

MANIPULATING PIG PRODUCTION II

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Editors: J.L. Barnett and D.P. Hennessy

AUSTRALASIAN PIG SCIENCE ASSOCIATION
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CONTENTS

CONTRIBUTORS	viii
ACKNOWLEDGMENTS	xiii
PREFACE	xiv

MEAT QUALITY

REVIEW: The effects of handling, transport, slaughter and chilling on meat quality and yield in pigs	1
<i>V. Tarrant.</i>	

CONTRIBUTED PAPERS: Evaluation of near infra-red (NIR) spectrophotometry as a method for determining the nitrogen and energy content of pig carcasses	26
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S.A. Beech, B. McAlpine, R. Elliott and E.S. Batterham.

The development of immunoassays for antibiotic residues in foodstuffs ...	27
<i>M. Smith and J. Huglin.</i>	

Effect of amount of feeding on persistence of dieldrin residues in pigs ...	28
<i>G. Takken and H. Mawhinney.</i>	

Restriction fragment length polymorphism (RFLP) in the detection of porcine stress syndrome (PSS) susceptible and carrier pigs	29
--	----

I.P. Hughes, C. Moran, F.W. Nicholas and W. Davies.

SYMPOSIUM: Factors around slaughter affecting the quality of pig meat. Introduction	30
<i>R.F. Thornton.</i>	

Growth promotants, repartitioning agents and pig meat quality	31
<i>R.F. Thornton and W.R. Shorthose.</i>	

Microbiological contamination of pig carcasses	38
<i>I.R. Morgan and F.L. Krautl.</i>	

Extending the storage life of chilled pork	46
<i>B.J. Shay and A.F. Egan.</i>	

Factors affecting the manufacturing properties of pig meat	56
<i>R.G. Campbell, D.R. Smith, J.M. O'Shea, J. Dirnbauer and B.G. Luxford.</i>	

Symposium conclusion	61
<i>R.F. Thornton.</i>	

CONTRIBUTED PAPERS: An assessment of the Hennessy grading probe for use in pig carcass classification	66
<i>W.C. Smith, G. Pearson and R.W. Purchas.</i>	

Comparative growth performance, carcass composition and meat quality of pigs sired by Duroc, Hampshire, Landrace and Large White boars	67
<i>W.C. Smith, R.W. Purchas and G. Pearson.</i>	

Salbutamol (β -agonist) and muscle fibre type in pigs	68
<i>N. Oksbjerg, A. Blackshaw and J. Fernandez.</i>	

The effect of exogenous growth hormone administration on pig meat quality	69
<i>R.D. Warner and R.H. King.</i>	

Effect of porcine somatotropin on non-esterified fatty acid glycerol kinetics in growing barrows	70
--	----

F.R. Dunshea, D.M. Harris, P.S. McNamara, D.E. Bauman, R.D. Boyd and A.W. Bell.

<i>CONTRIBUTED PAPER: Meat quality of pigs fed cimaterol</i>	71
<i>R.F. Thornton, D. Adamson, P.V. Harris, W.R. Shorthose and K.C. Williams.</i>	

THE YOUNG PIG

<i>REVIEW: Sow lactation</i>	72
<i>P.E. Hartmann and M.A. Holmes.</i>	
<i>CONTRIBUTED PAPERS: Pattern of milk production in sows</i>	98
<i>R.H. King, M.S. Toner and H. Dove.</i>	
Within litter variation in milk intake during suckling by piglets	99
<i>N.A. Smith and P.E. Hartmann.</i>	
Digestion of lactose and absorption of galactose and glucose in nursing piglets	100
<i>P.H. Bird, S.J. Morris and P.E. Hartmann.</i>	
<i>SYMPOSIUM: Neonatal mortality in the pig.</i>	
Introduction	101
<i>G.M. Cronin.</i>	
Neonatal mortality: The influence of the structural environment	102
<i>S.H. Baxter.</i>	
Neonatal mortality: The influence of maternal behaviour	110
<i>G.M. Cronin.</i>	
Neonatal mortality: The influence of lactation on piglet survival	116
<i>P.E. Hartmann, P.H. Bird and M.A. Holmes.</i>	
Neonatal mortality: The influence of management	122
<i>R.S. Cutler, E.M. Spicer and R.W. Prime.</i>	
Neonatal mortality: Conclusions	127
<i>G.M. Cronin.</i>	

THE GROWING PIG

<i>CONTRIBUTED PAPERS: The effect of vitamin E supplementation on the performance of growing pigs</i>	135
<i>Y.H. Wang and J. Leibholz.</i>	
The effects of folic acid supplementation on the performance of growing pigs	136
<i>N.J. Gannon and J. Leibholz.</i>	
Citric acid supplementation of creep-weaner diets	137
<i>W.A. Clarke and E.S. Batterham.</i>	
<i>SYMPOSIUM: Diet and management of weaner pigs. Introduction</i>	138
<i>K. Hutton.</i>	
Biological limitations imposed by the digestive system to the growth performance of weaned pigs	140
<i>P.D. Cranwell and P.J. Moughan.</i>	
Alternative feeding strategies for weaner pigs	160
<i>I.G. Partridge.</i>	
The nutritional management of weaner pigs	170
<i>R.G. Campbell.</i>	
Symposium conclusion	176
<i>K. Hutton.</i>	

<i>CONTRIBUTED PAPERS: Monosodium glutamate as a flavour enhancer in creep-weaner diets for piglets</i>	184
<i>W.A. Clarke and E.S. Batterham.</i>	
Protease inhibitors in cereals for pigs	185
<i>H.S. Saini and E.S. Batterham.</i>	
Utilization of ileal digestible lysine from different protein sources by growing pigs	186
<i>E.S. Batterham, L.M. Andersen and D.R. Baigent.</i>	
Criteria for assessing the requirements and availability of phosphorus in growing pigs	187
<i>P.P. Ketaren, E.S. Batterham, E.B. Dettmann and D.J. Farrell.</i>	
Retention of ileal digestible lysine by growing pigs	188
<i>E.S. Batterham, L.M. Andersen, D.R. Baigent and E. White.</i>	
Effects of cimaterol on pig growth and nutrient digestibility	189
<i>K.C. Williams, A.R. Neill, R.T. Peters and R.F. Thornton.</i>	
An evaluation of microwave-treated soybeans using laboratory rats	190
<i>J. Xian, R. Gerdes and D.J. Farrell.</i>	

BEHAVIOUR AND ENVIRONMENT

<i>REVIEW: Designing the pig pen</i>	191
<i>S.H. Baxter.</i>	
<i>CONTRIBUTED PAPERS: Energy expenditure in pigs: A new technique</i>	207
<i>L.R. Giles, J.M. Gooden, R.G. Tucker, E.F. Annison and J.L. Black.</i>	
Energy expenditure of pigs exposed to high temperature	208
<i>L.R. Giles, J.M. Gooden, R.G. Tucker, E.F. Annison and J.L. Black.</i>	
Rectal temperature of pigs exposed to high temperature	209
<i>L.R. Giles, E.S. Batterham and J.L. Black.</i>	
Lying-down behaviour of loose-housed sows during day 1 and day 8 after parturition	210
<i>J.K. Blackshaw.</i>	
The physiological and behavioural responses of pigs to differing designs of individual accommodation	211
<i>J.L. Barnett.</i>	
Physiological response to exercise in pigs	212
<i>S.H. Zhang, D.P. Hennessy and P.D. Cranwell.</i>	

BEHAVIOUR, ENVIRONMENT AND GENETICS

<i>CONTRIBUTED PAPERS: Operant responding by sows on restricted rations for additional food</i>	213
<i>G.D. Hutson.</i>	
Testing maternal behaviour in the pig: Responses to visual and tactile stimuli from a model piglet	214
<i>G.D. Hutson, J.L. Wilkinson and B.G. Luxford.</i>	
The pre-farrowing behaviour of sows and gilts with access to space	215
<i>M.J. Haskell and G.D. Hutson.</i>	

<i>SYMPOSIUM: Genetic selection: Which way forward?</i>	
Introduction	216
<i>M.E. Goddard.</i>	
Use of BLUP in selection for growth rate and litter size	217
<i>T. Long.</i>	
Practical experience with PIGBLUP	222
<i>A.R. Fyfe.</i>	
Performance testing and selection for efficient lean growth	225
<i>C.P. McPhee.</i>	
Methods and success of selection for litter size	229
<i>D.A. Treacy.</i>	
Symposium conclusion	234
<i>M.E. Goddard.</i>	

HEALTH

<i>SYMPOSIUM: The effectiveness of current pig vaccines.</i>	
Introduction	237
<i>J.C. Chin.</i>	
The role of pig vaccines in the host-micro-organism equation	238
<i>J.C. Chin.</i>	
Enzootic pneumonia: Recent advances and the future	242
<i>R.F. Sheldrake.</i>	
Vaccines and the control of swine dysentery	246
<i>D.J. Hampson.</i>	
Swine erysipelas vaccines	249
<i>G.J. Eamens.</i>	
The role and effectiveness of antileptospiral vaccines in pigs	256
<i>R.J. Chappel, B. Adler, R.S. Cutler, R.T. Jones and B.D. Millar.</i>	
Porcine proliferative enteropathies	260
<i>R.P. Gogolewski, R.W. Cook, E.S. Batterham and J.C. Chin.</i>	
<i>Streptococcus suis</i> in Australia	263
<i>R.P. Gogolewski, R.W. Cook and J.C. Chin.</i>	
Symposium conclusion	265
<i>J.C. Chin.</i>	
<i>CONTRIBUTED PAPERS: Vaccination against <i>Treponema hyodysenteriae</i>:</i>	
Artificial and natural challenge results	273
<i>P.J. Coloe, N.L. Gerraty and H. Geldard.</i>	
A vaccine to prevent parvovirus disease in pigs	274
<i>J. Bates.</i>	
Biochemical, serological and pathological studies of Australian isolates of <i>Actinobacillus pleuropneumoniae</i>	275
<i>L.E. Eaves, P.J. Blackall, R.J. Rogers, M. Fegan and K.F. Trueman.</i>	
Effects of the MHC on production and disease resistance in pigs	276
<i>F.W. Nicholas, M.J. Baglin, S.C. Brown, I.P. Hughes, R.J. Love and G.L. Willis.</i>	

