include noise induced hearing loss and respiratory illness. Donham and Leininger (1984), suggested that a latent period of 15 to 20 years for occupational lung diseases, could be masking a potential long term problem of chronic lung damage in American piggery workers. Similar problems have been developing over time in the Australian pig industry but the extent of the health and economic implications are yet to be identified. Evaluation of these issues will continue over the next 10 to 15 years. However, at present it is known that productivity of workers will be influenced by exposure to hazards in the piggery environment. The extent of zoonotic disease is also uncertain because of poor notification procedures, and unfamiliarity or underdiagnosis of the symptoms by the medical profession (Robertson and Davies, 1989).

In response, the Pig Research and Development Corporation have funded the Queensland Farmers' Federation to address these problems through the development of a management package as a component of total farm management. The package will consist of information highlighting the producers' responsibility; information leaflets detailing the problems identified by producers who attended focus group meetings and, a safety audit, which can be carried out to improve the standard of farm safety and give recognition to those areas where adequate controls are in place or are needed. The pig industry has been the first commodity group to specifically address workplace injury and illness. As such, this program is a benchmark for rural Australia. Industry personnel are being given the opportunity to gain an understanding of the package, through workshops being run in all States. The emphasis of these workshops will be on development of farm safety as part of staff training and total quality management programs.

All personnel working in the industry have a legislative responsibility to perform their work safely. Employers can aid in this process by providing a safe workplace and taking responsibility for farm safety development. If producers do not accept the responsibility of managing these issues Government intervention is inevitable.

### References

- DONHAM, K.J. and LEININGER, J.R. (1984). *American Journal of Veterinary Research*. 45:926-931.  
FERGUSON, K. (1994). *Farm Survey of Workplace Injury/Illness Factors to Support Activity Planning of Six Farm Safe Action Groups. Final Report. Division of Workplace Health and Safety. Queensland.*  
ROBERTSON, I.D. and DAVIES, P.R. (1989). *Medical Journal of Australia*. 151:238.

## AIR QUALITY IN WEANER ACCOMMODATION

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Poor air quality is recognised as a major risk factor in the development of respiratory disease in pigs (Donham, 1991). Important parameters of air quality include the volume of air-space per pig; concentration of respirable dust and the concentration of bacteria, especially streptococcal organisms (Skirrow *et al.*, 1995).

Comparisons of air quality were made between two types of weaner housing. These were either rooms, where pigs were housed in pens inside a mechanically ventilated room; or kennels, where pigs were housed in box-type structures with natural ventilation and outside runs. Kennels on 10 farms and rooms on 18 farms were compared.

Farms were visited four times at three monthly intervals. On each occasion the total and respirable dust levels were measured over a three hour period and bacteria concentrations in air samples were measured as the number of colony forming unit units (CFU), using an Anderson sampler loaded with horse blood agar and selective media for *Streptococcus* sp. Gas concentrations were measured three times during each visit using standard gas tubes (Kitagawa). The number of coughs and sneezes/100 pigs were recorded over three two minute periods at each visit.

**Table 1. Stocking rates and concentrations of dust, gas and bacteria in air samples collected in weaner rooms and weaner kennels (mean  $\pm$  SD).**

Parameters	Weaner rooms	Kennels
Volume (m <sup>3</sup> /pig)	0.86 $\pm$ 0.352	0.13 $\pm$ 0.043
Floor area (m <sup>2</sup> /pig)	0.21 $\pm$ 0.043	0.19 $\pm$ 0.084
Bacteria (CFU $\times$ 10 <sup>3</sup> /m <sup>3</sup> )	139 $\pm$ 54	67 $\pm$ 28
<i>Streptococcus</i> sp. (CFU/m <sup>3</sup> )	14,000 $\pm$ 11,320	5,171 $\pm$ 2,118
Total dust (mg/m <sup>3</sup> )	2.78 $\pm$ 3.046	6.32 $\pm$ 6.22
Respirable dust (mg/m <sup>3</sup> )	0.27 $\pm$ 0.28	0.45 $\pm$ 0.54
Ammonia (ppm)	4.51 $\pm$ 3.2	1.85 $\pm$ 2.34
Carbon dioxide (ppm)	1,749 $\pm$ 943	2,033 $\pm$ 1,003
Humidity (%)	63.03 $\pm$ 10.05	59.81 $\pm$ 12
Coughs/100 pigs/min	0.91 $\pm$ 2.05	1.00 $\pm$ 3.86
Sneezes/100 pigs/min	11.94 $\pm$ 11.49	9.17 $\pm$ 18.73

Mean concentrations of total and respirable dust in both rooms and kennels and the mean value for total bacteria in rooms were above target levels recommended to industry (Pointon *et al.*, 1995). The concentration of streptococci was 2.7 times greater in rooms than kennels. Other parameters were within recommended ranges (Pointon *et al.*, 1995).

Analysis of variance of the data indicated that coughing in pigs tended to be associated with higher levels of respirable dust ( $P < 0.05$ ) while sneezing tended to be associated with total dust ( $P < 0.01$ ), reflecting their respective ability to penetrate the various levels of the respiratory system.

The concentrations of respirable dust and streptococcal organisms reported in this study indicate that the air quality in many weaner rooms and kennels is sub-optimal and could contribute to an increased prevalence of respiratory disease in growing pigs.

### References

- DONHAM, K.J. (1991). *American Journal Veterinary Research*. 52:1723-1730.  
 SKIRROW, S.Z., CARGILL, C., NICHOLLS, R.R., MASTERMAN, N. and BANHAZI, T. (1995). *Proceedings Australian Association Pig Veterinarians Conference*, Melbourne, pp. 47-52.  
 POINTON, A., CARGILL, C. and SLADE, J. (1995). In "Good Health Manual for Pigs", p. 140. (Pig Research and Development Corporation: Canberra, ACT).

## EFFECTS OF PELLETING FEED ON AEROSOLS IN PIG SHEDS

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Dust and bacterial contamination of the air-space within pig sheds is recognised as a health hazard for both pigs and humans (Done, 1991; Donham, 1991) and can increase the prevalence of respiratory disease in pigs (Skirrow *et al.*, 1995). Dust is also reported as providing a vector for the maintenance of bacterial aerosols in air-spaces and the transport of bacteria into the respiratory passages (Mossfer and Fung, 1990).

Concentrations of dust and bacteria were measured in weaner and grower accommodation on 26 farms in which pigs were fed either pelleted or unpelleted diets. Weaners were housed in either kennels (eight farms) or rooms (18 farms) and growers were housed in naturally ventilated sheds. All diets contained between 2 to 3% tallow. Farms were visited four times at three-monthly intervals. Total and respirable dust were measured as mg/m<sup>3</sup> of air for periods of 3 h using either an IOM or a cyclone attachment respectively. Numbers of bacteria and fungi were measured as colony forming units per cubic metre of air (CFU/m<sup>3</sup>), using an Anderson sampler (for periods of 5 min) loaded with plates containing horse blood agar (HBA) for total count, HBA with naladixic acid for *Streptococcus* sp. and Sabouraud agar for fungi.

**Table 1. Mean concentrations of airborne bacteria, fungi and dust in weaner and grower facilities in which pigs received either pelleted or unpelleted feed.**

	Grower diets		Weaner diets	
	Pelleted	Unpelleted	Pelleted	Unpelleted
Bacteria (CFU/m <sup>3</sup> )	1.78 × 10 <sup>5</sup>	1.37 × 10 <sup>5</sup>	1.21 × 10 <sup>5</sup>	1.17 × 10 <sup>5</sup>
<i>Streptococcus</i> sp. (CFU/m <sup>3</sup> )	1.35 × 10 <sup>4</sup>	1.12 × 10 <sup>4</sup>	1.20 × 10 <sup>4</sup>	1.19 × 10 <sup>4</sup>
Fungi (CFU/m <sup>3</sup> )	1.36 × 10 <sup>4</sup>	1.36 × 10 <sup>4</sup>	1.13 × 10 <sup>4</sup>	1.16 × 10 <sup>4</sup>
Total dust (mg/m <sup>3</sup> )	1.85	1.61	4.61	2.70
Respirable dust (mg/m <sup>3</sup> )	0.24	0.17	0.412	0.194

Levels of respirable dust were significantly higher ( $P < 0.01$ ) in the weaner and grower facilities on farms where pelleted feed was used compared with those on farms where feed was not pelleted. Although total dust levels were significantly higher ( $P < 0.01$ ) in weaner facilities where pelleted feed was used, no differences were found between grower sheds using either type of feed. The amount of total dust recorded in weaner accommodation on farms where pelleted feed was used was much higher in those with kennels than rooms ( $8.72 \pm 2.26$  mg/m<sup>3</sup> vs  $2.49 \pm 1.57$  mg/m<sup>3</sup>;  $P < 0.01$ ; mean  $\pm$  SD). Also, kennels on these farms had significantly higher amounts of dust than kennels on farms where feed was not pelleted ( $8.72 \pm 6.92$  mg/m<sup>3</sup> vs  $2.82 \pm 2.26$  mg/m<sup>3</sup>;  $P < 0.01$ ). Total dust levels in weaner rooms were similar irrespective of the type of diet used. Although humidity levels were similar in kennels and rooms, the floors of all weaner rooms had wet areas whereas kennel floors were dry.

The results conflict with traditional advice which has recommended pelleting as a means of reducing dust levels. In previous studies higher dust levels have been associated with higher stocking densities (Done, 1991). While this was true for the population of grower pigs in the present study ( $P < 0.05$ ) it was not true for weaners. It could also be that a significant percentage of pellets, which usually contain more finely ground material (R. Williams, personal communication), had been damaged during delivery and feeding.

## References

- DONE, S.H. (1991). *Veterinary Record*. **128**:582-586.  
 DONHAM, K.J. (1991). *American Journal of Veterinary Research*. **52**:1723-1730.  
 SKIRROW, S.Z., CARGILL, C., NICHOLLS, R.R., MASTERMAN, N. and BANHAZI, T. (1995). *Proceedings of the Australian Association of Pig Veterinarians Programme, Australian Veterinary Association Conference, Melbourne 1995*, pp. 47-52.  
 MOSSFER, A. and FUNG, D.Y.C. (1990). *Critical Reviews in Food Science and Nutrition*. **29**:333-340.

## PHYSIOLOGICAL CHANGES IN GROWING PIGS ASSOCIATED WITH PLEUROPNEUMONIA CHALLENGE

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Physiological changes responsible for poor growth in pigs affected with respiratory disease are poorly understood. This study examined the associations among plasma cortisol, body temperature (BT), oxygen consumption ( $VO_2$ ) and voluntary feed intake (VFI) in pigs challenged with *Actinobacillus pleuropneumoniae* serotype 1 (App).

Five specific-pathogen-free PIC-hybrid gilts (mean live-weight, 70 kg) were fed *ad libitum* a diet containing 12.6 MJ DE/kg and housed at 22°C in individual metabolism crates under constant light. Each pig was surgically prepared for the measurement of  $VO_2$  (Giles *et al.*, 1995). The recording of BT, VFI and  $VO_2$ , and venous blood sampling began 2 weeks after surgery and continued for 7 d. One day after recording began, the pigs were sedated with ketamine/xylazine and intra-bronchially inoculated (right lung) with either 5 ml of saline (control group, n=3) or 5 ml of media containing either  $10^4$  (pig 1) or  $10^5$  (pig 2) colony forming units of App. Seven days post-inoculation, right lung damage (LD) was calculated from image analysis of serial lung slices.

Control pigs had zero LD. Pigs 1 and 2 had 30% and 2% LD respectively, despite pig 2 receiving the higher dose. All pigs ate up to 300 g/h prior to inoculation. An increase in VFI (up to 900 g/h) occurred after inoculation, in association with sedation, and then declined to zero for 40 h in App-infected pigs. Control pigs resumed pre-inoculation VFI after 12 h. There was a sharp rise in BT in App-treated pigs 6 h post-inoculation (Figure 1), while there was no change in control pigs. After inoculation,  $VO_2$  in pig 2 increased above control values for 18 h and then declined in association with the period of inappetence. Plasma cortisol rose in all pigs in response to sedation and was followed by marked increases in pigs 1 and 2 as a result of infection. The more rapid decline in cortisol, BT and  $VO_2$  in pig 2 post-inoculation suggests a more effective adaptation to App infection. This is consistent with the lower LD, and may have practical significance for reducing morbidity associated with respiratory disease.

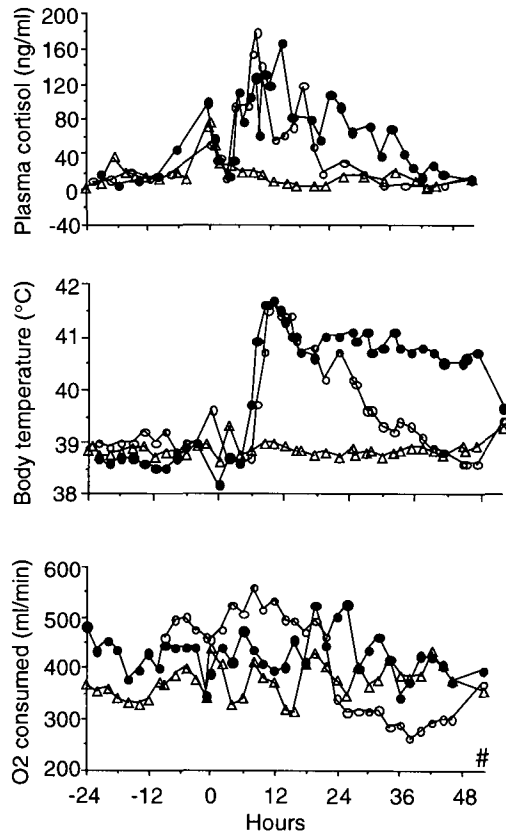


Figure 1. Plasma cortisol levels, body temperature and oxygen ( $O_2$ ) consumed in pigs challenged with *A. pleuropneumoniae* (saline  $\Delta$ - $\Delta$  (n=3); pig 1  $\bullet$ - $\bullet$ ; pig 2  $\circ$ - $\circ$ ). Inoculation occurred at 0 h. (#, Mean for day 6).

### References

GILES, L.R., ANNISON, E.F., BLACK, J.L., TUCKER, R.G. and GOODEN, J.M. (1995). *Journal of Agricultural Science, Cambridge* 124:113-118.



## GENETIC AND ANTIGENIC STUDIES ON *HAEMOPHILUS PARASUIS*

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*Haemophilus parasuis*, an organism dependent upon nicotinamide adenine dinucleotide (NAD) or V-factor for *in-vitro* growth, is the causative agent of porcine polyserositis and arthritis (Glässer's disease) (Nicolet, 1992). The principal lesions associated with this disease are fibrinous or serofibrinous meningitis, serositis, pleuritis, pericarditis, peritonitis and arthritis that can occur in various combinations or occasionally singly (Nicolet, 1992).

A total of 41 isolates of *H. parasuis* obtained from Australian pigs were serotyped by the Kielstein-Rapp-Gabrielson scheme (Kielstein and Rapp-Gabrielson, 1992). The isolates were assigned to the following serovars: serovar 1 (1 isolate); serovar 2 (3 isolates); serovar 4 (5 isolates); serovar 5 (13 isolates); serovar 9 (2 isolates); serovar 12 (1 isolate) and serovar 13 (7 isolates). Of the remaining nine isolates, four cross-reacted with serovars 7 and 10 while five could not be assigned to a serovar. Two different serovars (5 and 13) were detected in one herd. The only two isolates obtained from clinically normal pigs (from the same herd) were serovar 9. The common serovars were isolated from pigs with pneumonia as well as from pigs with clinical signs indicative of Glässer's disease. The serological heterogeneity amongst Australian isolates of *H. parasuis* has important implications for the use of vaccines to control Glässer's disease. Inactivated vaccines are effective only against those serovars present in the vaccine. As it has been established that eight different serovars of *H. parasuis* exist in Australia, therefore the choice of vaccine strains is clearly a major issue.

The genetic diversity among 40 of the Australian isolates and eight reference strains from overseas was assessed by the use of multi-locus enzyme electrophoresis. Thirty-four electrophoretic types (ET) were recognised with a mean genetic diversity per locus of 0.405. One ET was separated by a considerable distance from the rest of the isolates, suggesting that this organism may belong in a different species. The remaining 33 ET formed two divisions (A and B) which were quite distinct from each other as the genetic distance between the divisions was 0.506. Within Division A, five subgroups (I to V) were recognised. All 12 Australian serovar 5 isolates, plus the only two reference strain for this serovar, were included in Division A. The only other serovars present in Division A were Australian isolates of serovars 4 and 13. Within Division B, the four subgroups (I to IV) recognised contained a diverse range of serovars - Australian isolates of serovars 1, 2, 9 and 13 as well as the reference strains for serovars 1, 3, 4, 8 and 9.

These results support the suggestion of other studies based on DNA hybridisation that serovar 5 isolates of *H. parasuis* form a subspecies (Moruzumi *et al.*, 1986). However, the results also suggest that it is not just serovar 5 isolates that form this subspecies - Australian isolates of serovars 4 and 13 were also in the same genetic subdivision.

It has been suggested that serovar 5 isolates are predominantly associated with outbreaks of septicaemia or polyserositis (Nicolet, 1992). Further work is needed to determine if there is any link between pathogenicity and the genetic subdivisions that have been recognised in *H. parasuis*.