 THE BREEDING PIG

<i>SYMPOSIUM: Nutrition-reproduction interactions in the breeding sow.</i>	
Introduction	277
<i>P.E. Hughes.</i>	
Nutritional strategies for breeding sows	281
<i>D.J.A. Cole.</i>	
Nutritional influences on sows	285
<i>I.H. Williams and B.P. Mullan.</i>	
The endocrine basis of nutrition-reproduction interactions	290
<i>P.E. Hughes and G.P. Pearce.</i>	
Nutrition-reproduction interactions in the breeding sow. Conclusion	296
<i>P.E. Hughes.</i>	
<i>CONTRIBUTED PAPERS: The partition of nutrients during lactation and its relationship to reproductive performance</i>	
<i>B.P. Mullan and W.H. Close.</i>	
Immunization against the α subunit of bovine inhibin causes increased ovulation rate in gilts	303
<i>R.W. Brown, J.W. Hungerford, P.E. Greenwood, R.J. Bloor, D.F. Evans, C.S. Tsonis and R.G. Forage.</i>	
Oestrus detection in gilts exposed to exogenous boar stimuli	304
<i>A.J. Tilbrook and P.H. Hemsworth.</i>	
Long days delay puberty in the gilt	305
<i>A.M. Paterson and G.P. Pearce.</i>	
The effect on puberty of housing gilts with dry sows	306
<i>A.J. Peacock, G. Evans and R.J. Love.</i>	
The effect of group size and exposure pen area on boar-induced puberty in the gilt	307
<i>P.E. Hughes and G.P. Pearce.</i>	
Adrenal responsiveness and reproductive performance in gilts	308
<i>S.S. Wan, D.P. Hennessy and P.D. Cranwell.</i>	
<i>SYMPOSIUM: Pre-mating management of the gilt.</i>	
Introduction	309
<i>P.H. Hemsworth.</i>	
Age at mating and productivity of gilts	310
<i>A.M. Paterson.</i>	
Nutritional management to improve the reproductive performance of commercial gilts	315
<i>R.H. King.</i>	
Detection and mating of oestrous gilts	319
<i>P.H. Hemsworth.</i>	
Symposium conclusion	323
<i>P.H. Hemsworth.</i>	
AUTHOR INDEX	327

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PREFACE

Following the success of the Inaugural conference of the Australasian Pig Science Association in 1987 many delegates expressed a desire to attend a similar conference at the same venue a couple of years later.

The Second conference of APSA again considers topics of importance to pig production. Progress in pig production will largely be based on a greater understanding of scientific principles together with the development of relevant technologies at the production level. The purpose of the second APSA conference is to provide a forum in which research workers from a number of fields can consider and discuss basic research conducted in pig production. The disciplines covered in this conference include meat science, genetics, health, reproduction, nutrition, behaviour and welfare. Regardless of individual interest all those working in pig research have a common objective of improving productivity and quality of the end product.

The conference will also provide an opportunity for scientists to meet personally in Albury. It is hoped that the social as well as scientific interaction will provide the momentum for new as well as the continuation of collaborative efforts to advance our knowledge of pig production. The papers presented at this conference are contained in this book and should prove to be of value to anyone involved in aspects of pig research and production.

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THE EFFECTS OF HANDLING, TRANSPORT, SLAUGHTER AND CHILLING ON MEAT QUALITY AND YIELD IN PIGS

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Introduction

Pork quality and yield is greatly affected by preslaughter handling, method of slaughter and chilling. Consequently, a lot of work has gone into finding the best methods of performing these operations so as to optimise meat quality and yield. Although appropriate methods for transporting and slaughtering pigs are fairly well established, they are notoriously difficult to implement in practice. As a result of this failure, loss of yield occurs as does damage to meat quality, sometimes on a heroic scale, pushing up retail prices and lowering the quality of the end product. The net result is a loss of competitiveness affecting the entire pigmeat industry.

There are several sources of economic loss associated with transport, slaughter and chilling of pigs. In the extreme case animals may die as a result of mishandling. This is common in breeds with inherited stress-susceptibility. Losses of meat yield occur before slaughter as a consequence of fasting and dehydration and after slaughter as a result of evaporative chill loss from the carcass and drip from meat cuts. Skin lacerations and carcass bruising are the result of fighting and rough handling. Bone fractures and blood-splashed meat occur at stunning. The quality of the lean meat as a raw material for further processing is reduced by the development of PSE (pale, soft, exudative) pork. This defect is determined both by hereditary and preslaughter factors. The occurrence of DFD (dark, firm, dry pork), on the other hand, is wholly determined by preslaughter handling. DFD reduces the shelf-life of fresh pigmeat products.

The purpose of this review is to look at the events between farm and chiller that cause problems of meat quality and yield and to discuss possible remedies.

Colour, drip and keeping quality of pork

Before examining problems of transport and handling, a brief account of stress-related defects in meat quality is necessary. Losses to the UK pig industry from meat quality problems (PSE and DFD) are estimated at £20 million (US \$30 m)/annum (Guise, 1987). Abnormal paleness or darkness reduces the saleability of pork (Topel *et al.*, 1976; Wachholz *et al.*, 1978) and drip in retail packs lowers appearance and yield (Kauffman *et al.*, 1978; Smith and Lesser, 1982). PSE pork is more susceptible to discolouration during retail display (Topel *et al.*, 1976; Greer and Murray, 1988) and tastes less juicy after cooking (Bennett *et al.*, 1973; Jeremiah, 1984).

DFD pork spoils more quickly than normal pork (Rey *et al.*, 1976; Newton and Gill, 1981; Greer and Murray, 1988). This is a particular problem in the fresh, chilled, vacuum packaged pigmeat trade. Pork quality varies between cuts: DFD is more common in shoulder and ham muscles, PSE in the loin and outside ham.

There is little information about these pork quality defects under Australian conditions (Warner and Eldridge, 1988). Examination of a day's kill in a Victorian abattoir revealed 10% PSE and 15% DFD. If representative, this incidence is of real commercial significance.

Paleness and drip loss in pork are determined to a large degree by the rate of pH fall after slaughter. If the rate is rapid, so that the pH reaches a low value while the

temperature of the carcass is still high, the meat is pale and watery as a consequence of protein denaturation.

The rate of acidification after death is largely determined by the degree of muscle stimulation immediately before and during slaughter (Warriss, 1987); muscle temperature at death and rate of cooling is also important (Long and Tarrant, 1989). This, however, is an over-simplification because the physiological response to environmental stressors (physical exercise, heat, noise, mixing, etc.) is greatly influenced by pig genotype. Identical preslaughter handling of pigs from the same production unit may give vastly different results in terms of meat quality with different genotypes.

Unlike PSE there is no evidence for a strong genetic component in DFD, caused by muscle glycogen depletion before slaughter. In living muscle, the pH remains just above 7.0 except during heavy exercise when it may temporarily fall to about pH 6.4 (Sahlin *et al.*, 1978). This is caused by a transient increase in lactic acid content within the muscle fibres. Further acidification of the working muscles would be damaging and is prevented by the onset of fatigue.

After slaughter, breakdown of glycogen to lactic acid is the only energy-yielding metabolic pathway available in the anaerobic interior of muscles. In well-fed, rested animals, meat pH falls to about 5.5. Then glycogen breakdown ceases even if residual glycogen still remains undegraded in the muscle. When muscle glycogen is depleted before slaughter post-mortem acidification is curtailed. Any stress factor that depletes muscle glycogen below the amount that is required to achieve a final pH value of 5.5 will cause a high final pH value in the meat, if the animal is slaughtered before glycogen resynthesis can occur. At ultimate pH values above 5.8 the shelf life of fresh chilled pork products is progressively shortened. This is caused by the lower content of lactic acid and glucose in the meat, which facilitates bacterial spoilage (Newton and Gill, 1981).

On-farm handling and loading for transport

Time of last feed

The most important reasons for withdrawing feed from pigs before transport are to lower the mortality rate during transport and to reduce abattoir problems associated with spillage of gut contents and waste disposal (Nielsen, 1982; Eikelenboom, 1988).

Deaths in transit are higher in recently-fed pigs, particularly in hot weather and in stress-susceptible pigs. Vomiting during transport is fairly common and is thought to be caused by pigs being fed shortly before loading, pigs may die from choking and suffocation through inhalation of their own vomit (Guise, 1987). Lower transport-death losses on Mondays in abattoirs in West Germany were attributed to the absence of feeding on Sunday evenings, thereby allowing a 24 h interval between last feed and transport (Von Mickwitz, 1982). Feed given within 10 h of slaughter is not converted into carcass gain and is wasted. It increases the risk of spread of *Salmonella* at evisceration.

On the other hand, losses of carcass yield due to fasting will occur when the total time from feeding the last meal to slaughter goes over 18 h. Most pigs are fed either once or twice a day. Pigs fed only once are more likely to have been fasted for longer before slaughter. Twenty-two per cent of pig producers in Northern Ireland (Moss, 1986) fed their animals a final meal on the morning of delivery to the slaughter plant, 54% fed them the evening before delivery and 24% fed them on the morning of the day before delivery. The average time between the last meal and leaving the farm was estimated at about 14 h (Moss, 1986; Warriss and Bevis, 1987). When transport time is added on, it is apparent that pigs may experience prolonged periods without feed before they are killed. In four slaughter plants in England, Warriss and Bevis (1987) estimated that three-quarters of the pigs had been fasted for more than 8 h, half for

more than 18 h, and a quarter for over 30 h before slaughter. These data indicate that half of the pigs slaughtered at these plants may be subject to fasting times that reduce carcass yield, as discussed below.

Withdrawal of feed before pigs were collected for transport was shown in The Netherlands to reduce the incidence of pale, watery pork and to increase the meat final pH value (Eikelenboom, personal communication). Drip loss and cooking loss were reduced and pork colour was improved. There was some increase in the occurrence of DFD pork, but the net effect was favourable. Raising the meat final pH value is important, since exporters must meet the pH specifications of their customers who manufacture cooked hams. If these results prove to be consistent, Dutch slaughterhouses may develop a system in which producers will be penalised if the weight of stomach and intestines is above a certain value.

Fasting slaughter weight pigs for up to 96 h caused only an 8% increase in the incidence of DFD pork (Gonzales *et al.*, 1987). Becker *et al.*, (1989) also found no consistent increase in pork final pH value after 72 h of fasting including 11 h of transportation. These findings show that prolonged fasting and transport are by themselves insufficient to cause a major increase in DFD pork. Producers are left to their own intuition to strike a delicate balance between feeding close enough to the time of slaughter (within 18 h), but not too close to the time of collection (a 4-6 h gap is the minimum required, depending on the volume of feed given) (Guise, 1987). The present recommendation in Denmark and in The Netherlands (Nielsen, 1982; Eikelenboom, 1988) is to give pigs their last feed on the afternoon or evening, before collection the following morning. The pigs remaining in the pen are given access to feed again after the pigs for slaughter have been removed. In Britain, the recommendation (MLC, 1986) is not to feed pigs on the day of slaughter; pigs should receive only a light feed on the day before slaughter; water should always be available.

Moving pigs to the collecting pen

There are two stressful operations on the farm, firstly, driving pigs from the fattening pen to the truck and secondly, making pigs mount the loading ramp and enter the truck (Van Putten, 1982). Fattening pens should be equipped with a functioning door for removing the finished pigs. The door should be situated in a corner of the pen and open outwards, and be free of obstacles such as the feeding trough. This will allow one man to remove some or all of the pigs with the aid of a small wooden board. Pigs for slaughter should never have to be lifted out of the fattening pen.

Ideally, races and collecting pens should be of solid construction, not open mesh or tubular. Pigs like to keep together, so the corridor and doorways should be wide enough to allow a group of three or four pigs to be herded together to the collecting pen. Pigs move more easily in straight lines, so sharp turns should be avoided. Pigs also move more easily from dark to light, providing the light is not intense enough to blind them, in which case they will baulk. Van Putten (1982) found that pigs entered an unfamiliar pen three times faster when the pen was lighted. This behaviour can help the drover if light switches are put within easy reach to enable him to switch off lights where the pigs are and to switch on lights where they are going. The lamps must illuminate the floor and not shine into the eyes of the approaching animals. Pigs exposed to daylight during rearing readily move towards daylight, unless the sun is shining into their eyes (Grandin, 1988a).

Use of electric goads

Electric goads that deliver a stimulus of between 30 and 200 volts are available for use in farms and abattoirs. When used skilfully they make pigs move more rapidly and facilitate the loading of pigs at high stocking density. When misused they cause turmoil, resulting in darker meat and more skin blemishes (Guise and Penny, 1989a).

On balance, the disadvantages outweigh the advantages, and the abolition of electric goads is recommended (MLC, 1986). In the UK, about half of truckers surveyed said that they did not use goads (Anon., 1988c).

Marking and tagging

If ear tagging or slap marking is necessary then it is preferable to do this in the collecting pen rather than in the fattening pen. The additional stress is less disturbing for pigs in the novel environment of the collecting pen than in the familiar fattening pen. Better still, by using a dye, marking can be carried out up to three weeks before slaughter, when pigs are being weighed. In this way long loading times and rough treatment of the pigs before slaughter are prevented.

Typical waiting times for pigs in the collection pens before loading were found in Britain to range from 0-65 min but averaged only 9 min (Anon., 1988c). Pigs should be allowed to rest, unmixed, in the collecting pens before being loaded.

Social regrouping

Deep scratches, sometimes with underlying bruised muscle, are caused by the aggressive activity and riding that is triggered when pigs are mixed. The problem is exaggerated by the presence of entire males. Farm, vehicle and lairage pens are rarely of the same size, so in most cases mixing is unavoidable. If social groups have to be mixed, fighting damage is reduced if mixing takes place at loading; pigs fight less on a moving truck (Nielsen, 1982; MLC, 1986).

Over 40% of pig carcasses in the UK showed some evidence of fighting damage; the number actually downgraded for damage was about 4% (Warriss, 1984). A rough estimate of the reduction in carcass value caused by skin blemish to the UK pig industry is £1 m (US \$1.6 m)/annum (Guise, 1987). The incidence of fighting damage in boar carcasses was about twice as high as in castrates and females. Mixing pigs at loading or mixing in the factory lairage both increased skin blemishes, however, mixing at both places did not further increase skin blemishes (Guise and Penny, 1989b).

Another common problem caused by mixing is loss of muscle glycogen as a result of unaccustomed physical activity. This is a potent cause of DFD pork. Light exercise such as that associated with routine movements places little demand on muscle glycogen stores and is fuelled mainly by aerobic metabolism of fatty acids (Lindsay, 1981). As the intensity of exercise increases, so does the requirement for glycogen as an energy source. Eventually the aerobic capacity of the muscle is exceeded in very heavy exercise and glycogen becomes the main source of energy for continuing activity.

Rapid glycogen breakdown in living muscle may be triggered by an adrenergic mechanism (stress) or by a contractile mechanism (exercise), or by both mechanisms acting in concert. In addition, a slow depletion of muscle glycogen occurs during prolonged starvation.

Any environmental situation that triggers one or more of the above muscle glycogen breakdown mechanisms will result in DFD pork, if the stress is allowed to persist for long enough. Simultaneous adrenergic and contractile activation of muscle results in the maximum rate of glycogen breakdown. Such a situation may arise, for example, during preslaughter mixing of pigs, where circumstances demanding unaccustomed or heavy physical activity may also cause sympathetic arousal and adrenalin release.

In Ireland, pigs from small fattening units had the lowest incidence of DFD pork, while those from large fattening units had a higher incidence. Pigs from live auction marts and dealers had the highest incidence of all. The differences were attributed to differences in the preslaughter handling of the groups and particularly the likelihood that pigs from large units and markets had been mixed; pigs from small units tended

to be brought to the factory by the farmer (Gallwey and Tarrant, 1979).

Container transport for pigs

It is difficult to avoid mixing, even in an "all-in all-out" production system, because pens are rarely of similar size at the farm and in the truck and lairage. One resolution is to use a container transport system which would allow pigs to be moved from the fattening pen to slaughter without mixing. As well as eliminating the skin damage and dark meat caused by fighting and mounting activities, the use of special containers for transport would eliminate much of the stress of loading and unloading (Ring and Blendl, 1984) and also overcome the problem of painful locomotion caused by leg weakness, which may affect up to 50% of slaughter weight pigs (Van Putten, 1982). Drawbacks include the expense of a containerized transport system and the high degree of logistical organisation that is required. A wider range of live weights would have to be tolerated by the slaughter plant to allow for the different growth rates likely between members of the rearing group.

Loading

Loading facilities are often very poor on pig farms. The options, in order of preference, for loading pigs into trucks are:

- (1) a loading platform on a level with the truck deck;
- (2) a hydraulic tailgate lift on the truck;
- (3) a fixed, permanent loading ramp with a slope below 20° (about 1 in 2);
- (4) a portable loading ramp with a slope below 27° (about 1 in 3).

Loading ramps on trucks are frequently too steep; Van Putten (1982) suggested that pigs often refuse to climb a steep loading ramp because, lacking experience, they view it as an impassable obstacle. The maximum recommended angle for a permanent pig ramp is 20° and if space permits, a 15° angle is better. Experience indicates that livestock move more easily up and down stair-step ramps than cleated ramps. Stair-steps with a 7 cm rise and a 20 cm tread width work well with pigs (Grandin, 1988a). On wooden ramps the cleats should have a 20 cm space between them. A loading ramp should be wide enough to allow two or three pigs to climb it together.

A chute with two lanes is efficient for loading pigs. The outer fences must be solid to prevent the pigs from baulking at distractions outside the fence and the divider fence between the two lanes is "see through" to promote following behaviour (Grandin, 1988a). The interior of the truck should be well lit.

A very good loading system, now becoming widespread, is the hydraulic tailgate lift capable of holding groups of 8-10 pigs. A tailgate lift is particularly useful on multideck trucks. It reduces considerably the amount of stress at loading (Augustini and Fischer, 1982) and encourages good animal handling by the truck driver.

Transport to slaughter

Vehicle design

The transport vehicle should have a covered deck, effective mechanical ventilation and ventilation openings both low down and high up on the sides, as well as a tailgate lift, partitions and a non-skid rubber surface on the floor according to Nielsen (1982). Internal pens on the vehicle need to be variable in size to allow for different group sizes without mixing and to allow adjustment for different live weights. A variable penning system which is light in weight and which will enable pigs to be loaded swiftly is being designed in England (Anon., 1988c). A recent survey in the UK showed that loading time for 100 pigs can take as long as 2 h 15 min and that pigs were penned on

trucks in group sizes of 19 on average and ranging from 8-37.

In the UK most pig trucks are two-decked; triple-deck vehicles are in operation in several European countries. These vehicles often have hydraulically lifting decks, avoiding the need for internal ramps. Triple deckers are probably more suited to weaner transport, as the headroom allowed for each deck is restricted and access during transport is difficult. About four-fifths of UK pig trucks used bedding; all were naturally ventilated and two-fifths could not vary the ventilation rate. Deaths occurred on 5% of trucks included in the survey (Anon., 1988c).

Stocking density on trucks

Decisions about stocking densities on pig transporters are usually made by the haulier, who is likely to be primarily influenced by economics. Too little space leads to heat stress, fatigue, lower meat quality and higher death rate. Too much space is uneconomic and allows animals to be thrown about during sudden braking or cornering. Because pigs tend to lie down during transport, low density is less of a problem than overloading.

In practice, recommended space allowances are rarely adhered to, and space allowances as little as 0.3 m²/100 kg pig are frequently encountered (Guise and Penny, 1989a) which is equivalent to 97 head/12.2 m deck. Such tight packing is unacceptable as pigs have barely enough room to avoid standing or lying on each other. More fights were observed in densely packed pigs and there was more skin damage and a higher incidence of rectal prolapse (Anon., 1988c; Guise and Penny, 1989a). An inherited predisposition to rectal prolapse in some pigs may be triggered by the unaccustomed exercise and squeezing at high stocking density.