### References

- KIELSTEIN, P. and RAPP-GABRIELSON, V.J. (1992). *Journal of Clinical Microbiology*. 30:862-865.  
MOROZUMI, T., PAULI, U., BRAUN, R. and NICOLET, J. (1986). *International Journal of Systematic Bacteriology*. 36:17-19.  
NICOLET, J. (1992). In "Diseases of Swine" 7th edn, pp. 526-528, eds A.D. Leman, B.E. Straw, W.L. Mengeling, S. D'Allaire and D.J. Taylor. (Iowa State University Press: Ames).

## IMMUNOMAGNETIC CAPTURE POLYMERASE CHAIN REACTION FOR DETECTION OF *MYCOPLASMA HYOPNEUMONIAE*

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*Mycoplasma hyopneumoniae* is the major etiological agent of porcine enzootic pneumonia, the most significant respiratory disease afflicting commercial piggeries worldwide. This organism is extremely difficult to culture from affected lung, and although sensitive and pathogen-specific ELISAs are available to detect serum antibodies, the best do not detect positive status for at least 6-8 weeks post-infection. A rapid immunomagnetic capture assay for the detection of *M. hyopneumoniae* directly from lung and respiratory tract washings was therefore developed. The success of the assay was dependent on the ability to purify antibodies which react against surface accessible membrane antigens of *M. hyopneumoniae*. Rabbits were immunized against whole cells of *M. hyopneumoniae* and serum immunoglobulins (Igs) were purified by ammonium sulfate precipitation, then further fractionated into sub-classes (IgG<sub>1</sub> and IgG<sub>2</sub>) by ion-exchange chromatography.

Purified IgGs retained strong immunoreactivity to surface accessible membrane antigens measured by whole-cell ELISA using methanol-fixed *M. hyopneumoniae* as the target antigen. The target specificities of these were identified by immunoblotting and strong immunoreactivity was shown to subunit antigens of approximate molecular sizes of 80, 55, 50, 44 and 25 kDa. Immunoblots of subunit antigens (whole cell lysates) of the three porcine mycoplasmas commonly found in the respiratory tract of pigs (*M. hyopneumoniae*, *M. hyorhinis* and *M. flocculare*) reacted against the purified IgG<sub>2</sub> and showed strong cross-reactivity at these molecular sizes, suggesting that this reagent may bind a range of mycoplasmal organisms. Purified IgG<sub>2</sub> was labelled with biotin and incubated with serial dilutions of a measured concentration of *M. hyopneumoniae* (4 h, 37°C). Streptavidin-coated magnetic beads were added (1 h, 25°C) and the captured *M. hyopneumoniae* cells were subjected to lysis by boiling to release template DNA for polymerase chain reaction (PCR). The initial concentration was based on colony-forming units of strain J. Using freshly cultured organisms seeded into either phosphate buffered saline (PBS) or respiratory tract washings (RTW) derived from *M. hyopneumoniae*-free pigs, 10-fold dilutions from 10<sup>6</sup> to 10<sup>1</sup>/ml were used to determine assay sensitivity.

Specificity of detection was assessed by the use of PCR species-specific primers (Stemke *et al.*, 1994) which amplify a 238 base pair fragment of a portion of the 16S rRNA gene of *M. hyopneumoniae*. No amplification was observed using *M. hyorhinis* template DNA and a 400 bp fragment was amplified using *M. flocculare* DNA. Results of direct PCR and immunocapture PCR were compared in the diluted preparations of *M. hyopneumoniae* cells and in a sample of lesioned pig lung, derived from an experimental infection with strain Beaufort, which was subjected to homogenization in PBS (3 g/10 ml).

Direct PCR detected 10<sup>3</sup> *M. hyopneumoniae* cells/ml from seeded PBS or RTW. Its sensitivity was slightly reduced using RTW compared to PBS, with a faint 238 bp PCR product being amplified at this the most sensitive level of detection. In contrast, immunocapture PCR detected *M. hyopneumoniae* at concentrations down to 10<sup>1</sup> cells/ml, indicating that immunocapture PCR is 100 fold more sensitive than direct PCR. Only immunocapture PCR was able to detect *M. hyopneumoniae* in lesioned lung material at the dilution rate used. This technology is currently under assessment for the detection of *M. hyopneumoniae* in nasal swabs, lungs and RTW from naturally and experimentally infected pigs.

### References

STEMKE, G.W., PHAN, R., YOUNG, T.F. and ROSS, R.F. (1994). *American Journal of Veterinary Research*, 55:81-84.

## SUBUNIT ANTIGEN PROFILES OF GEOGRAPHICALLY DIVERSE PORCINE MYCOPLASMAS RECOVERED FROM PNEUMONIC LUNGS

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Because of the difficulty in culturing of field strains of *Mycoplasma hyopneumoniae* from pig lungs, a limited number of laboratory adapted strains (eg., strain J) have been used for the purification of candidate subunit antigens for experimental vaccine trials. Although several studies have compared antigen profiles amongst the three mycoplasma species which inhabit the porcine respiratory tract (*M. hyopneumoniae*, *M. hyorhinis* and *M. flocculare*), few studies have addressed the possibility of antigenic variability amongst field and type strains of *M. hyopneumoniae* from different geographic locations.

Protein and immunoreactive antigen profiles of eight strains of *M. hyopneumoniae*, three of *M. hyorhinis* and one of *M. flocculare* were compared using SDS-PAGE and immunoblotting. The *M. hyopneumoniae* strains Beaufort, Sue, LKR and OMZ407 were originally recovered from pneumonic pigs in Victoria whilst strain C1735/2 was isolated from pigs in Queensland. The *M. hyopneumoniae* strains J, 232 and YZ originated from England, the USA and France respectively. Strains GDL, BTS-7 and 1 untyped field strain of *M. hyorhinis*, and the type strain of *M. flocculare* (Ms42) were studied. The identity of all 12 mycoplasmas was confirmed using a species-specific polymerase chain reaction (PCR) amplifying a 238 bp fragment or a 400 bp fragment of a portion of the 16S rRNA gene of *M. hyopneumoniae* and *M. flocculare* respectively (Stemke *et al.*, 1994).

A comparison of protein profiles amongst the three mycoplasma species showed considerable diversity. However, the eight *M. hyopneumoniae* strains also displayed marked profile differences and it was not always possible to predict the species of mycoplasma from the protein profile. In contrast, *M. hyopneumoniae* strains (except C1735/2) produced very similar immunoreactive antigen profiles when immunoblotted using hyperimmune anti-*M. hyopneumoniae* sera. Major immunoreactive antigens of molecular size 200, 114, 106, 94, 78, 43, and 36 kDa were conserved. Although the two *M. hyorhinis* type strains (GDL and BTS-7) produced very similar immunoreactive protein profiles when reacted against hyperimmune anti-*M. hyorhinis* sera, identifying proteins species of molecular size 106, 94, 85, 80, 78, 52, 49, 47, 44 and 42 kDa, the *M. hyorhinis* field strain produced a unique profile suggesting variability amongst *M. hyorhinis* isolates. The *M. hyopneumoniae* strain C1735/2 displayed a poorly immunoreactive immunoblot profile when reacted against hyperimmune anti-*M. hyopneumoniae* sera.

A preparative SDS-PAGE profiling (PPP) method which characterises the ELISA reactivity of denatured subunit antigens resolved on the basis of molecular size was developed to study these isolates. Aliquots of fractions eluted from a BioPrep 491 column (Bio-Rad) were coupled to microtitre plates, washed, and reacted with appropriate pig anti-mycoplasmal antisera. Immunoreactive antigens were detected by the addition of horseradish peroxidase conjugate and the colour reaction measured spectrophotometrically in an ELISA reader. Typical ELISA profiles for *M. hyopneumoniae*, *M. hyorhinis* and *M. flocculare* were determined, and both species-specific and cross-reactive antigens identified. For *M. hyopneumoniae*, specific antigens of molecular size 43, 74 and 94 kDa were identified.

Geographically diverse strains of *M. hyopneumoniae* therefore show common immunoreactive components by Western blotting and ELISA, despite considerable diversity in their protein profiles. The findings also indicated some strains of *M. hyopneumoniae* are poorly immunoreactive on blots, while there is apparent diversity in immunoblot reactivity among strains of *M. hyorhinis*.

### References

STEMKE, G.W., PHAN, R., YOUNG, T.F. and ROSS, R.F. (1994). *American Journal of Veterinary Research*. 55: 81-84.

## STRATEGIES FOR DEVELOPING A SUBUNIT MYCOPLASMAL VACCINE FOR ENZOOTIC PNEUMONIA

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*Mycoplasma hyopneumoniae* colonisation of the mucosal surface of the respiratory tract results in subclinical respiratory disease and causes losses by lowering growth rate and feed efficiency, and increasing the usage of medication. Earlier studies in this laboratory indicated that recently weaned pigs are challenged heavily as they enter grower sheds in "continuous farrow to finisher piggeries" where disease is endemic. The aim of the present work was to identify protective protein subunits of *M. hyopneumoniae* which can ultimately be produced or delivered more cheaply than whole cell vaccines from overseas.

The present studies have targeted the protein components of the membrane which surrounds *M. hyopneumoniae*, a key structure in attachment to respiratory tract mucosa. After isolating membranes of strain J, their protein subunits were fractionated by molecular size; of six fractions, only two (Frac 2 and 3) were found to protect against severe pneumonia following experimental challenge in three pig trials. The measurement of pneumonia, by Goodwin lung scoring (Goodwin *et al.*, 1969), was found most suitable for making statistical comparisons when lung scores (LS) were transformed by the logit function: transformed LS =  $\log_e(\text{LS} + 0.5/55 - \text{LS} + 0.5)$ . Vaccines were made by mixing membrane fractions with a range of adjuvant preparations including aluminium hydroxide (AH), Auspharm oil (AO), Algammulin, DEAE/mineral oil and DEAE/AO. Superior results for pneumonia control were achieved with vaccines incorporating AH or AO given intra-muscularly and intra-peritoneally respectively. In groups of 3-4 pigs, average daily weight gain (ADG) was not significantly greater than controls with Frac 2 or 3 vaccines.

A recent study was undertaken involving a mixture (VM) of Frac 2 and 3 with a recombinant membrane protein of strain J (NrdF). These were made with AH, AO or the novel iscosome adjuvant SAMA4, given intramuscularly, intraperitoneally or intradermally respectively in groups of eight pigs. Two further groups of eight pigs received either Fraction 3/AO or no vaccine. Treatment groups were matched for body-weight and pigs randomly allocated among groups. Pigs were vaccinated at 6 and 10 weeks of age, experimentally challenged with infected lung homogenate at 12 weeks, and slaughtered at 19 weeks. The ELISA antibody responses to Frac 2, Frac 3, NrdF, *M. hyopneumoniae* whole cells and subunit membrane antigens were estimated in serum collected weekly, and in respiratory tract washings collected after vaccination and after challenge. Two of 40 pigs were excluded on the basis of unrelated pre-existing disease. After experimental challenge, the variability in pneumonia scores of the control (unvaccinated) group precluded useful comparisons between groups, although significant differences were detected in ADG among groups. The ADGs of pigs vaccinated with VM + AH were higher than controls (NS), and significantly higher than the standard Frac 3/AO vaccinated pigs ( $P < 0.05$ ) and all four groups taken together ( $P < 0.05$ ). The level of pneumonia in the VM + AH group ( $4.7 \pm 1.6$ ; mean LS  $\pm$  SE) was not significantly lower than controls ( $12.9 \pm 4.9$ ).

In all studies, there have been no firm indicators of pre-challenge protection from circulating or secreted respiratory antibodies. A consistent finding has been the lack of a detectable IgA response in respiratory tract washings of protected pigs after vaccination. A rapid rise in mucosal antibodies after challenge has been found in some but not all protected pigs, and adjuvants appear to play a role in modifying the serum and respiratory tract antibody response. Cell-mediated immunity requires further attention to identify reliable markers of immune protection for respiratory diseases like enzootic pneumonia.

### References

- GOODWIN, R.F.W., HODGSON, R.G., WHITTLESTONE, P. and WOODHAMS, R.L. (1969). *Journal of Hygiene (Cambridge)*. 67:193-208.

## A POST-MORTEM STUDY OF SOW DEATHS ON A LARGE VICTORIAN PIG FARM

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Sow wastage is comprised of sow deaths and unplanned culling. The mortality rate of Australian sows appears to be double that in the USA (Ransley and Cleary, 1995; Marsh *et al.*, 1992). Unplanned culling of sows also appears to be a significant cause of loss of sows in some Australian piggeries (Paterson *et al.*, 1995). Humane euthanasia is a component of unplanned culling.

Laboratory post-mortems were performed on sows which either died (n=26) or were euthanased (n=50) on a large Victorian piggery (7500 sows) from 3/1/95 to 19/4/95 (Table 1).

**Table 1. Post-mortem findings in 26 sows which died and 50 sows which were euthanased on a large Victorian pig herd.**

Deaths	No.	Euthanasia	No.
<b><u>Infectious causes</u></b>			
Pyelonephritis/cystitis	5	Bacterial arthritis	17
Pericarditis	2	Spinal abscess	6
Pneumonia	2	Pyelonephritis	1
Septicaemia	2	Metritis	1
Cardiac abscess	1	Meningitis/otitis interna	1
		Pneumonia	1
<b><u>Non infectious causes</u></b>			
Gastro-intestinal accident	6	Limb fracture	5
Gastric ulcer	3	Spinal column fracture	3
Farrowing problem	2	Farrowing problem	3
Stress	1	Gastric ulcer	3
Heart failure	1	Splayleg/muscle necrosis	2
		Ischial necrosis	1
		Large ovarian cyst (50-60 L)	1
No diagnosis	1	No diagnosis	5

A wide range of causes of death were found. Gastro-intestinal accidents and bacterial kidney and bladder infections (pyelonephritis/cystitis) were the two most common diagnoses. Five of the six gastro-intestinal accident cases occurred between farrowing and weaning, whereas all of the five pyelonephritis/cystitis deaths occurred at variable times post-mating.

By far the most common reason for euthanasia on this farm was lameness resulting from bacterial arthritis. Nine of the 17 cases of arthritis yielded a streptococcal species of bacteria (principally Group C and untypable) and six cases yielded *Actinomyces pyogenes*. *Actinomyces pyogenes* was also recovered from four of the six spinal abscess cases.

### References

- MARSH W.E., VAN LIER P. and DIAL G.D. (1992). *Proceedings of the 12th International Pig Veterinary Society Congress*, The Hague, The Netherlands, p. 512.
- RANSLEY, R. and CLEARY, G. (1995). "Pig Stats 94". (Pig Research and Development Corporation and Australian Pork Corporation: Canberra).
- PATERSON R.A., CARGILL C.F. and POINTON A.M. (1995). *Proceedings of the Australian Association of Pig Veterinarians Programme, Australian Veterinary Association Conference*, Melbourne 1995, pp. 11-15.

## LEPTOSPIROSIS - MORE THAN MEETS THE EYE

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Leptospirosis is caused by pathogenic strains of the bacterial genus *Leptospira*. Leptospirosis of pigs can affect reproductive performance, and human leptospirosis of pig origin is an important zoonosis. Leptospire show morphological similarity, but great genetic and antigenic diversity. They are classified into serovars, according to the agglutinating antigens of the surface lipopolysaccharide, and serovars with some antigenic similarity are associated in serogroups. There are over 200 recognised pathogenic serovars within 23 serogroups. Six pathogenic species have been defined using DNA-DNA hybridisation, but there is no relationship between the antigenic and genetic classifications. A number of serovars can infect pigs and cause reproductive disease. Clinical leptospirosis in Australian pigs is traditionally attributed to serovars *pomona* and *tarassovi*. Pig vaccines available in Australia contain these two serovars, or serovar *pomona* alone. However, it is now clear that other leptospire are present in Australian pigs. There is serological and other evidence for endemic infection with serovar *bratislava* which is a recognised pig pathogen in Europe and North America (Chappel *et al.*, 1993). Furthermore, isolates have recently been obtained from two Australian states of a new previously-unrecognised serovar within a new serogroup (Billinghurst *et al.*, 1994).

**Table 1. Results of the microscopic agglutination test (MAT titre) on sera from 522 pigs slaughtered in Victorian abattoirs. The number of sera for each titre and each leptospiral serovar is shown, together with their proportion of the total (as %).**

Serovar	MAT titre					Total
	<32	32	64	128	≥256	
<i>bratislava</i>	196 (38)	102 (20)	98 (19)	70 (13)	56 (11)	522
new serovar	234 (45)	102 (20)	91 (17)	56 (11)	38 (7)	521
<i>pomona</i>	462 (89)	20 (4)	10 (2)	10 (2)	20 (4)	522

	MAT titre					Total
	<100	100	200	400	≥800	
<i>tarassovi</i>	513 (98)	4 (1)	4 (1)	0	1 (0.2)	522
<i>hardjo</i>	516 (99)	5 (1)	1 (0.2)	0	0	522

Sera from 522 pigs from over 122 herds were collected in Victorian abattoirs and tested by the microscopic agglutination test (MAT) for five leptospiral serovars (Table 1). The order of serological prevalence was *bratislava* > new leptospire > *pomona* > *tarassovi* > *hardjo*. Thus currently available vaccines may not protect adequately against clinical leptospirosis in Australian pigs.