The effect of stocking density on meat quality is determined by breed and ambient temperature. For example, in Britain, transport of Landrace x Large White pigs for 192 km to slaughter at stocking densities of either 0.3 or 0.4 m²/100 kg live weight and at ambient temperatures of 14°C or 10°C had no effect on the incidence of PSE or DFD, which was very low under all of the above conditions (Guise and Warriss, 1989). In contrast, densities of 0.33 m²/100 kg compared with 0.43 m²/100 kg led to poor meat quality after transport of German Landrace pigs for 254 km (Gerber, 1984).

The recommended stocking density for slaughter weight pigs varies considerably between different regions and countries (Table 1). This variability may reflect regional differences in weather conditions and the proportion of the local pig population with inherited stress-susceptibility. The minimum recommended space allowance in Table 1 is 0.35 m²/100 kg pig. Not more than 30 pigs should travel in one pen (Nielsen, 1982). This space allowance/pig must be increased by 10% in hot weather, for example at ambient temperatures above 25°C and by 20% if the humidity is also high (Anon., 1988b). More space is also needed in conditions of heavy traffic or in urban areas where ventilation may be reduced (MLC, 1986). The present recommendation to the trade in Britain is that the space allowance on trucks should be increased to at least 0.4 m²/100 kg pig.

Allowing more floor space/pig would result in fewer pigs being transported on each load and hence higher transport costs. Some financial reward should be recovered by reducing the damage to carcasses caused by pigs fighting during the journey. To encourage the provision of enough floor space in pig trucks, it is essential that pen divisions can readily be moved and to give the correct area for the number (or weight) of pigs to be loaded (Anon., 1988c).

Fasting, transport and weight loss

Fasting results in a loss of live weight and carcass yield in pigs (Table 2). The rapid, early loss of live weight, amounting to 5% in the first 24 h, is mostly gutfill. However, in the post-absorptive period, commencing 12-18 h after the last meal,

catabolism of bodily energy reserves (glycogen and fat) results in a loss of carcass yield. Fasting causes a reduction in the killing-out percentage of about 1% in the first 24 h.

Dehydration

When fasting pigs are denied access to water also, or are unable to avail of the water supply, additional weight loss occurs due to dehydration. The rate of dehydration is greatly accelerated by road transportation (Warriss *et al.*, 1983; Becker *et al.*, 1989). Factors that would contribute to an increased rate of dehydration during transport, and which are likely to occur in transit, are increased ambient temperature and decreased humidity in the stockcrate, increased airflow and increased body temperature. Warriss *et al.*, (1983) found a 2.1% reduction in killing-out percentage after only 6 h of transportation (Table 2). The pigs showed evidence of haemoconcentration even though they had free access to water in the abattoir for 1 h before slaughter. This was apparently insufficient to allow complete recovery of live and carcass weight loss.

Table 1. Effects of different stocking densities used in the road transport of pigs to slaughter

Floor area (m ²)/ 100 kg pig	Observations	References
0.30	Frequently encountered in the trade; increased skin damage; may predispose to rectal prolapse. Meat quality unaffected in British pigs.	Guise and Penny (1989a) Guise and Warriss (1989)
0.33	Meat quality adversely affected in German pigs. Associated with overcrowding on long journeys, increased fatigue, higher carcass temperatures and dark meat.	Gerber (1984) Lambooy <i>et al.</i> (1985)
0.35	Recommended in Victoria; plus 10% when ambient temperature is over 25°C. Recommended in Denmark for 90 kg pigs.	Anon. (1985) Nielsen (1982)
0.40-0.50	Recommended in Britain.	MLC, (1980) Guise and Penny (1989a)
0.42	Body temperature elevated by 1°C compared with transport at 0.51 m ² /100 kg live weight.	von Mickwitz (1982)
0.44	Acceptable for welfare and meat quality.	Lambooy <i>et al.</i> (1985)
0.50	Recommended in West Germany; allows all pigs to lie down.	von Mickwitz (1982)

Dehydration causes loss of mass of muscle tissue which is about 75% water. Becker *et al.*, (1989) reported that carcass weight loss as a result of transport and fasting of slaughter hogs for 72 h resulted in a maximum carcass loss of 4.64 kg. The

most significant change in the carcass was a decreased loin muscle area; transported hogs had muscle areas up to 15% smaller than muscles from the control animals. Decrease in loin muscle area was associated with transport *per se* and was not found in control pigs denied feed and water.

Loss of live weight during a 2 h road journey in France (Dantzer, 1982) was accounted for by excretion of faeces and urine (41%) and by evaporation through the skin and respiratory tract. Some additional loss was related to respiratory exchanges; for a respiratory quotient of 1, 0.5 g is lost for every litre of oxygen consumed. Road travel by itself was responsible for about 36% of total loss in live weight, while 22% was attributed to stationary handling and confinement. Loss of weight in pigs during road transport was related to ambient temperature ($r=0.72$) and humidity ($r=-0.6$).

Pigs lose more carcass weight during transport on hot days (Anon., 1988a) because body moisture loss is greater. In Nebraska, Mayes *et al.*, (1988) reported that variation in carcass weight loss was mainly attributable to variation in ambient temperature.

Provision of water in trucks did not materially alter carcass weight loss because water intake was too low (Lambooy *et al.*, 1985). During 29 h road transport from The Netherlands to Italy pigs drank only 0.65 l of water each, whereas under normal housing conditions pigs of 100 kg live weight will drink 7-20 l/day. It was suggested that the low water intake may have been connected with the absence of food and/or dislike of drinking from bite nipples in a shaking truck. Pigs transported by road for 44 h in The Netherlands lost about 8 kg live weight, half of which was carcass weight. Carcass weight loss was mainly attributed to water loss as was shown by a lower percentage of water in the backfat of pigs that were transported for 44 h with water (15.6%) or without water (14.5%) compared with controls that were transported a very short distance to slaughter (18.4%). The pigs with access to water used only 5.4 l each over the two days of transport, part of which was wasted.

The results in Table 2 suggest that fasting alone causes about 1% loss of carcass weight/day and that dehydration, due to transportation and loss of water intake, can cause additional losses of the same or greater magnitude depending upon ambient conditions.

Yield of liver

Body organs and the alimentary tract showed small weight losses after 24 h fasting (Jones *et al.* 1988a). Loss of liver weight is economically the most important of these additional losses. This occurs rapidly on the first day of fasting. The data in Table 2 suggest that about 12% of liver weight is lost during the first 24 h off feed. A further loss of 7% occurs during the second day of fasting. Fasting for three or four days results in an additional loss of about 2% per day. Transportation of fasted pigs does not appear to cause additional weight loss in liver, in contrast to the findings with carcass weight loss. This suggests that dehydration is not a major factor in loss of liver weight.

The pattern of weight loss is partly explained by the liver's function as a reservoir of glycogen for the maintenance of blood glucose in the post-absorptive period. Liver glycogen content is variable, a typical value in non-fasted pigs is about 30 mg/g (Warriss and Bevis, 1987). By 9 h after food withdrawal from pigs, over 50% of stored glycogen (and the associated water) may have been mobilized and by 18 h the glycogen concentration was negligible (Warriss *et al.*, 1987). The eating quality of the pig livers was also adversely affected by fasting, with lower tenderness, juiciness and overall acceptability. Variable fasting times before slaughter could explain some of the variation in eating quality commonly found in pig livers.

In summary, a 12-18 h fast between last feed and slaughter is recommended to avoid deaths in transport and the hygiene problems associated with dressing carcasses with overfull guts, at the expense of some loss in liver yield. Fasting periods of more

than 18 h should be avoided because they are associated with significant reductions in carcass yield. Pigs should have easy access to water before and after transport as dehydration causes carcass weight loss even on relatively short journeys.

Table 2. Effect of fasting or fasting plus transport on losses of live weight, killing out percentage and liver weight in slaughter weight pigs

Time off feed (h)	Time in transport ¹ (h)	Loss of live weight (%)	Reduction in killing-out percentage ²	Loss of liver weight (%)	References
8	1	0.6	0.6		Warriss <i>et al.</i> (1983)
8	6	2.3	2.1		Warriss <i>et al.</i> (1983)
24	0	5.4	1.4	11	Gonzales <i>et al.</i> (1987)
24	0	5	1	16	Jones <i>et al.</i> (1988a)
24	0		0.5; 0.8	11	Mayes <i>et al.</i> (1988)
24	0			15	Warriss <i>et al.</i> (1987)
24 ³	0			8	Becker <i>et al.</i> (1989)
24 ³	11			9	Becker <i>et al.</i> (1989)
48	0	7.3	1.9	18	Gonzales <i>et al.</i> (1987)
48	0	7	2.2	20	Jones <i>et al.</i> (1988a)
48	0		1; 1.8	19	Mayes <i>et al.</i> (1988)
48	0			23	Warriss <i>et al.</i> (1987)
48 ³	0			17	Becker <i>et al.</i> (1989)
48 ³	11			17	Becker <i>et al.</i> (1989)
48	44	8	5		Lambooy <i>et al.</i> (1985)
72	0	8.3	2.4		Gonzales <i>et al.</i> (1987)
72 ³	0			23	Becker <i>et al.</i> (1989)
72 ³	11			17	Becker <i>et al.</i> (1989)
96	0	9.6	2.8	23	Gonzales <i>et al.</i> (1987)

¹Time in transport usually corresponds to time off water, except Lambooy *et al.* (1985) who provided water in the truck but consumption was low; ²killing-out percentage is carcass weight expressed as a percentage of the initial live weight; ³time off feed and water.

Deaths during transport

The most obvious problem in moving pigs to slaughter is death in transit. A survey in Britain found a loss of 0.07%, representing about 9,000 deaths in transit each year (Sains, 1980). On average, deaths in transportation cost the British producer 5p (7.5 US cents)/pig sold, but losses vary enormously and may reach 25-30 p/pig slaughtered (Guise, 1987). Much higher death rates have been reported in The Netherlands, West Germany, Denmark and Belgium, occasionally exceeding 1% (Sains, 1980). In the USA, 80,000 pigs die in transport each year, representing 0.04% of throughput rising to 0.05% in summer (Anon., 1988b). Hot weather, driving technique, genetic stress susceptibility, vehicle design and pig health are all associated with transport deaths. Length of journey is not a major factor, but losses are greater where pigs are fed on the day of transport, regardless of distance travelled. Death losses often double on hot, humid days (Anon., 1988a).

According to the "Victorian Code of Practice" (Anon., 1985) the range of temperature that affords optimum comfort for finishing pigs is 15-30°C and pigs are very susceptible to heat stress at temperatures of 38°C or more and may die if transported.

If road transport is undertaken during hot weather, shade should be provided and the truck should be well ventilated. When the vehicle is stationary the pigs should be unloaded promptly before there is a build up of heat within the enclosed stockcrate.

Alternatively the vehicle must be parked in the shade. Some new trucks have a built-in sprinkler system for hot weather hauling. Drivers should take rest breaks without pigs in the truck, so that the shortest possible transport times are attained. A recent survey at a large abattoir in Britain (Anon., 1988c) found that one-fifth of pig trucks stopped during the journey from farm to abattoir for various reasons. Typical waiting times on trucks before unloading were 12 min (average) and 5-35 min (range).

Effective mechanical ventilation in pig trucks in Denmark was credited with reducing deaths in trucks by 50% and reducing deaths in lairage by one-third (Nielsen, 1982). As well as hyperthermia, heart failure is a likely major factor in transport deaths. In German Landrace pigs of 100 kg live weight Augustini and Fischer (1982) found the highest heart rates at loading and unloading. Very high rates were recorded during assembly, weighing and loading but once inside the truck the heart rate dropped sharply and remained relatively constant during the journey. At unloading the heart rate increased but not as much as at loading. Body temperature also increased rapidly at loading but under good transport conditions it had returned to normal at the end of a 100 min journey, even when the ambient temperature was as high as 29°C. However, at a high stocking density (0.35 m²/100 kg) the rise in rectal temperature during transport was greater and the return to normal was delayed (Von Mickwitz, 1982; Augustini and Fischer, 1982). The pigs were unable to dissipate heat effectively even though they panted intensively.

The apparent association between the heart rate, body temperature and incidence of PSE in German Landrace led Augustini *et al.* (1977) to define the maximum heart rate (<85 beats/min) and rectal temperature (<38.8°C) at slaughter that was commensurate with desirable meat. PSE meat was associated with heart rates above 136 beats/min and temperatures above 40.1°C.

Under uniform transport conditions Augustini and Fischer (1982) observed large differences between German Landrace pigs in heart rate, body temperature and meat quality. These were almost wholly explained by the presence of the halothane gene. Furthermore, carcass conformation (muscling) showed a much stronger relationship with incidence of PSE than transport conditions of temperature, humidity and loading density.

Stress syndrome

Most cases of recurring high transport losses in pigs can be attributed to the malignant hyperthermia syndrome. This syndrome is genetically determined by an autosomal recessive gene. Stress susceptible piglets can be selected out at an age of 6-12 weeks by means of the halothane test. Transport losses in The Netherlands fell from 4.2 per thousand in 1972 to 3 per thousand in 1980, coinciding with a drastic reduction in the frequency of halothane reactors in the Dutch Landrace breed from 36% in 1977 to 1% at present (Eikelenboom, 1988). Because farmers are not yet paid for meat quality, the major reason for selection against halothane reactors is to reduce death losses due to stress.

Onset of the stress syndrome may be recognised by some of the following symptoms: High body temperature; fading and reappearing of purple skin blotches; dilated pupils and difficult irregular breathing. It is generally believed that the incidence of the porcine stress syndrome and the associated halothane gene is relatively low in the Australian pig herd (Warner and Eldridge, 1988).

Bruising

The US Livestock Conservation Institute estimated that roughly handled pigs lost as much as \$50/100 pigs killed (Anon., 1988a). A major source of bruising was rough, careless handling, particularly at loading and unloading. The distribution of bruises on pig carcasses was: Hams, 66%; belly, 17%; shoulder, 10%; loin, 7%.

A particular problem was "spreader injury" of the hind legs leading to damage and possible loss of both hams. This problem is caused by slippery floor surfaces and can be prevented by giving concrete surfaces a very rough broom finish.

Time in transport

Because the main stresses in pig transport are at loading and unloading, short journeys may be more detrimental than longer ones if the driving, stocking density and ventilation is good. Using Danish Landrace pigs, Nielsen (1982) compared a normal transport with no transport. In his experiment pigs were kept (and fed) at the abattoir for a week. Then half of the pigs were subjected to normal transport and holding, while the other half were driven quietly about 20 m to stunning. The incidence of PSE in two ham muscles (*gluteus medius* and *biceps femoris*) was much lower in the pigs that were transported (8% and 5% respectively) than in the pigs killed without transportation (33% and 21%). An extremely considerate treatment on the day of slaughter had thus resulted in a higher incidence of PSE meat. Several other investigators have reported a reduction in the incidence of PSE pork as transport time increases (Barton-Gade *et al.*, 1982; Malmfors, 1982; Heinz *et al.*, 1984). In pigs with an inherited predisposition to PSE meat, the gradual depletion of muscle glycogen reserves that occurs during transportation reduces the potential for a low final pH value in the meat and the development of PSE. Drip loss in pork decreases as the final pH value of the meat increases (Somers *et al.*, 1985; Tarrant and Long, 1986; Monin and Sellier, 1987).

On the contrary, prolonged transportation and holding times, e.g. overnight lairage before slaughter, cause more DFD pork. Here we are facing a dilemma, as with longer transport and lairage times the incidence of PSE falls but the incidence of DFD increases. The best results may be achieved by allowing a sufficient interval from last feed to slaughter to cause moderate depletion of muscle glycogen (less PSE) while giving the pigs as considerate a treatment as possible, thereby preventing exhaustion (less DFD pork) (Nielsen, 1982).

Unloading at the abattoir

Increased carcass bruising and lacerations are unavoidable at unloading unless appropriate equipment is provided. In the construction of unloading facilities certain behavioural characteristics should be taken into account, in particular that pigs have difficulty in descending a slope, therefore hydraulic ramps should be installed or fixed unloading ramps should be constructed on several levels that approximate to truck deck level. Good lighting is important because pigs dislike entering dark areas, and following instinct works best in wide, straight or curved passages.

The first pigs should be given sufficient time to walk off the truck by themselves then the rest driven using a push-board in such a way that the group is kept together. The use of electric goads is unnecessary. The long, narrow pens common in modern abattoirs can be subdivided by cross-gates to accommodate several groups without mixing. Gates and walls of lairage pens should be of solid construction to eliminate contact between pigs in adjacent pens (Gallwey and Tarrant, 1979).

Showering of pigs before slaughter

The practice of spraying pigs with cold water is fairly common in European countries. Showering has been recommended for several reasons:

- (1) to cool and clean the pigs after transport;
- (2) to reduce aggression and to quieten the animals;
- (3) to facilitate electrical stunning by lowering skin impedance;
- (4) to reduce bacteriological contamination of the water in the scalding tank.

However, information on the best procedure for showering pigs (time, temperature and flow rate) is scarce.

During transport pigs may be subjected to temperatures approaching their upper limit of thermal tolerance. To reduce the incidence of PSE it is important to get rid of this excess heat. Showering may lower the temperature of the muscles at slaughter by direct cooling of the superficial parts in contact with the cold water, and by peripheral vasoconstriction in response to the cold stimulus. Showering also lowers physical activity in the pens before slaughter, probably through distraction or through removal of sty odour.

Accelerated cooling is probably the main mechanism by which showering improves pork quality. The data in Table 3 show that showering with an average flow rate of 27 l/min/m² for two periods of 30 min with a 30 min break between each period brought about a drop in the temperature in the loin muscle of more than 3°C in the winter and 2°C in the summer. This degree of cooling was sufficient to reduce paleness and drip in loin chops in the winter experiment but not in the summer experiment. A possible explanation for the seasonal difference was the reduced cooling effect of showering in the summer when the shower water temperature was 7°C higher than in winter. In Dutch experiments the incidence of visually judged PSE was lowered by showering with water at 10°C in both winter and summer (Smulders *et al.*, 1983). There was no evidence for a significant increase in DFD pork as a result of cold showering in the Dutch or Irish experiments.

Observations of pigs during and after showering revealed no obvious signs of distress. The core temperature for showered pigs in Table 3 fell within the normal range of 38.7-39.8°C (Andersson, 1984), indicating that the cold treatment was not severe enough to overcome the homoeothermic regulatory mechanism of the pig. Shivering was not apparent and the animals behaved normally while being walked from the shower to the stunning pen. However, under more severe winter conditions, cold showering may be inappropriate.

Table 3. The effect of showering ("S") pigs with cold water on body temperature and meat temperature in winter and summer. Irish Landrace and Large White boars, 92 kg average live weight, were used. The results show that showering cooled the loin muscle but did not affect core temperature. Data are in °C (Long and Tarrant, 1989)

Time of year	Ambient temperature in the lairage	Shower water temperature	Rectal temperature at slaughter		Loin muscle temperature on the line	
			"S"	Non-"S"	"S"	Non-"S"
Winter	12	9	39.0	39.3	37.0 ^x	40.5 ^y
Summer	20	16	39.5	39.5	37.9 ^x	40.0 ^y

^{x,y}differ at $P < 0.001$

Time in lairage

Slaughter of pigs immediately after delivery to the abattoir resulted in a higher incidence of PSE pork than if pigs were rested before slaughter. A minimum rest period of 1 h was necessary for body temperature to decline to a normal value in German Landrace (von Mickwitz, 1982).

The current recommendation in Holland and Denmark is to rest pigs in a suitable lairage environment for 2-4 h (Eikelenboom, 1988). To achieve an adequate rest period, management must adjust the delivery rate to the slaughter rate. For this reason Eikelenboom (1988) estimated that the capacity of the lairage should exceed six times the slaughter rate in pigs/h. In slaughter plants in Britain nearly half of the pigs were killed after no more than 2 h in lairage, but about 30% were held overnight and slaughtered the next day.

Moving pigs forward to the stunning point and the stunning process itself may be the two most important sources of stress in slaughtering pigs (Troeger and Woltersdorf, 1989). Some lairage design features which help the free flow of pigs are (Anon., 1988c):

- (1) multiple lairage pens in parallel feeding into a single crowd pen the purpose of which is to provide a reservoir of pigs to avoid holdups due to a slow supply of pigs from the lairage pens;
- (2) remote control pneumatic gates on lairage pens, with guillotine or sliding action;
- (3) a circular crowd pen with follow-up gate designed to give the operator a good position and close control over the pigs;
- (4) twin races, that are curved and lighted, leading forward to the stunning area. These encourage the forward movement of pigs by using their inquisitiveness to look around corners. Twin races allow more pigs to be held in a given length. Double corridors with good visual contact between the lanes encourage the pigs' following instinct and are now in regular commercial use.