## References

- BILLINGHURST, M.L., CHAPPEL, R.J., MORROW, C.J. and ADLER, B. (1994). *Australian Microbiologist*, 15:A127.  
 CHAPPEL, R.J., BILLINGHURST, M.L., WAN, S.S., HENNESSY, D.P., ELLIS, W.A. and ADLER, B. (1993). In "Manipulating Pig Production. IV", p. 253, ed. E.S. Batterham. (Australasian Pig Science Association: Attwood).

## A REVIEW - EVALUATION OF THE PIG HEALTH MONITORING SCHEME AS AN INDUSTRY SERVICE

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### Introduction

The purpose of this review is to evaluate the Pig Health Monitoring Scheme (PHMS) so as to provide potential investors in the technology with an assessment of its advantages, constraints, ease of implementation and applications. Recent publication of the approach (Pointon *et al.*, 1992a), together with several studies evaluating the disease monitoring systems used and reports on the disease status of various countries using the same scoring system, enables potential users to judge the suitability of PHMS to address their needs.

### The Pig Health Monitoring Scheme

#### *Principles*

Recording disease data at slaughter defines herd health status for sub-clinical conditions, enabling veterinarians to link disease prevalence associated with certain environmental conditions and husbandry practices with biological and financial performance. The health status of pigs can be quantified during their most costly phase of production, the grower/finisher phase, by monitoring a range of organ systems from a representative sample of pigs taken from the "grower/finisher" population (Pointon *et al.*, 1992a).

#### *Objectives and methods*

Inspection of gross lesions in slaughtered pigs may be used for many purposes which include: To diagnose disease problems in herds and estimate prevalence of disease; for the surveillance of herds with high health status; in decision support models and quality assurance programs; and to help communicate with and motivate producers (Willeberg *et al.*, 1984/85, Elbers *et al.*, 1991, Hurnik, 1991, Pointon *et al.*, 1992a).

Secondary use of the data is made through the compilation of regional, state and national disease reports (Pointon and Davies 1991; Elbers *et al.*, 1991; Pointon *et al.*, 1992a; Bahnson *et al.*, 1992a; Pointon *et al.*, 1994).

#### *Users and uses of lesion data from monitoring pigs at slaughter*

##### 1. Producers and practitioners:

- Diagnosing herd problems.
- Monitoring endemic diseases.
- Evaluating disease control strategies.
- Evaluating disease exclusion programs.
- Aid to defining herd disease profile.
- Relating disease to performance.
- Detection of new diseases.

##### 2. Industry:

- Defining endemic disease problems across industry.
- Providing a basis for research/extension/development funding.
- Indirect evaluation of the adequacy of environment and management standards across industry.
- Monitor new or emerging diseases.

### 3. Researchers and funding agencies:

Definition of industry problems.

Identification of herds suitable to include in research programs.

Monitor impact of new production methods.

### 4. Pharmaceutical companies:

Direction for product development.

Marketing information to target sales.

### 5. Abattoirs:

Definition of disease problems on a herd and industry basis which interfere with processing.

Assists production of a standardized carcass which can be efficiently processed.

### 6. Extension services:

Targeting programs at regions/states/countries with particular problems.

### 7. Legislators:

Provides industry perspective on disease as a basis for implementing regulatory disease control/eradication programs

#### *Standardized Pig Health Monitoring Scheme protocols*

Protocols describing the standardized methods used in the Australian PHMS have been published (Pointon *et al.*, 1987, 1992a). Fourteen production limiting and zoonotic conditions are routinely recorded including papular dermatitis, nephritis, pleuropneumonia and ileitis which were not included in previous schemes. All lesions are recorded irrespective of severity. The severity scores are reported where they add useful diagnostic information; as has been demonstrated for sarcoptic mange (Davies *et al.*, 1992), leptospirosis (Chappel *et al.*, 1992) and proliferative enteritis (Jones *et al.*, 1993).

The recording of all lesions in PHMS prevents the under-estimation of diseases, such as pneumonia (Wallgren *et al.*, 1990), pleurisy and ascariasis (P. Wallgren personal communication), which can occur when meat inspectors only record moderate to severe lesions. The differentiation of pleuritic lesions into lungs with and without adhesions to the thoracic rib wall provides processors with an on-going assessment of the likely impact of these lesions on the efficiency of processing.

#### *Interpreting herd reports*

Slaughter reports provide both an assessment of the sub-clinical health status of the "finisher" pig herd and an indirect assessment of the suitability of the environmental and management conditions over preceding months. In this regard the prevalence is the product of two sets of risk factors, being those determining the incidence of infection (commonly commencing in the weaner accommodation) and those affecting the duration of lesions during the grower/finisher phase.

The validity of lesion data as an estimate of the lesion profile of slaughter pigs from a herd is determined by the sample size, inspection frequency and sampling procedure. The ability to extrapolate from the lesion profile to reach conclusions about the economic significance of the diseases in the herd is restricted by among herd variability in age of infection, lesion duration and slaughter age. Together with the incidence, these factors are major determinants of the lesion profile of slaughtered pigs, and knowledge of them will greatly facilitate the interpretation of herd monitoring data. The interpretation of PHMS reports also requires consideration of the statistical accuracy (Pointon *et al.*, 1992a); the level of confidence of the data; the clinical history of the slaughtered stock including duration of lesions; the growth performance of the group; the age of stock; and the environment/management conditions to which stock were exposed during growth.

To assist in the interpretation of slaughter surveillance data, practitioners should encourage producers to keep a record of clinical observations and treatments. The duration of infections, such as pneumonia for example, determines the degree of growth depression (Wallgren *et al.*, 1990; Noyes *et al.*, 1990). Therefore, on-farm recording



systems devised to identify peaks in infection will assist practitioners in relating morbidity and mortality information to lesions observed at slaughter. This can be achieved by establishing clinical indices for conditions of interest to determine the time of onset of peak infections and duration of lesions. Rubbing indices (rubbing episodes/pig/15 minutes) have been used as a basis of relating high average mange score at slaughter to infestation of weaners with sarcoptic mange (Davies *et al.*, 1992). Similar indices have been designed for pneumonia (Gardner and Hird, 1990), atrophic rhinitis (coughing-sneezing episodes/pig/15 minutes) and ileitis (total pen scour days/week).

Together with treatment and culling/mortality records, clinical indices add an important historical perspective to disease episodes and allow much more accurate interpretation of monitoring results. Each age group should be examined at an age and time when they are most likely to be showing clinical signs. For example, pigs might be examined for indications of pneumonia or atrophic rhinitis when first disturbed in the morning. Clinical indices should be recorded approximately once per week. A more definitive approach for determining the age at which clinical disease occurs in a group of pigs would be to perform cross-sectional serological profiles for each age group. Serological profiling has been suggested as a useful adjunct for slaughter surveillance of pneumonia (Wallgren *et al.*, 1990) and pleuropneumonia (Donadeu *et al.*, 1994).

Relating lesion prevalence and severity to reduced performance should be evaluated within each herd by comparing the results of repeated checks with herd growth efficiency. Studies of the economic significance of enzootic pneumonia (reviewed by Pointon *et al.*, 1992a) and sarcoptic mange (Cargill and Dobson, 1979) have attempted to evaluate the biological effects of these conditions under conditions similar to those commonly encountered in the field. While demonstrating a significant growth depressing effect, these results provide only a guide to the degree of economic impact that these conditions may exert; practitioners must perform the final evaluation using additional growth data collected in the herd.

The interpretation of data collected at slaughter on lesion prevalence, should therefore, be expected to vary from herd to herd according to the epidemiology of disease within that herd. Consequently, similar lesion profiles in different herds may have considerably different economic implications.

Field veterinarians should also be provided with up to date information on disease trends across industry and the epidemiology of diseases within herds to provide a background on which the significance of herd reports can be judged (Pointon *et al.*, 1995).

#### *Australian Pig Health Monitoring Schemes*

Initial reports on the establishment of PHMS as an industry based program came from South Australia and Western Australia (Pointon *et al.*, 1987; Mercy and Brennan, 1988). The scheme has progressively been implemented in Queensland, New South Wales and Victoria on a coordinated basis with assistance from the Pig Research and Development Corporation for the last two states. Membership of the national PHMS at the end of 1994 comprised 594 herds; SA with 133 herds, WA 156, Qld 135, Victoria 88 and NSW 82.

Participating herds comprise 35% of Australian herds with greater than 100 sows/herd. Greater definition of industry coverage will be obtained once herd details have been entered into the upgraded version of PIGMON3.0™. There remains considerable opportunity for PHMS to be adopted in the Australian pig industry and its increased use may in part depend on the ability of field veterinarians to interpret and apply the data accurately for producers (Pointon *et al.*, 1995). The enhanced graphics in producer reports and peer group comparisons of herd data, features of PIGMON3.0™, will provide another opportunity to market PHMS to producers nationally.

With the publication of state and national disease reports (Pointon *et al.*, 1994, 1995) priority has been given to funding major projects on risk factors, pleurisy and air quality in piggeries. On the basis of these reports the author concludes that the worsening of respiratory disease status nationally, despite substantial veterinary intervention, justifies investment in multi-site and segregated early weaning technologies (Henry 1995).

### *International implementation*

The use of PHMS internationally has been reported from the US (Bahnsen *et al.*, 1992a, 1992b; Jones *et al.*, 1993) and New Zealand (Christensen and Cullinane 1990). The use of techniques for monitoring sarcoptic mange developed in PHMS (Pointon *et al.*, 1992a) has been reported in Canada, Mexico, Spain (Garcia *et al.*, 1994), Italy (Gualandi *et al.*, 1994) and the UK (White, 1995). The PHMS is also being implemented for the South African pig industry (J. Robinson, personal communication).

### **Quality assurance and validation of Pig Health Monitoring Schemes**

#### *Quality assurance program*

Quality assurance exercises have been designed and conducted with the aim of standardising techniques of inspection and regulating the qualitative and quantitative observation of lesions. With this achieved it will be possible to:

- compile a national pig disease database;
- define national and regional problems;
- develop collaborative research and extension projects across the national pig industry.

To standardise the implementation of PHMS internationally, training and quality assurance (QA) exercises have been conducted with inspectors from each mainland Australian state and with the PHMS co-ordinator/reference inspector of the US pig industry. A manual describing the QA exercises and statistical methods is available from the author.

The QA procedure involves the inspection and scoring of a selection of organs for a range of lesions; at least 24 examples of each organ should be examined, with each identified by number. Test inspectors have their observations compared with those of the reference inspector for the same sets of organs. Atrophic rhinitis scoring is usually assessed using colour projection slides or photocopies of snout cross sections. Papular dermatitis scoring is most easily done at an abattoir on the moving chain of carcasses. For conditions such as respiratory diseases, roundworm infestation, nephritis, proliferative enteritis (PE) and proliferative haemorrhagic enteropathy (PHE), organs can be collected for subsequent observation either on-site or elsewhere.

Once recordings have been completed, the degree of concordance with the reference inspector can be calculated using several methods (P. Davies, personal communication). The detection agreement percentage is a measure of the extent to which the test inspector detects the presence or absence of lesions on a line of the same organs. Where organs can be categorized in more than two ways (eg., pneumonia can be absent, acute or chronic) kappa values (Fleiss, 1981) are used to describe the extent to which a test inspector's scoring or typing of organs matches the observations of the reference inspector. Weighted kappa values are used when more than one positive severity score is possible. This has the effect of crediting the test inspector for scores close to those of the reference inspector, while at the same time penalising for scores too far from reference scores. The concordance correlation coefficient (I-Kuei Lin, 1989) is used when comparing test and reference lung scores, and defines the extent to which all pairs of scores, when used as graph coordinates, will describe a 45° line through the axes origin.

The data presented in Table 1 summarises the results from two Australian QA workshops and one American exercise, where the Australian PHMS reference inspector was compared with the US reference inspector. The Australian exercises were conducted at abattoirs, and were limited by the availability of lesions, which is why some conditions were not observed.

**Table 1. Disease classification and scoring agreement, for five diseases, found among the reference inspectors (Ref.) from various Australian states and other Australian PHMS inspectors; and for the USA reference inspector as measured against the Australian reference inspector.**

OBSERVER	Western Australia		Victoria		NSW	QLD	USA
	Ref.	Other	Ref.	Other	Ref.	Ref.	Ref.
<i>Pneumonia</i>							
detection <sup>1</sup>	93	96	79	88	79	100	100
active/chronic <sup>2</sup>	0.63	0.59	0.36	0.70	0.74	0.75	1
scoring <sup>3</sup>	0.94	0.97	0.88	0.91	0.69	0.99	0.99
<i>Pleurisy</i>							
detection <sup>1</sup>	97	100	NA	NA	NA	65	87
scoring <sup>2</sup>	0.87	1				0.37	0.68
<i>Roundworm</i>							
detection <sup>1</sup>	NA <sup>4</sup>	NA	100	91	100	96	96
scoring <sup>2</sup>			1	0.66	1	0.76	0.96
<i>Papular dermatitis</i>							
detection <sup>1</sup>	NA	NA	91	61	86	NA	86
scoring <sup>2</sup>			0.32	0.21	0.31		0.57
<i>Atrophic rhinitis</i>							
detection <sup>1</sup>	NA	NA	NA	NA	NA	96	90
scoring <sup>2</sup>							0.90

<sup>1</sup>% agreement. <sup>2</sup>Kappa ranking;  $\geq 0.67$  = reasonable agreement,  $\geq 0.75$  = excellent agreement. <sup>3</sup>Concordance correlation coefficient for sample sizes used (24-32);  $> 0.5$  = good reproducibility,  $> 0.75$  = very good,  $> 0.9$  = excellent. <sup>4</sup>Not available.

There was a generally high degree of agreement for the detection of all five conditions. The kappa scores also showed reasonable agreement among the various assessors with the exception of papular dermatitis where the agreement was poor. This was largely attributable to overscoring the severity of lesions. The comparison between the US and Australian reference inspectors were consistently good to excellent, with the poorest result again, being recorded for papular dermatitis. The scoring technique for papular dermatitis has only recently been introduced into the PHMS. These results indicate that future training and QA exercises should place greater emphasis on scoring papular dermatitis. While this lack of agreement for papular dermatitis is a disappointing result, experience with the scoring technique indicates that it offers an easy, quantitative and highly specific method to diagnose mange hypersensitivity on a herd basis (Davies *et al.*, 1992), supporting its further evaluation.

#### Validation studies

##### *Determining the specificity of lesions*

While many of the lesions monitored are described morphologically (eg., pleurisy, peritonitis, arthritis, ileitis, nephritis), for some conditions the lesions are attributed to specific agents (*Sarcoptes scabiei*, *Mycoplasma hyopneumoniae*, *Ascaris suum*, *Actinobacillus pleuropneumoniae*, leptospira). Prevalence estimates are a function of the sensitivity and specificity of the inspection methods and of the sampling procedures employed. The range of disease conditions which contribute to reduced specificity of lesion monitoring was reviewed by Pointon *et al.*, (1987, 1992b).

Specificity, is determined by the prevalence of lesions, resulting from other agents, which may be mistaken for those of the disease of interest. The positive predictive value (PPV) of the presence of lesions depends upon the specificity of the lesions and the prevalence of the lesions resulting from the condition of interest. In estimating the prevalence of lesions of common diseases, in herds known to be affected with the diseases in question, the occasional presence of non-specific lesions will have a minor

effect because the PPV will be high. Where a herd is considered free from a given disease(s) (ie., expected prevalence of zero), interpretation is more difficult.

The threshold prevalence for suspecting the introduction of a specific agent into a previously free herd will vary between conditions according to specificity. Examples of lesions which appear to have high specificity include those of mycoplasma pneumonia (MP) (Pointon and Sloane, 1984; Hurnik, 1991) and ascariasis (Jubb and Kennedy, 1963). For these conditions non-specific lesions are uncommon and the presence of any lesions is suspicious and warrants further investigation and differentiation, in the case of MP to differentiate it from lesions caused by serovars of *Actinobacillus pleuropneumoniae*.

#### *Sarcoptic mange*

In contrast to enzootic pneumonia and ascariasis, a 20% prevalence of grade 1 papular dermatitis-like-lesions (estimated specificity of 79%) is not inconsistent with freedom from sarcoptic mange (Davies *et al.*, 1992). For this disease the assessment of herd status is enhanced by the severity scoring system, as grade 2 and 3 (generalized papules) lesions have a specificity of >98%. Similar results have been reported in several other countries (Garcia *et al.*, 1994).

The method, therefore, cannot be used to confirm absence of mange, but can be used with confidence to confirm its presence. Because of an apparent inverse relationship between the severity of lesions and ability to recover mites on an individual pig basis (Cargill *et al.*, 1995), monitoring papular dermatitis at slaughter offers both ease and accuracy in diagnosing sarcoptic mange.

#### *Proliferative enteritis*

Proliferative enteritis (PE) presents in two clinical forms, non-haemorrhagic PE and proliferative haemorrhagic enteropathy (PHE). The diagnosis of PE is based on the gross presence of thickening of the ileum resulting from adenomatous intestinal lesions. Confirmation is by demonstration of typical microscopic changes and the intra-cellular bacterium, ileal symbiont-intracellularis (IS-intracellularis), within the enterocytes (Rowland and Lawson, 1975; Lomax *et al.*, 1982; Gebhart *et al.*, 1994).

In a recent US study (Jones *et al.*, 1993), only 21% of intestines chosen, on the basis of macroscopic thickening, were positive for PE by the two diagnostic criteria. When thickening was accompanied by some oedema, 50% were diagnosed as PE. Detection increased to 89% when substantial oedema of the mesentery or ileum was evident. To increase the likelihood of confirming a diagnosis of PE, clinicians and slaughter monitoring inspectors are strongly advised to submit ileal lesions which have extensive oedema of the mesentery or ileum for laboratory confirmation.