The incorporation of one or more of these simple design criteria should improve the flow of pigs to the stunning area. Barton-Gade (1984) found that halothane reactors gave poor meat quality, irrespective of preslaughter handling. In contrast, halothane negative pigs (homozygotes) showed improved meat quality with less stressful handling and this was also true, but to a lesser extent, for the heterozygote. Genotypes which are extremely sensitive to stress may produce PSE meat no matter how careful the preslaughter handling is. Genotypes between these extremes will respond more or less favourably to improved handling. In any particular type of pig, therefore, the contribution made to meat quality by genotype and handling stress will vary. The relative contribution of genotype to meat quality is much greater in countries where the pig herd has a high incidence of stress-susceptibility.

Stunning

The law in many countries requires that pigs are stunned to render them insensible to pain during slaughter. A good stunning method must also be safe and should facilitate the smooth running of the killing line. Finally, the method should not damage the carcass or the quality of the meat. Electrical or carbon dioxide stunning methods are common in pig slaughter plants and despite years of experience there is little agreement on which procedure is best.

Carbon dioxide stunning

Developed in the USA in the 1940s, this is now the principal method used in Denmark. In CO₂ stunning, pigs are immersed in a mixture of CO₂ and air for about one minute. Typical of CO₂ stunning equipment are the three types manufactured by the Butina Engineering Firm in Denmark: The Oval Tunnel with a capacity of 120-600 pigs/h; the Compact Plant, with a capacity of 90-300 pigs/h; and the Dip-Lift system which can stun up to 100 pigs/h.

The arguments that CO₂ stunning is an inhumane procedure are now based on the assumption that the gas has an acidic flavour and is pungent when inhaled at high concentrations, plus the fact that it stimulates breathing frequency and may lead to respiratory distress (Gregory, 1988a).

The behaviour of pigs when lowered into a gas chamber containing 65% CO₂ was described in three phases by Barfod and Madsen (1988):

- (1) a lag phase of about 20 sec during which the animal is quiet;
- (2) a phase of motor activity which starts suddenly and lasts for about 10 sec during which the pig makes running movements;
- (3) a relaxation phase which starts approximately one minute after the animal is lowered into the chamber.

Convulsive behaviour may occur in phase 2 and is sometimes interpreted as escape behaviour. On the basis of electroencephalograms, Forslid (1987, 1988) suggested that the physical convulsions occurred after the loss of consciousness. Zeller *et al.* (1987) on the other hand suggested that the pigs appeared to be conscious during part of the convulsive episode. Disagreement on this point has created doubt about the humaneness of CO₂ stunning.

Because of the low oxygen concentration in the gas mixture (typically 6%) the question may also be raised as to whether the immobilising effect of the gas mixture may be due, in part, to suffocation. Experimental evidence, based on blood oxygen content (Barfod and Madsen, 1988; Ring, 1988) and time to recovery (Laursen, 1983), suggests that suffocation does not play a significant role in CO₂ stunning.

Grandin (1988b) noted that there was a wide variation in pig reaction to immersion in CO₂. Some animals jerked and struggled violently and others exhibited little or no movement. She suggested that the variation in pig response to CO₂ immersion may have a genetic explanation. Also, excitement and rough handling prior to entry into the chamber may increase adverse reactions to CO₂.

In the interests of a humane slaughter, pigs must be stuck within half a minute of exiting the CO₂ chamber. At 80% CO₂ in air, Forslid (1988) found that brain electrical recordings indicative of unconsciousness persisted for only one minute following the end of CO₂ inhalation. There may be a more rapid return to consciousness at lower CO₂ concentrations. The absence of clonic convulsive spasms during shackling and sticking makes CO₂ stunning safer for slaughtermen than electrical stunning.

Electrical stunning

This method was developed in the 1930s and was carried out using low voltage (70-90 volts) with the pigs free-standing on the floor of the stunning pen. The practical difficulties of stunning an unrestrained animal led to the development of restraining conveyors. Concern that pigs are not adequately stunned by the low voltage system led to the use of higher voltages (above 240 volts). Meat traders sometimes claim that these developments have led to increased blood splash in the meat and to broken shoulder blades (Gregory, 1987).

The stunning current may be applied across the head and in this case a temporary stun is the aim, or it may be applied across the head and heart (as in head-to-back stunning) in which case the stun may be irreversible due to cardiac arrest.

Low voltage electrical stunning, although permitted in many countries, has been criticised by Hoenderken (1983) and others on the grounds that it failed to guarantee stunning in all animals. If the current is switched off after a few seconds the pig immediately gets up and is able to stand, indicating that loss of consciousness is slower at low voltages. Data from electroencephalograms indicated that a minimum current of 1.3 amps through the brain of the pig is necessary for an effective stun, that is, loss of consciousness within one second. In order to guarantee this in all pigs, the average current during stunning needs to be higher. Under practical conditions in the slaughterhouse at least 240 volts is necessary to obtain this amperage (Hoenderken, 1983; Gregory, 1988b). The same minimum current of 1.3 amps will effectively stun

and cause a cardiac arrest in 99% of pigs if it is applied using head-to-back electrodes positioned so they span both the brain and heart (Gregory, 1988a).

A useful quality control check on the effectiveness of electrical stunning is the presence of the rigid (or tonic) phase of muscular contraction for at least 10 sec after the application of the electrodes (Blackmore, 1988). When the stunning current is applied to an animal it goes rigid. During the rigid (tonic) phase the head becomes raised and the hindlegs are flexed into the body. The forelegs may be flexed to begin with and then gradually straighten out during the tonic phase. Rigidity in the animal normally lasts for 10-20 sec and it is best to stick while it is in this state and before the kicking phase sets in. The normal kicking (clonic) phase usually follows on from the tonic phase and lasts for between 15 and 45 sec. The kicking phase is usually less pronounced following head to back stunning than with head only stunning. At the end of the kicking phase a quiet period sets in, by which time the animal will have started to breathe. From now on the animal will start to regain awareness of its surroundings (Gregory, 1988b).

As a general guideline pigs should be stuck within 15 sec of stunning, that is, before the kicking phase starts. There is a statutory requirement in some countries to stick pigs within 30 sec of electrical stunning. Cardiac arrest stunning is claimed to give a relaxed carcass that shows less kicking and is easier to handle. Cardiac arrest does not lead to poor bleeding in pigs provided they are stuck within 2 min.

Bone fractures

The incidence of bone fractures varies between abattoirs and may be noticed only in plants which bone-out the shoulder joint. The incidence of bone fractures in Danish bacon factories using different methods of stunning was 0% in the CO₂ Compact Plant, 1% using a 700 volt automatic stunner and 1.2% using a 300 volt manual stunner (Larsen, 1983). Broken shoulder blades are more common in pigs stunned while free-standing. They occur when the animal's forelegs make sudden impact with the floor at the start of stunning. This sends a shock wave up the leg and causes a complete fracture at the neck of the shoulder blade (van der Wal, 1976). A star-like fracture also occurs at the cup-end of the shoulder blade where it butts on to the humerus. The problem can be avoided by lifting the pigs off the floor at stunning, for example by using a V restrainer (Gregory, 1987).

Broken vertebrae also occur in pigs stunned with head-to-back tongs if the voltage is too high. The associated haemorrhaging requires trimming, which takes time. Gregory (1988b) suggested that a satisfactory stun with a minimum of fractures is obtained using 1.3 amp. Broken vertebrae are not usually associated with much bleeding if the heart is stopped at stunning since this stops the pump which would otherwise force blood out of the ruptured vessels. In a similar way, a cardiac arrest at stunning will reduce the expression of bruising inflicted either immediately before stunning or between stunning and sticking (Gregory, 1988b).

Blood splash

This problem shows up as small blebs of blood in the meat and may only be noticed when the carcass is jointed. It shows up most frequently in the muscles of the shoulder and less often in the ham and loin. Blood splash is particularly unsightly in high quality processed products such as cooked hams, and the raw meat must therefore be carefully trimmed. Warrington (1974) described two types of blood splash: Petechial haemorrhages which are pinhead to pea sized; and diffuse haemorrhages which are single irregular patches 2-5 cm in diameter and are caused by tearing of the tissue.

The causes of blood splash are not well established, but it is likely that any muscular contractions which cause aneurisms in the blood vessels while systolic pressure

is high enough to force blood through the perforations are of importance (Gregory, 1987). In pigs, high voltage stunning in a restraining conveyor leads to intense muscular contractions and a high incidence of blood splash, particularly in the shoulder muscles. In a comparison of pigs stunned in a V type restrainer versus free-standing using the same stunning equipment (475 volts for 3 sec), Lambooy and Sybesma (1988) found much more blood splash in the shoulders of the restrained pigs. Bone fractures were found also, but in the free-standing pigs only. Consistently lower levels of blood splash were reported in Danish factories using Compact CO₂ stunners compared with factories using high voltage manual or automatic stunning equipment. With CO₂ stunning, trim weight of shoulder meat averaged 8 g compared to 145 g for manual and 56 g for automatic electrical stunning, respectively (Larsen, 1983).

There is a rise in blood pressure during and after electrical stunning. It is not known whether this causes muscle haemorrhages or merely increases bleeding from existing haemorrhages. Either way, reducing the interval between stunning and sticking reduces the expression of blood splash. If possible, pigs should be stuck within 10 sec of electric stunning, well before the kicking (clonic) phase of muscle activity sets in.

There is evidence that blood splash is lower in pigs stunned using high voltages compared with low voltages (Larsen, 1983; Lambooy and Sybesma, 1988). The reason for the improvement may be that the higher voltage caused a greater incidence of cardiac arrests. Gregory (1987) pointed out that a beating heart was presumably required to force blood through the ruptured blood vessels.

Problems with blood splash and bone fractures are causing pig processors in Europe to change from electrical stunning to CO₂. The main worry is that CO₂ stunning may be banned sometime in the future on the grounds that the induction is unduly stressful.

PSE

The relationship between method of stunning and incidence of PSE was examined by Gregory (1987) who found no clear advantage associated with any particular commercial method. He concluded that the underlying requirement in any stunning method is to minimise stress and muscular activity, excessive stimulation of the animal during stunning can exacerbate the PSE condition. There is evidence that the Compact CO₂ stunner produces less PSE meat than either the earlier Oval Tunnel version or electrical stunning (Larsen, 1983). The excessive muscle contractions that can occur during electrical stunning are the major problem. It is unlikely that the high voltage systems as they are practised to-day produce more or less PSE meat than the traditional low voltage methods. Troeger and Woltersdorf (1989) concluded that any of the existing electrical stunning methods are likely to increase PSE because they cause a massive release of catecholamines into the circulation and cause direct stimulation of muscle contraction by the stunning current.

In summary, high voltage electrical stunning is preferred at the present time on grounds of welfare while CO₂ is preferred on grounds of less blood splash and fractures. Both methods if properly applied are humane but, to achieve this, there is a need for major improvements in operator training and quality control of stunning in abattoirs in many countries (Devine, 1988).

On-line measurement of pork quality

Measurement of meat quality on the killing line would give the processor much better control over raw material quality. Methods are being sought for on-line measurement of antibiotic and hormone residues, PSE, boar odour, softness of fat and toughness of meat. In the case of PSE and boar odour, on-line methods are already installed in commercial premises.

PSE was traditionally detected by visual examination on the cutting line and instrumental methods are based on measurement of pH, electrical impedance or optical reflectance. Glass pH electrodes are unsuitable for on-line monitoring. Unless there are some future developments such as solid state pH electrodes that are resistant to protein and grease, only electrical and reflectance measurements are fast enough and robust enough for the job.

Two electrical meters have been developed: A portable meat structure tester for measurement of electrical conductivity (Seidler *et al.*, 1987; Schmitt *et al.*, 1987); and a portable capacitance meter (Swatland, 1987). Despite improvements relative to pH, the error in these electrical methods is still unacceptably large for quality control purposes, particularly on populations of stress-resistant pigs where extreme PSE is infrequent and the detection method must be correspondingly more sensitive (Bendall and Swatland, 1988).

Commercial attention has focussed on optical reflectance for early detection of PSE because of the widespread use of fat-depth grading probes that work by detecting the optical boundary between fat and lean. Optical reflectance is closely linked to both visual paleness and drip loss in pork. This is so because the myofibrils in the muscle cell are the principal light-reflecting and water-holding elements in meat. The spatial configuration of the myofibrils is particularly sensitive to post-mortem muscle pH. Shrinkage of the myofibrillar lattice, causing increased reflectance of light and exudation of fluid, appears to be the key mechanism in the development of PSE pork (Offer and Trinick, 1983; Swatland *et al.*, 1989). In DFD pork the reverse holds, the myofibrillar lattice is expanded at high meat pH values, causing low reflectance and retention of fluid.

Available optical probes include the GP2-Q instrument (Hennessy-Phillips grading Systems, NZ) which operates at a wavelength of 570 nm, the MQM instrument (Meat Quality Marbling, Danish Meat Research Institute, Roskilde) using a wavelength of 940 nm and the Colormet meat probe (Metron Instruments, St. John's, NF) which provides a spectrum of wavelengths from 400-700 nm. The relative influence of pork pigment and pork paleness on reflectance varies with wavelength because myoglobin and residual haemoglobin both have absorption maxima in the region 400-630 nm. Lundstrom *et al.* (1988) compared the GP2-Q and MQM probes for pigment interference and found in favour of the latter, especially when the incidence of PSE is low. In a commercial situation with a higher incidence of PSE in the material, the GP2-Q instrument can also be used to discriminate between normal and PSE carcasses but there will be a small influence of pigment.

Despite the ready availability of good instrumentation, on-line detection of PSE using reflectance probes has limited application on the killing line because PSE develops slowly in some carcasses. Consequently, a substantial proportion of carcasses that pass a checkpoint on the killing line are rejected for PSE on the following day (Tarrant and Long, 1986; Lundstrom *et al.*, 1987). The resolution to this problem is to delay the PSE checkpoint until 90-120 min post-mortem, by which time the number of false negatives is much lower, or to abandon reflectance in favour of another variable that develops faster after slaughter.

Meat chilling

Food legislation specifies that meat must be chilled and maintained at an internal temperature of 6-8°C. Chilling slows down the onset of rigor and also slows the rate of evaporative weight loss, thereby affecting both meat quality and yield.

Conventional batch chilling operates on a 24 h cycle. Throughput of pigs is limited by the size of the chill rooms and any increase in throughput requires additional chill capacity. Nominally, batch chilling operates at 4°C, 0.5 m/sec air velocity and

90% relative humidity, but in practice has to pull down initial air temperatures of up to 19°C to 1-6°C with air velocities between 0.2 and 1.5 m/sec (James *et al.*, 1988). This is because about 50% of the total heat load is released in the first few hours of chilling.

Modern batch chilling systems either have to be substantially oversized to meet the initial peak heat load imposed by the hot carcasses or sized to meet the average load over the chilling cycle. In the first case the refrigeration system operates for the majority of the time at reduced capacity and low efficiency. In the second, there is an initial rapid rise in air temperature in the chill room followed by an extended pull down period, with the net result that the desired cooling regime is not achieved.

A rapid, quick-chill tunnel is a feature of some modern pig plants. Chilling takes place in two stages with the first stage consisting of a conveyerized pre-chilling tunnel operating at sub-zero temperatures. The pre-chiller rapidly lowers the surface temperature thereby reducing the rate of evaporative weight loss and has the capacity to absorb the initial peak heat load.

The ultra-rapid chilling of pork using air at -30°C and 1 m/sec can extract the total product heat load from a 70 kg pig carcass in a single-stage 4 h chilling process (James *et al.*, 1983). A whole or jointed carcass can therefore be packed within 5 h of slaughter and stored or transported without further chilling. A disadvantage may be that the belly in whole carcasses, and loin and belly in pork sides, is frozen.

In contrast to batch chilling, a continuous refrigeration system operates at constant loading and can be specified to operate at full power at very high efficiencies. Hot deboning processes allow better control over chilling rates and facilitate the development of continuous line chilling systems. Sow meat is hot boned in the USA for sausage manufacture, but commercial application to prime pork is not widespread because hot boning systems do not produce conventional joints.

Weight loss

The average energy cost of chilling pork in the UK is 0.18 p/kg and the cost of evaporative weight loss is 2.68 p/kg, some 15 times higher (Collett and Gigiel, 1986; Brown and James, 1988). Overall profits of meat wholesalers are about 2% of turnover so a weight saving of 1% in the chills can increase profits by up to 50%. To reduce evaporative weight loss during chilling and storage of carcasses, the surface temperature must be quickly lowered thereby minimising the vapour pressure difference between the meat surface and the air which is the driving force for evaporation (Gigiel and Badran, 1988). Once the surface temperature has been reduced, further heat transfer is limited by the rate at which heat can flow to the surface from the deeper parts of the carcass. During this second period, the relative humidity of the air becomes important and minimum weight loss is achieved by using a low air velocity and high relative humidity. After the pig has been cooled, similar conditions are required during storage.

Batch cooling systems for pigs show an average weight loss, measured over 24 h, of between 1.9 and 3.5% (Table 4). Incorporation of a pre-chill tunnel operating at sub-zero temperatures can reduce average weight loss during chilling to between 1.0 and 1.5%. In Denmark a three-stage system reduced evaporative weight loss to 0.7%. The first two stages are conveyerized tunnels, running at -18°C and -5°C respectively, which remove the majority of the heat before the temperatures are allowed to equalise in the holding chill (see James *et al.*, 1983).

Ultra-rapid chilling using air at -30 to -40°C and 1 m/sec can chill pork carcasses in 4 h with only a 1% loss in weight (James *et al.*, 1983; Gigiel and James, 1984). However, the potential saving was lost overnight unless the carcass was jointed and packaged. Long and Tarrant (1989) achieved consistent savings in overnight chill loss by holding at -20°C at less than 0.5 m/sec for 2 or 3 h before conventional chilling (Table 4).

In immersion chilling, pork sides are hot jointed into primals, vacuum packed and chilled by immersion in brine at 0°C or below (James *et al.*, 1988). The potential advantages of immersion chilling include shorter chilling time, decreased chill loss and a reduction of space and energy requirements when compared with conventional 24 h chilling (Brown *et al.*, 1988).

Ultra-rapid blast chilling systems and immersion chilling systems both require large investment in new plant. A less costly way to reduce chill loss is to increase the humidity of the air in the chilling system. This can be achieved using ice bank refrigeration systems that produce air at 0°C and 99% RH (Gigiel and Badran, 1988). Alternatively carcasses can be spray chilled, a process in which water lost by evaporation is replaced by spraying a small amount of water onto the surface of the carcasses at intervals during cooling. Ice bank and spray chilling are easier to integrate with existing systems and can produce significant weight savings (Table 4). They have little influence on meat quality (Jones *et al.*, 1988b) since the actual cooling rates achieved in the meat with both methods are reasonably similar to those achieved by conventional batch chilling, but spray chilling can adversely affect rind-side appearance.

Table 4. Evaporative weight losses in pig sides during chilling

Chilling regime	Nominal cycle time (h)	Chill loss (%)
1. Conventional chill (+4°C)	24	1.9 to 3.5
2. -40°C, then conventional	24	2.0
3. -20°C, then conventional	24	1.7
4. -20°C, then conventional	24	1.5
5. High humidity chilling	24	1.9
6. Spray chilling	24	1.0 ^a 0.6 ^b
7. Ultra-rapid chill	4	1.1
8. Ultra-rapid chill	4	1.0

1.	4°C, 0.5 m/sec, 90% RH (Gigiel, 1984);
2.	-40°C, 1 m/sec for 80 min, then conventional (Gigiel and James, 1984);
3.	-20°C, <0.5 m/sec for 2 h, then conventional (Long and Tarrant, 1989);
4.	-20°C, <0.5 m/sec for 3 h, then conventional (Long and Tarrant, 1989);
5.	2°C, 1.5 m/sec decreasing to 0.4 m/sec, 99% RH (Gigiel and Badran, 1988);
6.	^a 4°C, 0.3 m/sec, carcasses sprayed with 250 ml of water every 20 min for first 6 h of chilling (Brown and James, 1988);
	^b 1°C, 0.5 m/sec, carcasses sprayed for 60 sec every 15 min for the first 10 h of cooling (Jones <i>et al.</i> , 1988b);
7.	-30°C, 1 m/sec (James <i>et al.</i> , 1983);
8.	-40°C, 1 m/sec for 80 min, then conventional for 130 min (Gigiel and James, 1984).