Because non-haemorrhagic PE often occurs in young pigs, 6-24 weeks of age (Wilson *et al.*, 1986; Holyoake *et al.*, 1994); and that lesions may recover spontaneously over 4-6 weeks (Rowland and Lawson, 1992), lesions detected at slaughter may be in the process of healing, ie., minimal oedema. While lesions in the early stages of healing may have a lower specificity, they should not be discounted as signs of PE; ie., they are likely to still be a reasonable predictor of active PE in the herd.

The age of onset of clinical signs is a major determinant in detecting lesions at slaughter. Non-haemorrhagic PE has been reported in pigs 6-17 weeks of age in the US and 6-24 weeks of age in Australia (Wilson *et al.*, 1986; Holyoake *et al.*, 1994). Consequently, it is not surprising that among the 6 herds studied in these reports in which non-haemorrhagic PE was diagnosed by post-mortem examination, that lesions were detected at slaughter in only one herd. However, inadequate sample size, which for some herds was down to 20 age matched pigs of normal market weight, may equally have accounted for the negative results. For conditions which occur at low prevalence, inspecting a sufficient sample of pigs to ensure detection at the expected prevalence is essential. In the case of PE a sample of 54 pigs is required to ensure detection (with 95% confidence) of one positive pig, when lesions are present at a 5% prevalence.

Proliferative haemorrhagic enteropathy has been reported to typically present as dysentery and sudden death in pigs aged 20-30 weeks (Love *et al.*, 1977) and 16-38 weeks (Holyoake *et al.*, 1994). As might be predicted, PHE is more closely associated with ileal lesions at slaughter. In 4 of 4 PHE-affected herds, ileal lesions were detected at

slaughter within 2 weeks of clinical outbreaks (Holyoake *et al.*, 1994). These findings support those of Pointon (1989) who found that 14 of 21 herds in which PE lesions were detected, at their first slaughter inspection, had experienced fatal cases of PHE in the preceding 10 weeks.

Despite the apparent low sensitivity of slaughter inspections for the detection of non-haemorrhagic PE, clinically-based diagnosis of morbidity appears poorer. Holyoake *et al.* (1994) reported that the on-farm morbidity of non-haemorrhagic PE was 5% (range 0-20%) compared with the prevalence of PE lesions at slaughter of 9% (range 0-20%). A similar relationship was found for PHE for which on-farm morbidity was estimated to be 6% (range 0.5-15%) compared with 17% (range 6-40%) at slaughter (Holyoake *et al.*, 1994). Differences in morbidity rates suggest that many pigs are sub-clinically affected (Holyoake *et al.*, 1994), making an accurate clinical assessment of the severity of the problem difficult, or worse, making the detection of a PE infection, difficult upon a veterinary inspection.

In herds with persistent PE problems, even slaughter inspections done as frequently as every 12 weeks may miss the presence of early warning lesions. Clinical cases of PHE have been observed in 6 herds in which ileitis was not detected at inspections conducted at 12 week intervals (Pointon, 1989). If PE is a persistent problem, an alternative to preventive medication of finishers is to conduct slaughter inspections every 4-6 weeks to detect early signs of disease.

### *Nephritis*

Because of the difficulty in diagnosing nephritis in growing stock on the basis of clinical signs this disease is especially suited to diagnosis through slaughter inspections. In SA in 1987, 11% of all pigs monitored had nephritis (Pointon *et al.*, 1987). These pigs were from 96 herds, of which 19% had >50% of pigs with lesions. Positive titres to *Leptospira pomona* were easily demonstrated in these herds. Chappel *et al.* (1992) demonstrated leptospires in 28% of 368 lesioned kidneys collected from Victorian abattoirs, thus confirming widespread leptospirosis infection in the Australian pig industry.

Leptospiruric pigs, with or without kidney lesions (Jones *et al.*, 1987) from infected herds, pose a threat to piggery and abattoir staff. In a serological survey of pig producers, 8% of 140 had positive titres to leptospira (Chappel *et al.*, 1990). As the infection appears to be endemic in industry, herd reports of low lesion prevalence should not be discounted; these may still pose a threat to abattoir workers and to a lesser degree farm staff and those involved in livestock transport. Improved diagnostic methods based on detection of specific DNA will also assist our understanding of the cause of these lesions.

## **Advantages and limitations of Pig Health Monitoring Schemes**

### *Comparison with other schemes*

Among the limitations of gross lesion data are imperfect sensitivity and specificity of gross examination, and the fact that these vary among observers. Variability among observers is assumed to be a major contributor to variability in lesion prevalence in slaughter monitoring programs in Europe (Willeberg *et al.*, 1984/85; Elbers *et al.*, 1991). This places substantial limitations on the use of data as an aid in herd health management. Failure to report mild lesions could result in early cases of new or emerging diseases being missed, thus falsely indicating the absence or presence of diseases. Such information should be reliably known when purchasing replacement breeding stock.

In the Australian PHMS greater emphasis is placed on recording production limiting conditions compared to the European schemes, in which meat inspection staff predominantly record lesions related to carcass wholesomeness. Fourteen production limiting and zoonotic conditions are recorded in Australia (Pointon *et al.*, 1992b), including ileitis (proliferative enteritis), nephritis (leptospirosis), pleuropneumonia (*Actinobacillus pleuropneumoniae*) and papular dermatitis (sarcoptic mange). These have not been included in previous schemes. All lesions are recorded irrespective of severity.

Furthermore, severity scores are reported where appropriate, eg., enzootic pneumonia, papular dermatitis, pleurisy, nephritis, liver spots and atrophic rhinitis. The recording of all lesions limits the underestimation of diseases, such as pneumonia (Wallgren *et al.*, 1990; Elbers *et al.*, 1992), pleurisy and ascariasis (P. Wallgren personal communication), which occurs when meat inspectors only record moderate to severe lesions.

The PHMS inspections are performed upon request by producers who pay a fee to the inspecting service (government or private). Reports include all lesions and severity scores. This contrasts with other systems such as the Danish one (Willeberg *et al.*, 1984/5) where the data is collected on all pigs, but the reports are only provided to producers when disease rates exceed programmed tolerance levels. In this situation the information could easily be under-valued by producers, who have not directly requested or paid for the data. The Danish approach also limits the use of reports as they cannot be used to assist in monitoring freedom from specific diseases.

The PHMS approach is particularly suited to pig industries which do not operate on the basis of producer cooperatives, where services are provided centrally for all of industry; eg., meat inspection services providing incomplete information on lesions observed at slaughter. The PHMS provides detailed information on a broad range of production limiting diseases which are a common feature of large herds, and which would not be of interest to meat inspection services.

A further limitation of the European approach is the low motivation of meat inspectors. This when coupled with the high speed of the slaughter chain and the variable perception and discrimination, results in the recording of data of variable quality (Elbers *et al.*, 1991).

#### *Producer evaluation*

Producer surveys to evaluate the usefulness of slaughter monitoring programs have generally indicated a high level of satisfaction (Shadbolt *et al.*, 1987; Mercy, 1992; Davies *et al.*, 1993). Most had found the system useful in diagnosing sub-clinical disease problems and a high proportion had taken action to modify disease control procedures. Producers require lesion diagnosis to be as detailed as possible and the severity of lesions should be reported. While veterinary services are regularly used by producers, concern was expressed in the Canadian survey (Shadbolt *et al.*, 1987) regarding the level of veterinary expertise in the interpretation of findings. While producers felt that they should contribute the majority of funds, support from national or state producer bodies is warranted.

#### *Constraints to interpretation of the data*

##### *Determinants of prevalence and lesion detection*

The prevalence of detectable lesions at slaughter is a function of incidence, age at infection, lesion duration and age at slaughter. For example, the prevalence of lesions resulting from chronic pleurisy has been shown to increase rapidly with decreasing slaughter age (Mousing, 1988). Over time, market-led changes in the distribution of age and weight of slaughtered pigs may affect estimates of industry lesion prevalence derived from slaughter monitoring data, while the disease incidence may actually remain unchanged. Thus, such estimates derived from health scheme data must be interpreted cautiously, given the biased nature of the sample and the number of factors which may influence lesion prevalence at slaughter.

The sensitivity of the inspection procedure for detection of disease in a herd is largely a function of sample size as well as the age of onset and duration of lesions. Guidelines for selecting sample size and estimating duration of lesions were reviewed by Pointon *et al.* (1992). Conclusions in PHMS reports, in which inadequate sample sizes were used for lesions which typically occur at low prevalence (eg., proliferative enteritis in the 5-10% range; Wilson *et al.*, 1986) should be viewed with caution.

### *Industry disease status*

Because PHMS inspections and reports are provided only to producers requesting the service, summary reports are likely to be biased towards larger herds, breeding stock suppliers, producers who are interested in optimising health and to those with current herd health problems.

The prevalence of diseases in the Minnesota PHMS data base have been compared with disease prevalence in randomly monitored pigs at the same abattoirs (Bahnsen *et al.*, 1992a, 1992b). Despite the above biases the study demonstrated that PHMS gave a reliable estimate of the respiratory disease status of industry. However, it substantially underestimated the prevalence of liver spots and papular dermatitis.

### **Support services required for Pig Health Monitoring Schemes**

When the original PHMS was initiated in South Australia (Pointon *et al.*, 1987) only those producers willing to nominate a veterinary service to assist in the interpretation of reports were admitted as members. This policy was adopted to provide uniformity in the epidemiological interpretation of the reports. Veterinarians were provided with training seminars and publications aimed at enhancing their interpretation of the reports. Subsequently some producers requested that membership not be restricted only to producers with nominated veterinarians. When nomination of a veterinary service was made optional 70% of producers still requested that their inspection reports be sent direct to their preferred veterinarian as well as receiving a copy themselves.

Veterinary laboratories provide an important backup service to PHMS. This is especially the case during implementation to check the specificity of classifications made by staff conducting inspections. In Australia, laboratory diagnostic support has also played an important role in confirming the emergence of pleuropneumonia across the Australian pig herd. Where lung abscesses occur it is important that lesions and tonsils from the affected pig are submitted for bacterial culture to confirm the presence of *Actinobacillus pleuropneumoniae* (B. Fenwick, personal communication). Similarly, pneumonic lesions indicative of mycoplasma pneumonia should be submitted from herds considered free of infection. Reliable laboratory support is essential for the credible use of PHMS as a diagnostic aid to veterinarians. Providing diagnoses without appropriate confirmatory testing can quickly erode client confidence.

### **Establishing Pig Health Monitoring Schemes**

#### *Implementation*

##### *Models*

A range of models have been used for the implementation of PHMS. Across Australia each state based PHMS has adapted the scheme to suit its particular existing resources and location of abattoirs. Implementation of the standardized monitoring protocols and a Quality Assurance (QA) program to monitor the accuracy of inspectors are, however, fundamental. Inspections are conducted by technical officers trained by the author in SA, clinical veterinarians in WA and NSW, veterinarians and technical officers in Victoria and state employed meat inspectors in Queensland. Each state has a data bureau coordinator who generates producer and summary reports from PIGMON™.

The preferred model is where the inspection service provider understands pig production, how producers intend to use results and is the first point of contact for receiving feedback from users of the information.

##### *Resources*

The goodwill of pig processors is probably the key to the success of implementing PHMS. Cooperation at the lairages can increase the efficiency of inspection through holding groups for inspection until the inspector arrives and staggering lines of pigs to enable one inspector to conduct the inspections. In abattoirs with chain speeds in excess of approximately 300 pigs/hour an assistant will be required to monitor carcass identification and record results. Use of a voice activated recorder can overcome the need

for an assistant in some circumstances. The Australian PHMS does not require the installation of computer keyboards for data entry such as is used in Europe. Basic equipment and data recording sheets required to conduct an inspection have been described by Straw *et al.* (1986) and Pointon *et al.* (1992a).

#### Validation studies

When initiating PHMS in a new country or region it is strongly advised that validation studies be performed for papular dermatitis (Davies *et al.*, 1992), nephritis (Chappel *et al.*, 1992), proliferative enteritis (Jones *et al.*, 1993) and pleuropneumonia. This will enable inspectors to set critical limits to the classification of lesions. Procedures should be established for the laboratory investigation of atypical or unexpected lesions; these should not be reported until confirmed, especially if the herd has been free of specific infections. Producers should be advised that suspicious lesions were detected and that laboratory results will follow. The QA program described previously is an essential part of the implementation of PHMS.

### Industry uses of Pig Health Monitoring Schemes

#### State and national health status

National PHMS data are published in this report for the first time (Table 2). Because of the use of common protocols and QA audit system for inspectors these can be directly compared with the US data provided by Bahnson *et al.* (1992a, 1992b). While the prevalence of (mycoplasma) pneumonia is substantially less in Australia than the US, the prevalence of pleurisy (Table 2) is substantially worse (Bahnson *et al.*, 1992a, 1992b).

#### Defining problems - pleuropneumonia and pleurisy

The rise in the prevalence of pleurisy in Australia (Figure 1) has occurred concurrently with a rise in the proportion of herds with pleuropneumonia lung lesions (Figure 2). While only 1.1% of pig lungs had lesions (Table 2), this hides the finding that from 18-50% of herds now have pleuropneumonia lesions observed annually. The relatively high level of respiratory disease in the USA has launched into the development of alternative pig production systems, eg., Segregated Early Weaning Systems (Henry, 1995) which are now being implemented in Australia for similar reasons (Thornton, 1995).

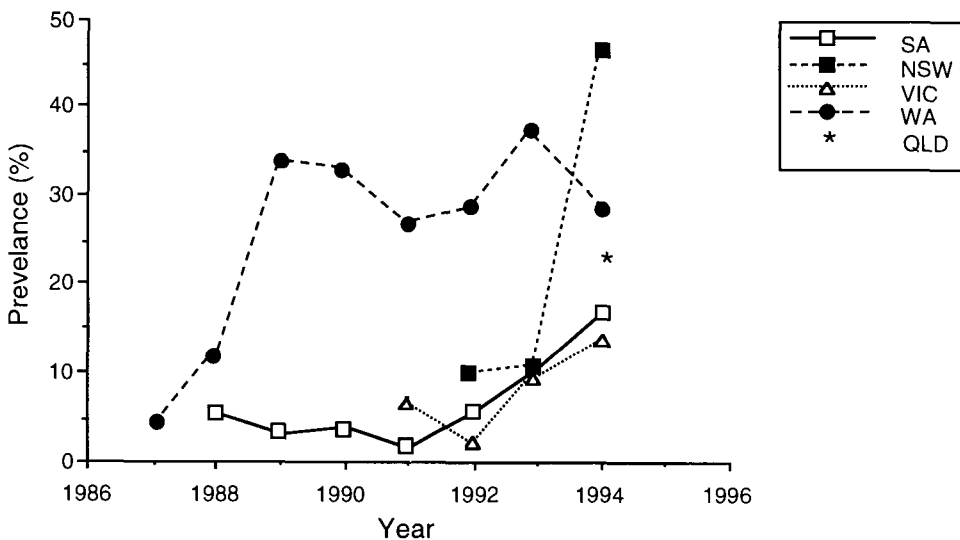


Figure 1. State herd prevalence of pleuropneumonia: percentage of herds with lesions.



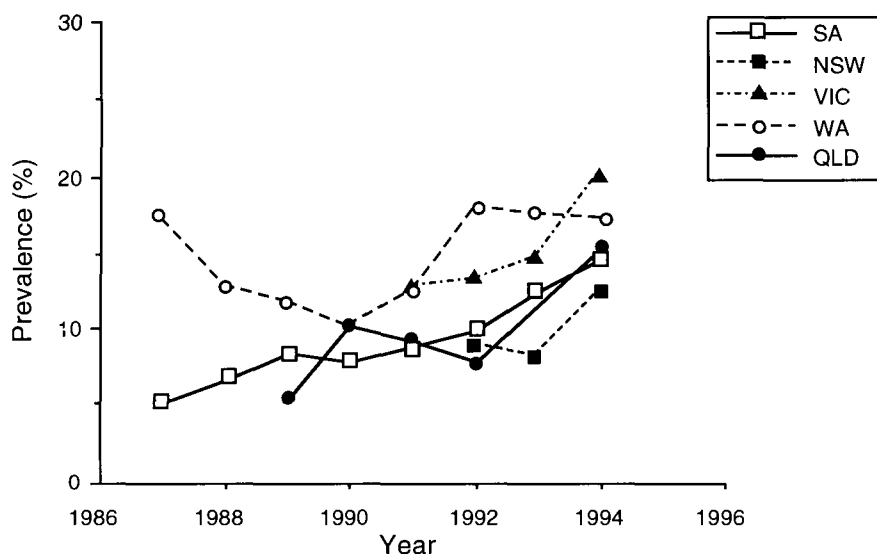


Figure 2. State pleurisy prevalence: percentage of all pigs monitored.

#### *Demonstrating trends in herd health*

While respiratory disease rates of PHMS herds in SA have increased in recent years (Figures 1 and 2), papular dermatitis scores have decreased (Pointon *et al.*, 1994). The average dermatitis lesion scores of 54 common herds monitored from 1987 to 1993 declined from 0.8 to 0.4 indicating a sustained improvement in the control of Sarcoptic mange. While PHMS cannot be held directly responsible for this improvement, the improvement may have in part been the result of positive feedback of objective data reflecting the efficacy of mange control procedures.

#### *Monitoring lesions affecting carcass "safety and wholesomeness"*

Lesion data obtained from meat inspection agencies in Queensland, Victoria and South Australia does not include information on the two conditions of major zoonotic and aesthetic importance; nephritis (leptospirosis) and pleurisy, respectively. During 1993 and 1994 the prevalence of nephritis recorded for all pigs inspected in PHMS was 4.5% and 7.1%, respectively (Table 2). The prevalence of grade 2 pleurisy -lesions, (adhesions between lung and ribs) which potentially requires partial carcass trimming, was 9.6% and 18.3% for the same years. These prevalences for nephritis and pleurisy greatly exceed the total condemnations for all causes reported by meat inspection services in South Australia in 1989-90 (Pointon and Arthur, 1995), which for partial and whole carcasses ranged from 0.17% to 1.46% of all pigs slaughtered.

While lesions related to herd growth performance are given priority in PHMS, carcass lesions reported include arthritis and abscessation (primarily from sites other than the head) which comprise the majority of causes for condemnation by meat inspection authorities (Pointon and Arthur, 1995).

#### **Ability to address changes in industry**

##### *Increasing herd size*

The continuing trend for increasing herd size in Australia (Ransley and Cleary, 1995) has maintained the pressure for respiratory disease to emerge as the industry's major disease problem because the prevalence of pneumonia is clearly associated with herd size (Pointon *et al.*, 1992b). Unless alternative production systems are employed,

Table 2. Lesion prevalence (%) in pigs at slaughter as recorded by state PHMS.

Lesion	South Australia		Western Australia		Victoria		Queensland		New South Wales		Total**	
	1993	1994	1993	1994	1993	1994	1994	1994	1993	1994	1993	1994
Pneumonia	50.6	55.4	29.9	28.6	41.2	53.4	52.5	62.3	46.3	38.6	46.3	
Pleurisy	17.8	20.7	20.2	20.7	17.9	37.1	15.1	13.8	16.5	19.1	20.7	
(Grade 2*)	(10.2)	(15.2)	NA	NA	(8.5)	(28.2)	NA	(3.7)	(13.0)	(9.6)	(18.3)	
Liver spots	1.4	1.6	7.5	7.3	9.3	5.1	1.8	1.3	1.4	5.2	4.1	
Nephritis	7.3	13.5	2.8	3.3	NA	NA	4.1	6.7	1.6	4.5	7.1	
Pleuropneumonia	1.5	1.2	0.8	0.8	0.5	1.7	0.3	0.2	4.8	1.0	1.1	
Pericarditis	2.6	2.5	2.4	2.4	1.9	2.3	2.1	1.2	2.1	2.4	2.3	
Peritonitis	1.8	2.5	1.5	3.4	0.3	0.3	1.0	0.3	0.2	1.5	2.0	
Arthritis	0.9	0.5	0.8	0.7	0.3	0.2	0.6	0.2	0.2	0.8	0.5	
Abscessation	1.0	0.7	0.3	0.3	0.7	1.0	1.4	0.6	0.1	0.6	0.8	

\* Grade 2 lesions expressed as a % of all pigs inspected (Pointon *et al.*, 1992a). \*\* Pooled data from all pigs monitored nationally. NA = not assessed.

such as segregated early weaning, to break the continual infection with respiratory diseases in growing stock, PHMS will continue to provide a useful tool in monitoring the severity of respiratory disease.

#### *New production systems*

If new production systems are implemented to break transmission of respiratory diseases, PHMS may fulfil an alternative role of monitoring freedom from lesions. Herds with a high health status, while subject to epidemic disease, are also susceptible to proliferative enteritis and infections which cause polyserositis for which PHMS can provide a useful diagnostic aid.

#### *Pig meat production quality assurance*

The point of slaughter provides an opportunity to monitor the prevalence of hazards (ie., lesions) that have been caused in the initial farm production step on the way to producing retail pig meat. Quality Assurance systems for the production of pig meat are increasingly using the HACCP (Hazard Analysis Critical Control Point) system to identify and eliminate hazards. Feedback of information enables producers to modify or implement production processes aimed at eliminating the hazard. In this way PHMS will provide producers with a valuable mechanism for the production of safe and wholesome pig meat.

### **Development of Pig Health Monitoring Schemes**

#### *PIGMON3.0™*

The computer program PIGMON, which is used to collate and analyse PHMS data and print producer reports, has been upgraded to PIGMON3.0™. The new version has been developed in Microsoft Access. The new version has many additional features and advantages compared with the old version including enhanced graphics and characterisation of herds.

It is envisaged that PIGMON3.0™ reports will be used by different sectors of the pig industry to assess disease of the Australian pig herd, determine disease research priorities and gauge the effectiveness of control and eradication programs.

Producer reports from PIGMON3.0™ will provide the previous 24 months data for every disease in an easy to understand graphical format. For each disease there will be comparative animal prevalence figures for those herds of the same size category and health status in the state. This feature will provide seasonal patterns and comparison of each herd's health status relative to its peer group. This will provide a sense of perspective on the relative health status for each particular herd and provide motivation for producers and consultants to address problems.

PIGMON3.0™ has greater flexibility than previous versions in terms of reporting and data analysis. This is because more information characterising the herds will be recorded and the program itself is capable of more complex data extractions. The program will be able to be used to examine the following effects on disease prevalence:

- production and housing system (eg., all-in all-out/continuous, intensive/extensive, multi-site/farrow to finish);
- health status (eg., specific pathogen free, segregated early weaning, conventional);
- climate (eg., Mediterranean, temperate and tropical);
- season;
- type of stock (eg., porker/baconer);
- herd size, and
- regional analysis

The standard epidemiological report in PIGMON3.0™ will list animal and herd disease prevalence for the last 10 years, however, a variety of other time periods and options will be available. Using the data analysis and query by example capabilities of Access, a vast capacity to ask specific questions on the disease and prevalence data in PIGMON3.0™ will be available.

Such reports will enable producers and industry support services to target resources on better defined problems and identify emerging problems requiring formulation of new approaches.

#### *Increasing the range of services*

##### *Sow evaluation*

Inspecting cull sows at slaughter has been found to provide a useful aid in diagnosing herd problems which are amenable to control through changes to farm management or conditions (Geudeke *et al.*, 1992). Foot lesions and ulceration of the stomach were found to be the most common lesions, however, the lesion prevalence was found to vary substantially among herds. The monitoring system described is logistically possible and forms a useful adjunct for investigation of high sow culling rate problems.

##### *Infertility evaluation*

Evaluating reproductive failure by monitoring sows at slaughter has been strongly advocated by Almond and Richards (1992). They found that non-infectious causes of reproductive failure and urogenital infection could be easily confirmed at slaughter at minimal cost to the producer.

##### *"Cull grower" evaluation*

Inspection of groups of poor growing pigs, or "cull-pork pigs", from piggeries in South Australia has been performed on request from producers in addition to their regular inspections. Several herds have requested repeated inspections of cull pork pigs over many years.

In a recent SA study, combinations of related and unrelated problems were evident among 16 groups of cull-pork pigs. The cull-pork pigs had severe mycoplasma pneumonia (prevalence >70%) in 12 of the 16 groups. This was usually accompanied by pleurisy and to a lesser degree, pericarditis and peritonitis. Tail biting also appeared to be a major cause of culling in one group. Other conditions observed were proliferative enteritis, pleuropneumonia and severe mange.

Each of these 16 cull-pork inspections was "matched" with a routine inspection of baconer pigs from the same herds, and was conducted within two months of the cull-pork inspection. Statistical comparison of the average lung scores (ALS) between cull-pork and matched bacon pig groups found significantly greater ALS in 10 of the 16 cull-pork groups, frequently greater than ALS 20, when compared to the bacon pigs.

The demonstration of widespread lesions with the potential to severely impair growth in cull-pork groups supports the use of slaughter inspections in the investigation of these growth problems. A limitation on the interpretation of these results is that the age and clinical history of pigs inspected was not available. If these culled stock were still relatively young (ie., 14-18 weeks) when slaughtered, these results may indicate substantial disease problems in younger growing stock. However, if these pigs were relatively old (20-24 weeks), severe lesions observed may have contributed to their slow growth, but may not have been responsible for their initial setback in growth. As the composition of cull-pork groups may include young pigs slaughtered for salvage and older pigs which have been retained to avoid antibiotic residues following earlier parenteral treatment, knowledge of the clinical history and age of stock inspected is essential to interpret the results.

Severe mycoplasma pneumonia appears to play an important role in the cull-porker syndrome. This observation is based on the ALS being significantly higher in cull-pork groups from herds with underlying respiratory disease infection(s). Straw (1993) demonstrated clearly that pigs with severe lung lesions (score  $\geq 20$ ) performed poorly, even under optimum environmental and management conditions. While similar severe

mycoplasma pneumonia encountered in this study may not be a primary cause of cull-pork pigs, its underlying severity in these groups is, therefore, likely to be contributing to the subsequent poor growth. In view of this, clinicians would be well advised to consider treating poor growing pigs ("sick-pen" pigs especially) for mycoplasma pneumonia and secondary infections, irrespective of the primary cause of the growth check.

Greater confidence in the interpretation of the significance of these lesions could be obtained by comparison of lesion prevalence and severity found in cull-pork pigs with that of weight for age pork pigs (65-75 kg live-weight) from the same herd.

#### *International marketing opportunities for herd health monitoring schemes*

##### *Availability of PIGMON3.0™*

The upgraded version of PIGMON3.0™ has been released in Australia for use in the national PHMS program. Industry policy is to retain the rights to use of technology developed in Australia for a period of one year following its commercial release. Subsequently the program can be purchased from the Pig Research and Development Corporation. PIGMON3.0™ was released in May 1995. The QA manual and training/QA exercises can be arranged through the author.

##### *Availability of quality assurance support*

The consistently strong agreement between the Australian and US reference inspectors (Table 1) provides the basis for the standardized implementation of PHMS internationally. A North American QA exercise in which 55 veterinarians participated was reported by Davies *et al.*, (1993). Good agreement was achieved between veterinarians who had been trained by the reference inspector. Poorer agreement was found for pleurisy, although cases were generally mild. Nevertheless, these findings should be used to focus training programs for inspectors.

#### **Increasing the application of Pig Health Monitoring Schemes**

##### *Education of service providers*

The education of pig health service providers is pivotal to the application and reputation of the PHMS (Pointon *et al.*, 1995). Overstatement of the significance of results and inaccurate diagnoses must be guarded against through the education of field veterinarians, actively encouraging use of laboratory support and regular contact between the inspection service and field veterinarians.

##### *Training and quality assurance*

Underpinning the implementation of PHMS is the standardized classification of lesions and reporting of lesion data. The current Pig Research and Development Corporation project aims to implement these procedures to demonstrate that the approach is adaptable to differing regional conditions and resources, and at the same time produce data which is comparable internationally.

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## References

- ALMOND, G.W. and RICHARDS, R.G. (1992). Evaluating porcine reproductive failure by the use of slaughterchecks. *Compendium of Continuing Education for Practising Veterinarians*. 14:542-547.
- BAHNSON, P.B., POINTON, A.M., DIAL, D.G. and MARSH, W.E. (1992a). Prevalence of lesions at slaughter at a Minnesota swine slaughter plant. *Proceedings of the 12th International Pig Veterinary Society Congress*, The Hague, The Netherlands, p. 464.
- BAHNSON, P.B., POINTON, A.M., DIAL, D.G. and MARSH, W.E. (1992b). Prevalence of lesions at slaughter in Minnesota swine herds. *Proceedings of the 12th International Pig Veterinary Society Congress*, The Hague, The Netherlands", p. 485.
- CARGILL, C.F., DAVIES, P.R., CARMICHAEL, I., HOOKE, F. and MOORE, M. (1995). Treatment of sarcoptic mange in pigs with injectable Doramectin. *Veterinary Record*. (In press).
- CARGILL, C.F. and DOBSON, K.J. (1979). Experimental *Sarcoptes scabiei* infestation of pigs II. Effects on production. *Veterinary Record*. 104:33-36.
- CHAPPEL, R.J., PRIME, R.W., CUTLER, R.S., JONES, R.T., MILLAR, B.D. and ADLER, B. (1990). Antileptospiral antibodies in Australian pig farmers. *Medical Journal Australia*. 152:105.
- CHAPPEL, R.J., PRIME, R.W., MILLAR, B.D., MEAD, L.J., JONES, R.T. and ADLER, B. (1992). Comparison of diagnostic procedures for porcine leptospirosis. *Veterinary Microbiology*. 30:151-163.
- CHRISTENSEN, N.H. and CULLINANE, L.C. (1990). Monitoring the health of pigs in New Zealand abattoirs. *New Zealand Veterinary Journal*. 38:136-141.
- DAVIES, P.R., DIAL, G.D., MARSH, W.E. and BAHNSON, P.B. (1993). In "Feasibility of implementing a national swine slaughter monitoring system for the collection of data from American swine herds", eds P. Davies, G.D. Dial, W.E. Marsh and P.B. Bahnsen. (University of Minnesota: Saint Paul MN).
- DAVIES, P.R., MOORE, M.J. and POINTON, A.M. (1992). Sarcoptic mite hypersensitivity and skin lesions in slaughtered pigs. *Veterinary Record*. 12:390-392.
- DONADEU, M., SIMON, X., RUBIES, X., JOVELLAR, J., GIL, E., LUENGO, J. and PIJOAN, C. (1994). Serological study of *Actinobacillus pleuropneumoniae* and Aujeszky's Disease virus infection in multiple origin finishing farms. *Proceedings of the 13th International Pig Veterinary Society Congress*, Bangkok, Thailand, p. 48.
- ELBERS, A.R.W., TIELEN, M.J.M., SNIDJERS, J.M.A., CROMWIJK, W.A.J. and HUNNEMAN, W.A. (1991). Epidemiological studies on lesions in finishing pigs in the Netherlands. I. Prevalence, seasonality and interrelationship. *Preventative Veterinary Medicine*. 14:217-231.
- FLEISS, J.L. (1981). "Statistical methods for rates and proportions", p. 224, (Wiley Interscience: New York).
- GARCIA, R., PICHE, C., DAVIES, P. and GROSS, S. (1994). Prevalence of sarcoptic mange mites and dermatitis in slaughter pigs in North America and Western Europe. *Proceedings of the 13th International Pig Veterinary Society Congress*, Bangkok, Thailand, p. 250.
- GARDNER, I.A. and HIRD, D.W. (1990). Host determinants of pneumonia in slaughterweight swine. *American Journal of Veterinary Research*. 51:1306-1311.
- GAULANDI, G.L., BONI, P., VARISCO, G., PAIARO, E., GARCIA, R. and GROSS, S. (1994). Study of the prevalence of sarcoptic mange in pigs at slaughterhouses in major swine production areas in Northern Italy. *Proceedings of the 13th International Pig Veterinary Society Congress*, Bangkok, Thailand, p. 246.
- GEBHART, C.J., M'CORIST, S., LAWSON, G.H.K. and COLLINS, J.E. (1994). Specific in situ hybridisation of the intracellular organism of porcine proliferative enteropathies. *Veterinary Pathology*. 31:462-467.
- GEUDEKE, M.J., TIELEN, M.J.M., VERHEIJDEN, J. and HUNNEMAN, W.A. (1992). The use of slaughterhouse information in monitoring systems for herd health control in sows. *Proceedings of the 13th International Pig Veterinary Society Congress*, Bangkok, Thailand, p. 567.
- HENRY, S. (1995). Segregated Early Weaning and Multi-site production. *Proceedings of the Australian Association of Pig Veterinarians Programme, Australian Veterinary Association Conference*, Melbourne 1995, pp. 6-10.
- HURNIK, D. (1991). Incorporating slaughterhouse information. *Compendium of Continuing Education for Practising Veterinarians*. 13:1864-1867.
- HOLYOAKE, P.K., CUTLER, R.S. and CAPLE, I.W. (1994). A diagnostic dilemma: detecting proliferative enteritis in pigs at slaughter. *Australian Veterinary Journal*. 71:308-309.
- I-KUEI LIN, L. (1989). A concordance correlation coefficient to evaluate reproducibility. *Biometrics*. 45:255-268.
- JONES, G.F., DAVIES, P.R., ROSE, R., WARD, G.E. and MURTAUGH, M.P. (1993). Comparison of techniques for diagnosis of proliferative enteritis of swine. *American Journal of Veterinary Research*. 54:1980.
- JONES, R.T., MILLAR, B.D., CHAPPEL, R.J. and ADLER, B. (1987). Macroscopic kidney lesions in slaughtered pigs are an inadequate indicator of current leptospiral infection. *Australian Veterinary Journal*. 64:258-259.
- JUBB, K.V.F. and KENNEDY, P.C. (1963). "Pathology of Domestic Animals", pp. 139-159, (Academic Press: New York).
- LOMAX, L.G., GLOCK, R.D., HARRIS, D.L. and HOGAN, J.E. (1982). Naturally occurring proliferative enteritis: Pathologic and bacteriologic findings. *American Journal of Veterinary Research*. 43:1615-1621.
- LOVE, R.J., LOVE, D.N. and EDWARDS, M.J. (1977). Proliferative haemorrhagic enteropathy in pigs. *Veterinary Record*. 100:65-68.
- MERCY, A.R. (1992). In "The Western Australian Pig Health Monitoring Scheme", (Department of Agriculture, Western Australia).
- MERCY, A.R. and BRENNAN, C.M. (1988). The Western Australian pig health monitoring scheme. *Acta Veterinaria Scandinavica*. (Supplement) 84:212-214.
- MOUSING, J. (1988). Chronic pleurisy in pigs: The relationship between weight, age and frequency in three conventional herds. *Acta Veterinaria Scandinavica*. (Supplement) 84:253-255.
- NOYES, E.P., FEENEY, D.A. and PIJOAN, C. (1990). A comparison of antemortem and postmortem pneumonic lesions in swine using a noninvasive radiographic technique and slaughter examinations. *Journal of the American Veterinary Medicine Association*. 197:1025-1029.
- POINTON, A.M. (1989). Campylobacter associated intestinal pathology in pigs. *Australian Veterinary Journal*. 66:90-91.

- POINTON, A.M. and ARTHUR, R. (1995). In "Proceedings of Pig Meat Hygiene Program Workshop, Melbourne", ed. A. Pointon. (Pig Research and Development Corporation: Canberra).
- POINTON, A.M. and DAVIES, P.R. (1991). Evaluation of gross lesion data from monitoring slaughtered pigs. *Proceedings of the Epidemiology Chapter, Australian Veterinary Association Conference*, Sydney 1991, pp. 43-68.
- POINTON, A.M., DAVIES P.R., MERCY, A.R., BACKSTROM, L. and DIAL, G.D. (1992b). Disease Surveillance at Slaughter and Evaluation of Industry Trends. *Proceedings of Post Graduate Committee in Veterinary Science Refresher Course*, University of Sydney. 186:69-126.
- POINTON, A.M., FARRELL, M., CARGILL, C.F. and HEAP, P. (1987). A pilot pig health scheme for Australian conditions. *Proceedings of Post Graduate Committee in Veterinary Science Refresher Course*, University of Sydney. 95:743-777.
- POINTON, A.M., MERCY, A.R., BACKSTROM, L. and DIAL, G.D. (1992a). Disease surveillance at slaughter. In "Diseases of Swine, 7th edn", pp. 968-987, eds A.D. Leman, B.E. Straw, W.L. Mengeling, S. D'Allaire and D.J. Taylor. (Iowa State Press: Ames).
- POINTON, A.M., SKIRROW, S. and MOORE, M. (1995). Pig Health Monitoring Scheme: Interpreting results properly. *Proceedings of the Australian Association of Pig Veterinarians Programme, Australian Veterinary Association Conference*, Melbourne 1995, pp. 35-46.
- POINTON, A.M., SKIRROW, S., MOORE, M. and CARGILL, C.F. (1994). Monitoring disease trends in the Australian pig herd. *Proceedings of the 13th International Pig Veterinary Society Congress*, Bangkok, Thailand, p. 441.
- POINTON, A.M. and SLOANE, M. (1984). An abattoir survey of the prevalence of lesions of enzootic pneumonia of pigs in South Australia. *Australian Veterinary Journal*. 61:408-409.
- RANSLEY, R. and CLEARY, G. (1995). In "Pigstats94", (Pig Research and Development Corporation and Australian Pork Corporation: Canberra).
- ROWLAND, A.C. and LAWSON, G.H.K. (1975). Porcine intestinal adenomatosis: A possible relationship with necrotic enteritis, regional ileitis and proliferative haemorrhagic enteritis. *Veterinary Record*. 97:178-180.
- ROWLAND, A.C. and LAWSON, G.H.K. (1992). Porcine Proliferative Enteropathies. In "Diseases of Swine, 7th edn", pp. 560-569, eds A.D. Leman, B.E. Straw, W.L. Mengeling, S. D'Allaire and D.J. Taylor. (Iowa State Press: Ames).
- SHADBOLT, P.V., MITCHELL, W.R., BLACKBURN, D.J., MEEK A.H. and FRIENDSHIP, R.M. (1987). Perceived usefulness of the collection of subclinical and other disease entities detected at slaughter. *Canadian Veterinary Journal*. 28:439-445.
- STRAW, B.E. (1993). Performance measured in pigs with pneumonia and housed in different environments. *Journal of the American Veterinary Medical Association*. 198:627.
- STRAW, B.E., BACKSTROM, L. and LEMAN, A.D. (1986). Evaluation of swine at slaughter, Part 1 - The mechanics of examination, and epidemiologic considerations. *Compendium of Continuing Education for Practicing Veterinarians*. 8:541-548.
- THORNTON, E. (1995). Using three site approaches to control pleuropneumonia. *Proceedings of the Australian Association of Pig Veterinarians Programme, Australian Veterinary Association Conference*, Melbourne 1995, pp. 4-5.
- WALLGREN, P., MATTSO, S., ARTURSSON, K. and BOLSKJE, G. (1990). The relationship between *Mycoplasma hyopneumoniae* infection, age at slaughter and lung lesions at slaughter. *Proceedings of the 11th International Pig Veterinary Society Congress, Lausanne, Switzerland*, p. 82.
- WHITE, M.E.C. (1995). A clinical update of parasites in the pig. *The Pig Journal*. 33:41-53.
- WILLEBERG, P., GERBOLA, M.A., KIRKEGAARD PETERSEN, B. and ANDERSEN, J.B. (1984/5). The Danish pig health scheme: Nation-wide computer-based abattoir surveillance and follow-up at the herd level. *Preventative Veterinary Medicine*. 3:79-91.
- WILSON, T.M., CHANG, K., GEBHART, C.J., KURTZ, H.J., DRAKE, T.R. and LINTNER, V. (1986). Porcine proliferative enteritis: Serological, microbiological and pathological studies from three field epizootics. *Canadian Journal of Veterinary Research*. 50:217-220.

## A COMPARISON OF P2 BACKFAT MEASUREMENTS ON THE LIVE PIG, AT SLAUGHTER AND AT DISSECTION

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In the Australian pig industry the P2 backfat measurement is the standard for assessing carcass fatness. Different instruments are used to measure P2 in the live pig, at slaughter on the hot carcass, and at dissection on the chilled carcass. Anecdotal evidence also suggests that stretching of the hanging carcass may influence P2 measurement.

The AUSPIG model uses P2 to determine carcass fatness (Black *et al.*, 1986). As part of a larger study of the relationship between P2 and total body fat (to complete the characterisation of PIC genotypes in AUSPIG), a number of measures of P2 were obtained for 56 female Large White pigs ranging in live-weight from 10 to 185 kg (P2 range 3-34 mm). On the day prior to slaughter P2 was measured on-farm using a Meritronics Ultrasound system. At slaughter P2 was measured on the hot carcass using a Hennessy Grading Probe (HGP) and an Introscope, with the carcass both hanging and lying. On the day after slaughter P2 was measured on the hanging chilled carcass with the HGP and Introscope, and with callipers at dissection.

Table 1. Comparison of measurements of P2 backfat.

P2 measurement	Regression		
	Slope $\pm$ SEE	Significance <sup>†</sup>	r <sup>2</sup>
On-farm vs Dissection	0.846 $\pm$ 0.041	***	0.889
HGP vs Dissection	1.016 $\pm$ 0.078	NS	0.884
Introscope vs Dissection <sup>1</sup>	0.900 $\pm$ 0.032	**	0.935
HGP vs On-farm	1.284 $\pm$ 0.149	NS	0.772
<u>Pooled within-group regressions</u>			
Hanging vs Lying	0.960 $\pm$ 0.026	NS	0.968
Hot vs Cold	0.875 $\pm$ 0.021	***	0.977

<sup>†</sup>Significance of slope not being equal to 1; NS, not significant,  $P > 0.05$ ; \*\*  $P < 0.01$ ; \*\*\*  $P < 0.001$ . <sup>1</sup>In all cases the estimate of intercept was not significantly different from zero with the exception of Introscope vs Dissection (Intercept = 3.634  $\pm$  0.44).

The results (Table 1) indicate that on-farm measurements were significantly lower than at dissection, whereas HGP measurements were similar to those at dissection. Although the slope estimate of Introscope versus dissection was significantly less than unity, the large positive intercept resulted in Introscope P2 being greater than dissection P2 (for the P2 range in this study). The HGP measurements tended to be higher than those made on-farm, although this difference was not significant. There was no significant difference between the hanging and lying P2 measurements, but cold carcass P2 was significantly higher than hot carcass P2.

It was concluded that care should be taken when comparing on-farm P2 with slaughter and dissection P2, and that of the two instruments commonly used at slaughter, the HGP agreed most closely with dissection measurements.

### References

BLACK, J.L., CAMPBELL, R.G., WILLIAMS, I.H., JAMES, K.J. and DAVIES, G.T. (1986). *Research and Development in Agriculture*. 3:121-145.



## DETERMINATION OF FAT AND MOISTURE CONTENT IN SAUSAGES BY NEAR-INFRARED SPECTROSCOPY

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Measurements of fat and moisture contents in food commodities are routine tasks in laboratories of food manufacturers and government regulatory agencies. Standard methods for determining food composition are time consuming. Near-infrared (NIR) spectroscopy has potential for rapid determination of chemical composition of various kinds of food products (Williams and Stevenson, 1990). The objective of this study was to examine the accuracy of the NIR technique for measuring fat and moisture content in commercial sausages.

Twenty different brands of sausages were purchased from local supermarkets. They were minced, using a household mincer, stored in airtight containers at 4°C and analysed within 2 d. Fat content in the prepared samples was determined by the Soxhlet extraction method (AOAC, 1990), and moisture content was determined by oven-drying at 105°C for 16 h. For NIR analysis a single (20 g) sample of each brand of sausage was scanned; for chemical analyses duplicate or triplicate sub-samples were used.

Near-infrared reflectance spectra measurements were performed with a NIR scanning spectrophotometer (Model 6500, NIR System, Inc., Silver Spring, MD). The spectral data were recorded as  $\log 1/R$ , where R is the reflectance energy, at 2 nm intervals at the wavelength range of 400 to 2500 nm. A ceramic disk was used as reference. The spectral data in the NIR range (1100 to 2500 nm) were selected and correlated with the values of moisture and fat contents to generate calibration equations using the statistical models of multiple linear regression (MLR), principal component regression (PCR), partial least square regression (PLS) and modified partial least square regression (MPLS) (ISI, 1994).

The percentage range of moisture and fat content, and the correlation coefficients and standard errors of the calibration using different statistical models are presented in Table 1. The MPLS model gave the highest correlation coefficients for both moisture and fat contents ( $r^2=0.99$  and  $r^2=1.00$ , respectively).

**Table 1. Range of moisture and fat percentages in twenty different brands of sausages, and the correlation coefficients ( $r^2$ ) and standard errors of the calibration (SEC) using MLR, PCR, PLS or MPLS.**

	n	Range	MLR		PCR		PLS		MPLS	
			$r^2$	SEC	$r^2$	SEC	$r^2$	SEC	$r^2$	SEC
Moisture	20	53.3-64.8	0.87	1.03	0.98	0.39	0.92	0.82	0.99	0.33
Fat	20	12.0-26.2	0.86	1.46	0.99	0.32	0.97	0.63	1.00	0.26

The results show that NIR spectroscopy, using PCR or MPLS, is an accurate method for the determination of fat and moisture content in commercial sausages. The accuracy of the NIR method in determining protein, sodium chloride and sodium nitrite content in the sausages is currently being tested. The NIR spectroscopy has many advantages over the conventional methods, such as speed and that chemicals are not required, and it may become a very useful technique in food analytical laboratories.

### References

- AOAC (1990). "Official Methods of Analysis", 15th edn. (Association of Official Analytical Chemists: Virginia, USA).  
 ISI (1994). "NIRS 2, Routine Operation and Calibration Development Manual, Version 3.00". (Infrasoft International: Silver Spring, MD, USA).  
 WILLIAMS, P.C. and STEVENSON S.G. (1990). *Trends in Food Science and Technology*. 1:44-48.

## PIGMEAT EATING HABITS OF CHILDREN

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Little information is known about the pigmeat eating habits of children, with most studies of this type being concerned primarily with adults or those in their late teens. Accordingly, this lack of knowledge has resulted in little attention being given to producing pork based products for children. To provide a baseline set of data on the pigmeat products children are eating in the 1990s, a study has been conducted with 838 children aged between 5 and 16 years from high and low socio-economic groups in the Western Sydney region. Data was collected over 4 d during 1 week that included two weekdays and two weekend days. Each child was given a four-day diary, a cup to measure the quantity of minces and casseroles, and a ruler to measure the dimensions of pieces of meat eaten. Weight of meat eaten was calculated from calibrated measures and tables provided in English and Lewis (1991). Multiple regression analyses were used to assess the influence of age, gender and socio-economic status on the number of meals where meat and various types of meat were eaten, and the amount of meat eaten.

The results showed that there is little difference in the frequency of consumption of pigmeats, that include ham, bacon or pork, between 5 and 16 years of age. Regardless of gender, two meals per week was the average number of meals in which pigmeat was eaten. However, differences were found between females from high and low socio-economic backgrounds. Those from the high and low groups ate pigmeat on 3 and 1.5 occasions per week, respectively. As regards the amount of pigmeat eaten, socio-economic status and age influenced consumption by males to a much greater extent than females ( $P < 0.05$ ). Under the age of 10 years, males from the low socio-economic group consumed more pigmeat per week (325 g) than children of both sexes from the high socio-economic age group (125 g), and females from the under 10 years low socio-economic group (215 g). Similar amounts of pigmeat were eaten by 14 to 16 year-old males from both socio-economic groups (390 g), which was significantly higher ( $P < 0.05$ ) than the quantities recorded for females of all ages (215 g for 5 - 9 years; 269 g for 14 - 16 years). These latter values for females indicate that socio-economic status had no influence on the amount of meat eaten by females from 5 to 16 years of age. As regards the types of meats eaten, ham was by far the most commonly eaten pigmeat, being eaten at about 1.5 meals over all the children studied, with bacon the second most popular (about once per week), and pork chops a distant third (about once per month). Thus pork chops, roast and steak are eaten very infrequently by school-age children.

An important outcome of the study, was that a relatively stable pattern of eating pigmeats is established by 5 years of age with little change in the frequency and quantity of meat eaten until at least 14 - 16 years. These results, therefore, have significant ramifications for the marketing of pigmeats to children, since it is possible that there is only a narrow window of opportunity, namely from weaning through to less than 5 years, to influence which meats children will tend to eat in future years. Accordingly, there is an urgent need to develop new marketing strategies and products specifically for children during these formative years if the overall market for pigmeat is going to grow substantially both in volume and in value.

### References

- ENGLISH, R. and LEWIS, J. (1991). "Nutritional Values of Australian Foods." (Australian Government Publications: Canberra).

## pH DECLINE AND MEAT QUALITY IN RED AND WHITE MUSCLES OF PSE AND NORMAL PORK CARCASSES

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The occurrence of the pale, soft, exudative (PSE) condition in pork muscles is determined by the rate of pH and temperature decline post-slaughter. It is generally recognised that only 'white' muscles, which have a high proportion of white muscle fibres and rely on anaerobic metabolism, are susceptible to the PSE condition, whereas 'red' muscles, which rely on aerobic metabolism, contain a high proportion of red fibres, are generally not as susceptible. The aim of this experiment was to compare pH decline and quality characteristics of normal and PSE carcasses for major red and white muscles.

Ten Large White × Landrace pigs were slaughtered at 22 weeks of age (~99 kg live-weight), and pH was measured at 0.75, 1.25, 2, 6 and 24 h post-slaughter in two 'white' muscles, [*longissimus thoracis et lumborum* (LTL) in the loin and *semimembranosus* (SM) in the leg] and in two 'red' muscles [*triceps brachii* (TB) in the shoulder and *rectus femoris* (RF) in the leg]. Meat quality was assessed in the muscles at 24 h post-slaughter by measuring surface exudate (mg) and lightness (CIE-L\*) (Warner, 1994). Carcasses were classified PSE if the LTL exhibited surface exudate >100 mg and lightness >50 units (n=4), while the remaining carcasses were classified normal (n=6).

**Table 1. Meat quality measurements for LTL, SM, RF and RF for PSE and normal carcasses. Within each muscle there was a significant difference (P<0.05) between PSE and normal for all quality traits except for exudate in the TB and RF and pH differences in the LTL.**

Muscle Quality	LTL		SM		RF		TB		
	PSE	normal	PSE	normal	PSE	normal	PSE	normal	
Exudate (mg)	Mean	150	84	132	88	87	66	60	41
	SE	17	8	19	9	25	6	7	4
Lightness	Mean	51.9	47.4	48.9	45.4	46.3	43.2	45.5	41.9
	SE	0.2	1.2	0.6	1.3	1.5	1.1	1.1	0.6
pH at 24 h	Mean	5.67	5.87	5.66	5.70	5.89	5.94	5.86	5.98
	SE	0.02	0.08	0.02	0.01	0.04	0.03	0.03	0.03

At all measurement times post-slaughter, the LTL exhibited the lowest pH, the two red muscles had the highest pH while the SM was generally intermediate. For all muscles from PSE carcasses at 24 h post-slaughter, surface lightness values were higher and pH values lower than for normal carcasses, with greater differences in the white muscles. In addition, surface exudate values were higher in PSE carcasses than in normal carcasses for the white muscles. The LTL was used to classify the carcasses as PSE but the SM, RF and TB from PSE carcasses were within the acceptable range, ie., not PSE. It is generally recognised that only white muscles can exhibit the rapid rates of post-mortem glycolysis that cause protein denaturation and the subsequent less acceptable colour and exudate in PSE carcasses. The results show that in PSE carcasses, red muscles can exhibit a paler colour and there was also a tendency for higher exudate. In conclusion, the quality of both red and white muscles are detrimentally affected in carcasses exhibiting the PSE condition.

### References

WARNER, R.D. (1994). Physical properties of porcine musculature in relation to post-mortem biochemical changes in muscle proteins. PhD Thesis. University of Wisconsin.

## QUALITY ASSURANCE IN THE AUSTRALIAN PIG INDUSTRY

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A pilot quality assurance (QA) program aimed at reducing antibiotic residues in Australian pigmeat was started in early 1995. The program is an initiative of the Pig Research and Development Corporation, and is being co-ordinated by the Western Australian Department of Agriculture, in collaboration with Paul Higgins, a veterinarian from Victoria. The program is similar to the US Pork Producers Quality Assurance Program (Sundberg and Henry, 1995), but uses a quality standard to which producers will be audited by an independent third party. Australian producers will benefit from QA programs by reducing their costs of production, improving herd health, improving carcass consistency and quality, improving the competitiveness of pork and protecting the domestic and export markets from drug residues.

Phase 1 of the pilot program involved writing quality assurance manuals and procedures, developing the quality standards to which producers will be audited, and testing the program on pilot farms in Western Australia (n=21) and Victoria (n=10). Phase 2 of the program will involve training co-ordinators in the states of South Australia, New South Wales and Queensland, implementing the program on a pilot group of farms in those states and developing further technical and promotional information for the Australian pig industry. To date, a number of internal audits have been conducted on phase 1 pilot farms, which indicate that producers have a good understanding of the technical aspects of reducing antibiotic residues.

The quality standards that will be used in this quality assurance program are the Safe Quality Food 2000 standards (SQF 2000 Quality Code, 1995). These were developed by the Department of Agriculture, Western Australia for use by primary producers and small food manufacturers, based on the principles of the International Standards Organisation (ISO) 9000 series and the Hazard Analysis and Critical Control Point (HACCP) system. The SQF 2000 standard comprises six key elements of the ISO 9000 standards, as shown in Table 1. It is anticipated that the SQF 2000 standard will be nationally and internationally recognised, and is likely to be the "umbrella" under which many primary producers in Australia develop quality assurance programs.

**Table 1. Safe Quality Food 2000 elements.**

SQF 2000 elements	Details
Commitment	Policy statement, organisation structure, staff training
Suppliers	Purchasing, purchasing data
Control of production	Process control, corrective action, handling, storage packing & delivery
Inspection and testing	Inspection of product, equipment, internal audits
Quality records	Records of production process and inspections
Product identification	Product identification for tracing

Implementing the QA program on farms involves a number of steps, including the following: Training key personnel in QA, HACCP and SQF 2000 principles; identifying critical control points (in terms of antibiotic use on each farm); involving the veterinary consultant in developing protocols for antibiotic use on each farm; training workers in feed mixing, water medication and injecting pigs; designing simple, easy to use records. Checklists have been developed to help producers conduct on farm audits of the system. At the conclusion of the pilot program, farms will be audited by a professional auditing body, and random urine and tissue samples collected at slaughter for residue testing.

### References

- SQF 2000 QUALITY CODE (1995). (Agwest, Department of Agriculture: Perth).  
 SUNDBERG, P. and HENRY, S. (1995). *Proceedings of the Australian Association of Pig Veterinarians Programme, Australian Veterinary Association Conference, Melbourne 1995*, pp. 53-56.

## ASSESSMENT OF THE MAGNITUDE AND SIGNIFICANCE OF "BOAR TAIN" IN AUSTRALIA

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Boar taint presents as a distinct unpleasant smell, when fat or meat from entire mature boars is cooked. This odour is rarely detected in meat from either castrated or sexually immature males or females. Two major compounds responsible for causing taint are 5 $\alpha$ -androstenone (5 $\alpha$ ) and skatole (Sk). The project objective was to use the fat tissue concentration of these compounds to determine the potential incidence of taint.

The fat concentration of 5 $\alpha$  and Sk was assessed in 2 surveys by GC-ECD and HPLC respectively. Survey 1 (S1) involved approximately 110 Duroc and 110 Large White/Landrace crossbreed (LWL) boars and 60 LWL females. Survey 2 (S2) involved approximately 100 LWL boars. All animals were in the weight range of 44-144 kg. The mean 5 $\alpha$  and Sk concentration for both surveys is outlined in Table 1. In S1 a breed effect was apparent with 65% of Duroc having 5 $\alpha$  concentrations above the commonly accepted sensory threshold of 0.5 mg/g fat compared with 50% of LWL boars. When the boar data was divided into 33.3 percentiles of lowest, middle and heaviest weight groups for both surveys, it was apparent that 5 $\alpha$  concentrations rose with increasing weight. The lightest weight groups had a lower concentration and a lower percentage of animals with a 5 $\alpha$  concentration above the threshold compared with pigs of the heavier weight range ( $P < 0.05$ ) (Table 2). This trend of body-weight effect was not apparent for Sk.

**Table 1. The 5 $\alpha$ -androstenone and skatole concentrations (mean  $\pm$  SD) and the percentage of animals above the 5 $\alpha$  and Sk thresholds for the Duroc and the Large White/Landrace (LWL) crossbreed in surveys 1 (S1) and 2 (S2).**

Breed	5 $\alpha$ -androstenone			Skatole		
	n	mg/g	%>0.5 mg/g	n	mg/g	%>0.2 mg/g
S1 Duroc	98	0.91 $\pm$ 0.8	65.3	109	0.12 $\pm$ 0.1	15.6
S1 LWL	98	0.63 $\pm$ 0.5	50.0	107	0.11 $\pm$ 0.1	12.1
S1 Sows	58	0.18 $\pm$ 0.1	0.0	56	0.04 $\pm$ 0.03	0.0
S2 LWL	97	1.02 $\pm$ 0.8	63.7	102	0.11 $\pm$ 0.08	11.5

**Table 2. The 5 $\alpha$ -androstenone (5 $\alpha$ ) concentrations (mean  $\pm$  SD) and the percentage of total boars above the 5 $\alpha$  threshold when the boars in Surveys 1 (S1) and 2 (S2) were sorted into 33.3 percentiles according to live-weight.**

Percentiles (kg)	Study 1 (n=196)		Study 2 (n=97)	
	5 $\alpha$ (mg/g)	%>0.5 mg/g	5 $\alpha$ (mg/g)	%>0.5 mg/g
Lowest 3rd (44-87)	0.66 $\pm$ 0.66	46.9	0.90 $\pm$ 0.71	54.5
Middle 3rd (87-107)	0.80 $\pm$ 0.57	64.2	0.93 $\pm$ 0.64	65.0
Heaviest 3rd (107-144)	0.88 $\pm$ 0.76	60.7	1.30 $\pm$ 1.03	78.0

The results indicate that for boars up to 140 kg live-weight approximately 60% and 13% of carcasses had high 5 $\alpha$  and Sk concentrations respectively. Carcasses with high 5 $\alpha$  and/or Sk are likely to be offensive to consumers who are sensitive to either compound. This potential for boar taint will have implications on the further development of local and export markets and it would appear that the slaughter weight of Australian pigs could not increase without dramatically increasing the already high potential for taint.

## EFFECTS OF GENDER AND AGE AT SLAUGHTER ON THE QUALITY OF PIG MEAT

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For meat production, rearing of entire male pigs (boars) instead of castrated animals (barrows) is economically advantageous because of the anabolic effects of the male sex hormone, testosterone. Boars grow faster, use their feed more efficiently and produce leaner carcasses than barrows or gilts. However, commercial acceptance of boar carcasses is hampered by boar taint, an unpleasant odour associated with cooking boar meat. Boar taint is believed to be caused by the deposition in body fat of androstenone, a C19-steroid, and skatole, a metabolite of tryptophan (Hennessy and Wan, 1993). In this study the concentrations of androstenone and skatole were measured in the backfat of pigs of different ages and gender.

A total of 24 pigs, eight males, eight castrated males and eight females, were selected from three litters of Landrace and one litter of Large White pigs. Four pigs of each gender were slaughtered at 153 days of age (average body-weight 77 kg) and the remaining pigs were slaughtered at 198 days of age (average body-weight 112 kg). Samples of backfat were collected from each pig after slaughter and stored at -20°C until further analysis. Androstenone and skatole were extracted from fat using the procedures described by De Brabander and Verbeke (1986), and measured by gas chromatography and mass spectrometry (GC-MSD; Hewlett Packard Co. Ltd). Data were analysed by two-way analysis of variance followed by least significant difference test or Student's t-test.

Androstenone was detected in the backfat of four male pigs from two litters (one Landrace and one Large White). The concentrations were 0.48 µg/g and 0.51 µg/g in two 153-day-old pigs and 1.40 µg/g and 1.46 µg/g in two 198-day-old pigs. Skatole was detected in the backfat of all animals (Table 1). The concentrations were highest in the backfat from male pigs and lowest in the backfat from castrated pigs; the concentrations increased with age in all pigs.

**Table 1. Concentration (ng/g) of skatole in the backfat of male, castrated male and female pigs slaughtered at 153 or 198 days of age (mean ± SEM).**

Age at slaughter	Male	Castrated Male	Female
153 days	45.6 ± 8.0 <sup>a</sup> **	0.66 ± 0.12 <sup>b</sup> **	3.79 ± 0.53 <sup>c</sup> **
198 days	67.4 ± 12.0 <sup>a</sup>	1.63 ± 0.44 <sup>b</sup>	12.4 ± 1.48 <sup>c</sup>

<sup>abc</sup>Mean values in the same row with different superscripts are significantly different (P<0.05). \*\*Mean values in the same column are significantly different (P<0.01).

The results of the present study demonstrate that the concentration of androstenone and skatole in the body-fat is influenced by both gender and age. It is known that androstenone is synthesized in the testes and that its production is affected by the sexual maturity of the animal. Skatole is known to be produced in the intestine from microbial breakdown of tryptophan. The transportation pathway of skatole from intestine into body-fat is unclear. How the gender and age of the pig affect skatole deposition in the body-fat remains to be examined.

### References

- DE BRABANDER, H.F. and VERBEKE, R. (1986). *Journal of Chromatography*, 363:293-302.  
HENNESSY, D.P. and WAN, S.S. (1993). In "Manipulating Pig Production IV", pp. 155-161, ed. E.S. Batterham. (Australasian Pig Science Association: Attwood).

## EVALUATION OF PORK QUALITY DEFECTS

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Considerable variation in the incidence of pork quality defects occurs within and among abattoirs. Trout (1992) reported that the incidence of pale soft exudative (PSE) pork was 32% across four abattoirs, with a range from 0 - 62% among abattoirs. Variations may be the result of different genotypes and variations in practices including on-farm handling, transport and lairage conditions, stunning technique, carcass processing and chilling regime.

The National Pork Quality Improvement Program aims to reduce the incidence of PSE by 50% through the introduction of best practice standards for pre and post-slaughter management of pigs. Four abattoirs were audited over the period November to December 1994 to ascertain the incidence of meat quality defects, assist in identifying processes contributing to quality defects and suggest remedial measures to reduce quality defects. Further audits will be conducted to monitor changes in quality defect occurrence resulting from implementation of remedial action.

To determine the incidence of quality defects, pre and post-slaughter treatments of 1,116 pigs were monitored at four abattoirs (n=243, 253, 424 and 196). Carcass muscle pH was measured at two sites; loin (*M. Longissimus dorsi* at the last rib) and ham (*M. semimembranosus* adjacent to the *tuber ischii*). If pH was  $\leq 5.6$ , muscle paleness was measured using either the Colormet probe ( $L^*$ ) or the Fibre Optic Probe (FOP). Meat quality was described as PSE for pH  $\leq 5.6$  and  $L^* \geq 28$  or FOP  $\geq 45$ , reddish-pink soft exudative (RSE) for pH  $\leq 5.6$  and  $L^* < 28$  or FOP  $< 45$ , normal for pH between 5.6 and 6.0 and dry, firm and dark (DFD) for pH  $> 6.0$ . Carcasses were described as having extensive soft exudative (SE) meat if both loin and ham pH were  $\leq 5.6$ , and localised SE meat if pH  $\leq 5.6$  in either the ham or the loin. Colour was not used in the allocation of these descriptions. Normal carcasses had no SE meat and extensive and localised DFD carcasses had pH  $> 6.0$  at both or one site respectively. The incidence of meat quality defects for four abattoirs is shown in Table 1.

**Table 1. Percentage incidence of meat quality in four abattoirs.**

	Abattoir A	Abattoir B	Abattoir C	Abattoir D	Weighted mean
Extensive SE	15	26	17	36	23
Localised SE	26	34	25	28	28
Normal	30	19	39	26	30
Localised DFD	21	12	11	5	12
Extensive DFD	7	9	8	6	7

The rate of carcass processing and chilling appeared to have some effect on the incidence of SE meat. Relationships between transport distance and time and the incidence of SE meat are unclear. Future audits will be conducted to clarify these relationships.

### References

- Trout, G. (1992). In: "Meat Research Newsletter" 92/3. Pale Watery Pork. CSIRO, Division Of Food Processing, Meat Research Laboratory, Brisbane.

## EVALUATION OF MEAT QUALITY IN COMMERCIAL PIGS IN NEW ZEALAND

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Meat quality in pigs is determined by the breed of pig and the husbandry practices used before and after slaughter. There is some concern that appearance, palatability and manufacturing quality of pork in New Zealand may be declining. The two major meat quality problems encountered in pork are pale, soft and exudative (PSE) and dark, firm and dry (DFD) meat.

A study was conducted to determine the occurrence of PSE and DFD characteristics in carcasses of commercial pigs in New Zealand. Data were collected in one abattoir from 11 farms over 21 slaughter days. An Orion 250A portable pH meter was used to measure pH values in the loin at the level of the last rib (*musculus longissimus dorsi*) 45 min (pH<sub>45</sub>, 1840 carcasses) and 24 h (pH<sub>24</sub>, 574 carcasses out of 1840) after slaughter. Comparisons were made using a t-test based on the arcsine square-root transformation of the percentages for PSE and DFD.

Overall, 14% of the carcasses had PSE (pH<sub>45</sub><5.6) and 7% DFD (pH<sub>24</sub>>6.2) characteristics. A large variation in the incidence of PSE and DFD was observed among batches of pigs (one batch = pigs from one farm on one slaughter day) ranging from 0 to 29%, and 3 to 37%, respectively. The incidence of PSE was significantly higher ( $P<0.001$ ) in Landrace (24%), compared to Large White (10%) and crossbred pigs (13%) (Table 1). The high incidence in the Landrace pigs may be caused by the presence of the halothane gene in this breed (M. Skorupski, personal communication). No differences in the incidence of DFD were found among carcasses from crossbred (7%), Large White (6%), and Landrace (4%) pigs (Table 2).

**Table 1. Incidence (%) of PSE meat in crossbred and purebred pigs.**

Meat quality	n	PSE	Tends to PSE	Subnormal	Normal
		pH <sub>45</sub> <5.6	5.60-5.79	5.80-5.99	≥6.0
Crossbred	1331	13	17	32	38
Large White	289	10	16	28	46
Landrace	220	24	23	27	26

**Table 2. Incidence (%) of DFD meat in crossbred and purebred pigs.**

Meat quality	n	DFD	Tends to DFD	Normal	Acid Meat
		pH <sub>24</sub> >6.20	6.00-6.19	5.40-5.99	<5.40
Crossbred	409	7	9	83	1
Large White	87	6	13	81	1
Landrace	78	4	0	95	1

Transport and lairage time influenced the incidence of PSE. The highest incidence of PSE (22%, pH<sub>45</sub><5.6) occurred in carcasses from pigs which had travelled for more than 4 h and had spent less than 2 h in lairage. However, when the lairage time was extended to 3-7 h following a 4 h trip, the incidence of PSE significantly decreased to 11% ( $P<0.001$ ). After a transport time of less than 2 h, the lairage time (<2 h or 3-7 h) did not significantly influence the incidence of PSE (10% versus 14%). It is concluded that the PSE and DFD conditions occur in New Zealand pork. The results suggest that the incidence of PSE and DFD may be reduced by improving the genotype and the environment prior to slaughter.



## THE EFFECTS OF ADVERSE HANDLING OF PIGS ON FARM AND AT THE ABATTOIR ON MEAT QUALITY

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The majority of the studies investigating the effects of handling pigs on meat quality have examined handling during transportation from the farm to the abattoir and at the abattoir. This study investigated the influence of handling pigs on farm and pre-slaughter handling at the abattoir on subsequent meat quality.

Thirty-six crossbred (Large White  $\times$  Landrace) boars were randomly allocated to treatments. The treatments were (a) positive (involved patting the pig) and negative (involved shocking the pig with an electric prod) handling imposed 2 min/day for 5 weeks on-farm and (b) minimal (minimal force) and negative (10 shocks with electric prod) handling which were imposed at the abattoir between pen and stunning.

The pigs negatively handled on farm had lower muscle glycogen at 5 min and 40 min post-slaughter, and a lower  $\text{pH}_u$  in the *Longissimus thoracis* (LT) than positively handled pigs, however there were no differences in 24 h muscle glycogen, surface lightness ( $L^*$ ), % drip loss or % PSE (drip loss  $>5\%$  &  $L^* >50$ ) of the LT muscle. While there were no significant interactions between on-farm and abattoir handling procedures on meat quality, pigs that were negatively handled at the farm and at the abattoir had lower muscle glycogen,  $\text{pH}_u$ , higher % drip loss and higher %PSE.

**Table 1. Effect of farm and abattoir handling treatments on muscle glycogen, pH and meat quality indicators in the *Longissimus thoracis*. POS = positive handling; NEG = negative handling; MIN = minimal handling.**

Farm Treatment (F)		POS		NEG		SED	Significance <sup>1</sup>	
		MIN	NEG	MIN	NEG		F	A
Abattoir Treatment (A)								
Glycogen (mg/g)	5 min	10.08	9.49	8.46	7.55	0.55	**	ns
	40 min	7.24	7.53	6.28	4.95	0.66	**	ns
	24 h	1.73	1.19	1.91	0.70	0.38	ns	*
Surface Lightness ( $L^*$ )		50.01	48.31	49.64	50.33	2.84	ns	ns
% Drip loss		4.70	4.07	3.90	6.10	1.28	ns	ns
Ultimate pH		5.60	5.58	5.49	5.48	0.04	**	ns
% PSE		13	22	25	34	-	ns	ns

<sup>1</sup> \*  $P < 0.05$ ; \*\*  $P < 0.01$ ; ns not significant. <sup>2</sup> Chi-square test used.

These data show that negative handling on-farm or at the abattoir can influence the post-slaughter levels of muscle glycogen although there were no effects on meat colour or drip loss. Also, pigs which were handled negatively on-farm and at the abattoir had inferior meat quality. This experiment has demonstrated the potential for handling procedures on-farm and at the abattoir to influence meat quality.

## GENETIC RELATIONSHIPS BETWEEN CARCASS COMPOSITION AND MEAT QUALITY IN AUSTRALIAN PIGS

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To be successful in today's markets the meat quality and carcass composition of pork carcasses must be of a high standard. Most selection programs consider leanness as the optimum characteristic of the carcass and neglect meat quality. In doing so this may lead to problems of poor pork quality. The inclusion of carcass composition and meat quality in a breeding program requires knowledge of the genetic correlations between these characteristics. The aim of this study was to estimate genetic parameters for carcass composition and meat quality in Australian Large White and Landrace pigs.

Carcass composition is described by estimated lean meat yield (LEAN%) and ham lean weight (HAM). The LEAN% was derived using the following prediction equation:  $LEAN\% = 64.2704 + 0.1090 SHCW - 1.0231 FDP2$  (Ferguson *et al.*, 1994) in which SHCW is the standardized hot carcass weight and FDP2 is the fat depth at P2. The HAM consists of the derinded, defatted and slash boned back leg excluding the hock muscles. These carcass traits were analysed using a derivative free Restricted Maximum Likelihood procedure (Meyer, 1993) fitting an animal model. The fixed effects in the model were hot carcass weight, recording date and breed. A description of the meat quality traits, consisting of pH measured 45 min after slaughter in the *longissimus dorsi* muscle (pH<sub>45</sub>), colour of the *longissimus dorsi* muscle (CLD) and drip loss percentage (DLP) together with the other methods used is given in Hermesch *et al.* (1995).

**Table 1. Number of records and heritabilities for lean meat yield and ham weight together with their genetic correlations with meat quality traits.**

	Records n	Heritabilities		Genetic correlations between carcass and meat quality traits <sup>a</sup>			
		h <sup>2</sup>	SE	HAM	pH <sub>45</sub>	CLD	DLP
LEAN%	2249	0.48	0.06	0.68	-0.53	0.13	0.20
HAM	2094	0.46	0.06		-0.48	0.33	0.40

<sup>a</sup>Range of standard errors (SE) for genetic correlations: 0.03 - 0.10.

Both carcass traits are highly heritable (Table 1). The negative correlations between LEAN% and pH<sub>45</sub> and between HAM and pH<sub>45</sub> indicates that selection for higher meat content of the carcass will lead to a lower pH<sub>45</sub>. The unfavourable genetic correlations between LEAN% and HAM with ultimate meat quality traits CLD and DLP are of lower magnitude for LEAN% than for HAM. Selection for higher LEAN% and higher HAM will lead to a lighter colour of the meat associated with a higher DLP. These results indicate that selection for improved LEAN% will have smaller negative effects on ultimate meat quality traits than selection for higher ham lean weight.

### References:

- FERGUSON D.M., CHANDLER, R.C., MAYNARD, P. and THOMAS, M. (1994). "The validation of equations for the prediction of lean meat yield in pig carcasses." PRDC Final Report, Project LMA6.P. (Pig Research and Development Corporation: Canberra).
- HERMESCH, S., LUXFORD, B., and GRASER H.-U. (1995). *Proceedings of the 11<sup>th</sup> Conference of the Australian Association of Animal Breeding and Genetics*, Adelaide, 1995, pp. 631-634.
- MEYER, K. (1993). "DFREML User Notes V.2.1." (Animal Genetics and Breeding Unit, UNE: Armidale).

## EFFECTS OF DIETARY VITAMIN E ON POST-MORTEM MUSCLE QUALITY TRAITS OF PIG

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Pigs which are homozygous positive for the 'hal' gene have an abnormality in their anti-oxidant defense mechanisms which is reported to be alleviated by supplementation of the diet with vitamin E (Duthie *et al.*, 1989). Dietary vitamin E supplementation is also reported to reduce drip loss in pork (Monahan *et al.*, 1994) and prevent the occurrence of the pale soft exudative (PSE) condition. The objective of the present experiment was to investigate the effects of dietary vitamin E supplementation on post-mortem muscle quality traits.

Nine 'hal' positive PIC pigs were offered either control (0 IU supplemented vitamin E/kg of feed; n=3 as one pig died) or vitamin E (200 IU supplemented vitamin E/kg of feed; n=5) diets from 4 weeks of age until slaughter at commercial carcass weight (~78 kg). After slaughter the muscle *longissimus thoracis et lumborum* (LTL) was removed. At 6 h post-mortem muscle samples (50 g) were suspended at 4°C in sealed bags for 0, 0.5, 1, 2, 3, 6 and 9 d post-mortem. At each time period, drip loss, cooking loss, total protein solubility, and lipid oxidation (TBARS) were assessed, as described by Warner (1994). Meat quality measurements of colour and pH were assessed at 24 h post-mortem, and muscle pH was measured regularly up to 24 h post-mortem (Warner, 1994).

All carcasses were classified PSE, which was assessed by low pH (pH<5.5) in the LTL at 1 h post-mortem, and by extreme paleness ( $L^*=58.3 \pm 1.1$ ) and high exudation ( $8.9 \pm 0.2\%$  drip loss over 48 h) in the LTL at 24 h post-mortem. Vitamin E treated pigs exhibited lower muscle lipid oxidation post-mortem than control pigs ( $0.15 \pm 0.03$  vs  $0.35 \pm 0.04$ ;  $P<0.05$ ) but no other differences between dietary treatments were evident.

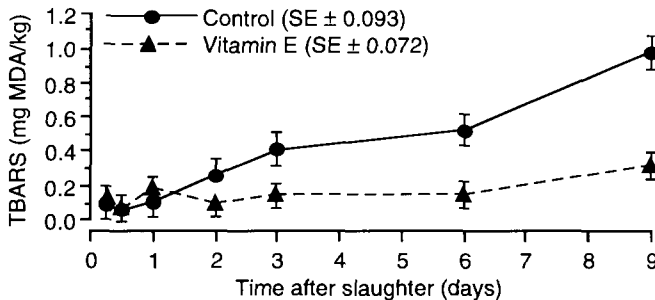


Figure 1. Changes in lipid oxidation (TBARS) with time after slaughter.

There was a significant interaction ( $P<0.05$ ) between diet and days of suspension post-mortem. Vitamin E treated samples had similar lipid oxidation during suspension, except for a significant increase at day 9 ( $P<0.05$ ), whereas lipid oxidation in control samples increased after 2 d of suspension ( $P<0.05$ ) and continued to increase (Figure 1). Drip loss and total protein solubility increased ( $P<0.05$ ) from 0 to 9 d post-mortem (6.0 to 14.6%; 75.1 to 104.5 mg of protein/g of muscle, respectively) whereas cook loss declined ( $P<0.05$ ) over the same period (31.9 to 20.7%). In conclusion, dietary supplementation with 200 IU of vitamin E/kg of feed reduced lipid oxidation but had no major effects on water loss post-mortem and failed to prevent the PSE condition.

### References

- DUTHIE, G.G., ARTHUR, F.M. and WALKER, M. (1989). *Research in Veterinary Science*. 46:226-230.  
 MONAHAN, F.J., GRAY, J.I., ASGHAR, A., HAUG, A., STRASBURG, G.M., BUCKLEY, D.J. and MORRISSEY, P.A. (1994). *Journal of Agriculture and Food Chemistry*. 42:59-63.  
 WARNER, R.D. (1994). Physical properties of porcine musculature in relation to post-mortem biochemical changes in muscle proteins. PhD Thesis. University of Wisconsin.

## AUTHOR INDEX

Ahrens, F.	39
Adler, B.	231
Aherne, F.X.	129
Allen, J.G.	196
AL-Matubsi, H.Y.	138
Andersen, L.M.	38
Anderson, J.D.G.	24
Atwood, C.S.	94
Atyeo, R.F.	186
Auldist, D.E.	114, 137
Ayonrinde, A.I.	179, 180
Bach Knudsen, K.E.	190
Baker, J.	29, 30, 31, 32
Banhazi, T.	223, 224
Barnett, J.L.	22, 81
Barr, A.R.	32
Barry, T.N.	37
Batterham, The Late E.S.	33
Bent, M.J.M.	62
Bikker, P.	187
Billinghurst, A.	52
Black, J.L.	225
Blackall, P.J.	226
Bodin, J.C.	26, 28
Boisen, S.	40
Borg Jensen, B.	190
Bornholdt, U.	39
Bradley, L.R.	249
Bray, H.J.	225
Brewster, C.J.	189
Butler, K.J.	193
Buwalda, T.R.	181
Cadogan, D.J.	189
Cameron, R.D.A.	91
Campbell, R. G.	1, 81, 83, 131, 138, 174, 175, 176, 177, 178 187, 189, 193, 194
Cargill, C.	223, 224
Carlson, D.	137
Casey, K.D.	202
Catt, S.L.	88
Cegielski, A.C.	129
Chappel, R.J.	231
Chappell, D.S.	135
Chen, J.	40
Chin, J.	227, 228, 229
Choct, M.	29, 30, 31, 34
Clarke, I.J.	86
Clowes, E.C.	129
Connaughton, I.D.	230
Cranwell, P.D.	174, 175, 176, 177, 178
Cronin, G.M.	20
Currie, E.	201
D'Souza, D.N.	252, 258
Darragh, A.J.	132
Davies, P.R.	171
Delaney, S.	228

Dial, G.D. ....	23
Djordjevic, S.P. ....	227, 228, 229
Donham, K.J. ....	203
Dryden, G. McL. ....	91
Dunaiski, V. ....	86
Dunshea, F.R. ....	36, 42, 83, 86, 258
Eamens, G.J. ....	227, 228, 229
Eason, P.J. ....	133
Eggum, B.O. ....	39
Eldridge, The Late G.A. ....	256, 258
Evans, R. ....	249
Evans, G. ....	87, 88
Fagan, P. ....	229
Fairclough, R.J. ....	138
Farrell, D.J. ....	33
Forsyth, W.M. ....	230
Foss, D.L. ....	171
Francis, G.L. ....	83
Gannon, N.J. ....	27, 36
Gardner, E.A. ....	202
Gaughan, J.B. ....	91, 198
Giles, L.R. ....	38, 225
Goddard, C. ....	86
Gooden, J.M. ....	225
Granzin, B.C. ....	198
Graser, H-U. ....	259
Grela, E. ....	185
Hampson, D.J. ....	139, 170, 186, 226
Handley, G. ....	127
Harrison, D.T. ....	174, 175, 176, 177, 178, 189, 193
Hartley, D.G. ....	257
Hartmann, P.E. ....	94, 130, 172
Hasse, D. ....	171
Hastrup, T. ....	194
Head, The Late R.H. ....	134
Hemsworth, P.H. ....	81, 89
Hennesy, D.P. ....	254
Hermesch, S. ....	259
Higgins, P. ....	253
Hodgkinson, S.M. ....	35
Holyoake, P.K. ....	23, 171
Hooper, J. ....	196
Hosking, B. ....	85
Hughes, P.E. ....	89, 90, 92, 131
Hutchinson, I. ....	251
Hutson, G.D. ....	21
Inbarr, J. ....	190
Irwin, D.P. ....	199
Jackson, D.A. ....	26
Jackson, A.G. ....	222
Jakobsen, K. ....	190
Jerome, D. ....	260
Joergensen, E. ....	7
Jones, G.F. ....	171
Jones, M.R. ....	84
Jongbloed, A.W. ....	191, 195
Jorgensen, H. ....	39
Karabinas, V. ....	187

Karlsson, B.W.	173, 182
Kauffman, R.G.	260
Kemme, P.A.	191, 195
Kennaugh, L.M.	94, 130
Kershaw, S.S.	131, 189
Kerton, D.J.	36
Keys, J.R.	84
Kiela, P.	182
Kies, A.K.	28, 191, 195
King, A.K.	127
King, R.H.	93, 114, 127, 131, 133, 137
King, V.L.	23
Knowles, H.M.	256, 258
Kopinski, J.S.	197
Krasucki, W.	185
Laing, D.G.	251
Lee, C.Y.J.	255
Leury, B.J.	36, 252, 258
Lo, Y.Y.T.	255
Luxford, B.G.	259
Ma, L.	174, 175, 176, 177, 178
Maillard, R.	26
Mäkinen, M.	191, 195
Manickam, S.	85
Mao, Y.L.	184
Marsh, W.E.	23
Masterman, N.	223, 224
Mattsson, I.	173
Maxwell, W.M.C.	87, 88
Maynard, P.	256
McCauley, I.	52
McCauley, R.	179, 180
McGahan, E.J.	202
McGuigan, K.	197
McKenzie, C.	85
Meads, N.	192
Moore, G.	251
Moore, G.A.	200, 201
Morel, P.C.H.	24, 40, 181, 192, 257
Morgan, P.O.	52
Morley, W.	83
Morrish, L.	133, 137
Mosenthin, R.	39
Moughan, P.J.	25, 35, 37, 40, 132, 192
Mroz, Z.	185, 191, 195
Mubiru, J.N.	183
Mullan, B.P.	128, 135, 136, 170, 179, 180, 196, 199
Murtaugh, M.P.	171
O'Brien, J.K.	88
O'Halloran, J.H.	252
Officer, D.I.	38
Oram, N.	251
Owen, J.	251
Owens, J.A.	83
Owens, P.C.	83, 86
Panaccio, M.	171
Paterson, R.A.	230
Paterson, A.M.	253

Payne, H.G.	19
Pearson, G.	40, 181, 192, 257
Pedersen, B.K.	7
Pengelly, A.	256
Pethick, D.W.	170
Pettigrew, J.E.	101, 119
Philip, G.	90
Pierzynowski, S.G.	173, 182
Pluske, J.R.	41, 75, 129, 170
Pointon, A.M.	232
Prawirodigdo, S.	36
Prestegar, N.	188
Quinn, K.J.	83
Ranford, J.L.	128, 135, 136, 196
Rantzer, D.	182
Rapp-Gabrielson, V.J.	226
Rasmussen, P.B.	194
Reeds, P.J.	27
Revell, D.K.	41, 75, 128, 135, 136, 199
Robinson, S.J.	87
Romalis, L.	228
Rose, G.	251
Ruby, V.	7
Rutherford, S.M.	25, 35
Ryan, P.	253
Sali, L.	254
Salvatore, L.	254
Sauer, W.C.	39
Scarman, A.	228, 229
Schirmer, B.N.	20
Schollum, L.M.	181
Selle, P.H.	193
Siba, P.M.	170
Simmins, P.H.	28
Simpson, G.J.	20
Siswadi, R.	92
Skirrow, S.Z.	196, 223, 224, 253
Skou Jensen, M.	190
Smits, R.J.	128, 135, 136, 188, 199
Stevenson, B.J.	24
Svensden, J.	182
Szarvas, J.	29, 30, 31, 32
Tarvid, I.	174, 175, 176, 177, 178
Taylor, V.	228
Taylor, I.A.	22
Thaela, M-J.	173, 182
Thompson, M.J.	94, 172
Tilbrook, A.J.	89
Toussaint, J.	133
Trigg, T.	23
Tritton, S.M.	131, 138
Trott, D.J.	139, 226
Tso, M.Y.W.	184
Turner, A.I.	89
van Barneveld, R.J.	29, 30, 31, 32, 34, 36
van Laar, H.	187
Vavala, R.	176
Verstegen, M.W.A.	187

---

Wakeford, C.M.....	137, 172
Waldron, D.....	254
Walker, M.J.....	227, 229
Walsh, R.....	254
Walton, P.E.....	42, 86
Wang, T.....	82, 183
Warner, R.D.....	252, 256, 258, 260
Westbrook, S.L.....	52
Weström, B.R.....	173
Whitaker, A.....	194
Wigan, G.C.....	33
Williams, I.H.....	107, 128, 129, 134, 135, 136, 179, 180, 188
Williams, P.E.V.....	26, 28
Williams, S.R.O.....	200, 201
Wynn, P.C.....	84, 225
Xu, R.J.....	82, 183, 184, 250, 255
Yeung, Y.H.....	250
Yu, F.....	37
Zak, L.J.....	129
Zalunardo, M.....	227