Meat quality

PSE

Borchert and Briskey (1964) originally suggested that the PSE condition could be prevented if the pork muscles were chilled quickly enough. Using hot-boned pork muscles, Honikel (1987a) noted that fast chilling reduced drip loss and paleness of PSE material but not sufficiently to give the quality attributes of normal pork. He concluded that fast chilling after hot boning can diminish but not solve the PSE problem.

Drip

Some investigators have found a reduction in drip from pork cuts with faster chilling (Taylor and Dant, 1971), while others have found the positive effect to be insignificant (Table 5). However, ultra-rapid chilling has a negative effect on drip. This

apparently contradictory finding may be explained by the phased response of pre-rigor muscle to falling temperature:

- (1) Cooling pork down to about 16°C is beneficial and the faster this is done the lower the drip loss will be. Taylor and Dant (1971) achieved a twofold reduction in drip from pork joints by "quick cooling" sides (in still air at 0°C) compared to conventional batch cooling. Less denaturation of proteins and consequently reduced shrinkage of the myofibrillar lattice is the probable explanation for the improvement in water binding in pork cooled quickly to 16°C.
- (2) Faster cooling to temperatures near 0°C before rigor, causes cold-shortening of the muscles with increased drip loss and toughening. Honikel (1987b) reported a linear relationship between muscle sarcomere length and drip loss in pork pieces, with drip doubling as sarcomeres shortened to half the resting length. The increase in drip may be sufficient to eliminate any prior advantage gained from rapid cooling to 16°C, therefore, cooling to 0°C should not occur pre-rigor.
- (3) A further disadvantage accrues in ultra-rapid chilling systems where partial or "crust" freezing of the meat surface occurs. A fourfold increase in pork drip was found by James *et al.* (1983) and the increase in drip was proportional to the amount of meat surface frozen (Londahl and Eek, 1986). The increase in drip is caused by disruption of the ultrastructure of the meat by the growth of ice crystals, thereby facilitating the exudation of cell water. The use of carbon dioxide ice or snow in fresh meat chilling has limited application because partial freezing is unavoidable with cryogenic systems.

Table 5. Effect of chilling regime on pork quality

Chilling regime	Drip	Paleness	Toughness	Reference ¹
-40°C, then conventional	unchanged	unchanged	increased	2
-20°C, then conventional	unchanged	decreased	unchanged	3
-20°C, then conventional	unchanged	decreased	unchanged	4
High humidity chilling	unchanged	unchanged	unchanged	5
Spray chilling	unchanged	unchanged	unchanged	6
Ultra-rapid chill	increased	decreased	increased	7
Ultra-rapid chill	unchanged	unchanged	increased	8
Electrical stimulation and ultra-rapid chill	increased	increased	decreased	8

¹See Table 4 for references 2-8

After chilling and cutting of pork carcasses, the holding temperature during distribution is important. Cuts held at +10°C for four days lost about 3% drip compared with half that amount for cuts held at 2.5°C (Londahl and Eek, 1986).

Paleness

Rapid chilling reduces paleness in pork and the tendency of PSE (Table 5). This was shown by lower probe reflectance values in pork sides pre-chilled at sub-zero temperatures (James *et al.*, 1983; Long and Tarrant, 1989). Although the beneficial effect is small it is nonetheless important in view of the strong discrimination against pale pork in some trade outlets.

Toughness

The main risk in ultra-rapid chilling systems is increased toughness (Table 5). This effect may be explained by physical shortening of the muscles as the temperature approaches 0°C pre-rigor, together with a slowdown of the natural enzymic conditioning process in meat at lower temperatures (Bouton *et al.*, 1984; Troy and Tarrant, 1987).

Increased toughness is associated with ultra-rapid chilling systems employing blasts at -30°C or lower and is sufficient to deter the commercial application of the process (James *et al.*, 1983; Brown and James, 1988). Toughening is greater in carcasses with a slow development of rigor mortis (Moller and Vestergaard, 1988), that is, in carcasses with high pH₁ values (6.1 and above are at risk) but with normal final pH values (Barton-Gade *et al.*, 1987). An investigation at Danish bacon factories using pre-chilling tunnels operating at -25°C and average air speeds of 3 m/sec showed that toughening occurred in certain factories but not in others (Barton-Gade *et al.*, 1987). The most likely explanation was irregular air blasts in some pre-chillers. A blast of 12-15 m/sec immediately under the ventilators resulted in cold toughening of the meat at one of the plants.

Cold-toughening in pork was prevented by electrical stimulation of the carcass after bleeding (Gigiél and James, 1984). However, the detrimental effects of electrical stimulation included a substantial increase in paleness and a fourfold increase in drip in retail packs. An alternative means of preventing cold-induced toughening in ultra-rapid chilling systems is to introduce a delay before chilling. In hot boned pork loins, conditioning at 15°C for 5 h before immersion chilling at 0°C improved tenderness (Moller and Vestergaard, 1988). Conditioning temperatures above 15°C should be avoided due to increased paleness and drip. Brown *et al.* (1988) proposed that toughening of hot cut, vacuum packed pork primals during immersion chilling at 0°C could be alleviated by using a two-stage immersion process, with higher fluid temperature in the first stage.

Moller and co-workers (1987) concluded that slow chilling produced the most tender pork but was associated with a higher risk of PSE, higher bacterial counts and greater weight loss. Pelvic suspension (rather than the conventional Achilles tendon) combined with fast chilling (-18°C, 3 m/sec for 65 min and then conventional) gave an acceptable degree of tenderness.

Conclusions

It is hardly an exaggeration to say that the 24 h before and after slaughter are the most important with respect to meat quality. The yield of carcass and prepared meats, the tenderness, colour, drip and keeping quality of the meat, the problems of rindside damage and blood splash may all benefit from the control of transport, slaughter and chilling operations. There is now a good body of knowledge on which to base recommendations for achieving an excellent quality and yield of end product. Working back from the end product, and bearing in mind the essential welfare requirements of both workers and animals, it is possible to define optimum procedures at each step. Many of the difficulties arise during preslaughter handling. Here, the proverb "*is treise an duchar na an oiliuint*" (instinct is stronger than teaching) is a useful guide for the management of both animals and their handlers.

References

- ANDERSSON, B.E. (1984). In "Duke's Physiology of Domestic Animals" tenth edition, pp. 719-727, ed. M.J. Swenson (Cornell University Press: Ithaca).
- ANONYMOUS. (1985). Code of Accepted Farming Practice for the Welfare of Livestock. *Victorian Government Gazette*. Number 91:3381-3399.
- ANONYMOUS. (1988a). "Livestock Handling Guide" (Livestock Conservation Institute: Madison, Wisconsin).

- ANONYMOUS. (1988b). "Livestock Trucking Guide" (Livestock Conservation Institute: Madison, Wisconsin).
- ANONYMOUS. (1988c). "Open Day 1988" (Cambac JMA Research Ltd., Unit 4, Wards Farm: Greenmore Lane, Woodcote, Reading, RG8 0RB, UK).
- AUGUSTINI, C., FISCHER, K. and SCHON, L. (1977). Welche Information Konnen Unmittelbar vor der Schlachtung Erhobene Physiologische Messwerte Uber Die zu Erwartende Fleischbeschaffenheit Geben? *Fleischwirtschaft*. 57:1028-1033.
- AUGUSTINI, C. AND FISCHER, K. (1982). In "Transport of Animals Intended for Breeding, Production and Slaughter" pp. 125-135, ed. R. Moss (Martinus Nijhoff: The Hague).
- BARFOD, K. and MADSEN, K.B. (1988). In "Workshop on Stunning of Livestock" (Proceedings 34th International Congress of Meat Science and Technology: Brisbane), pp. 6-7, eds. C.E. Devine and F.D. Shaw.
- BARTON-GADE, P. (1984). Influence of halothane genotype on meat quality in pigs subjected to various preslaughter treatments. *Proceedings European Meeting of Meat Research Workers*. 30:8-9.
- BARTON-GADE, P., BEJERHOLM, C. and BORUP, V. (1987). Influence of different chilling procedures on the eating quality of pork chops. (Proceedings 33rd International Congress of Meat Science and Technology: Helsinki), pp. 181-184, ed. E. Petaja.
- BARTON-GADE, P.A., BUSK, H. and PEDERSEN, O.K. (1982). Influence of different transport times on the meat quality of pigs of known pedigree. *Proceedings 28th European Meeting of Meat Research Workers*, paper 1.07, 1:24-27 (Instituto del Frio, CSIC: Madrid, Spain).
- BECKER, B.A., MAYES, H.F., HAHN, G.L., NIENABER, J.A., JESSE, G.W., ANDERSON, M.E., HEYMANN, H. and HEDRICK, H.B. (1989). Effect of fasting and transportation on various physiological parameters and meat quality of slaughter hogs. *Journal of Animal Science*. 67: 334-341.
- BENDALL, J.R. and SWATLAND, H.J. (1988). A review of the relationships of pH with physical aspects of pork quality. *Meat Science*. 24:85-126.
- BENNETT, M.E., BRAMBLETT, V.D., ABERLE, E.D. and HARRINGTON, R.B. (1973). Muscle quality, cooking method and aging vs. palatability of pork loin chops. *Journal of Food Science*. 38:536-538.
- BLACKMORE, D.K. (1988). In "Workshop on Stunning of Livestock" (Proceedings 34th International Congress of Meat Science and Technology: Brisbane), pp. 48-49, eds. C.E. Devine and F.D. Shaw.
- BORCHERT, L.L. and BRISKEY, E.J. (1964). Prevention of pale, soft and exudative porcine muscle through partial freezing with liquid nitrogen post-mortem. *Journal of Food Science*. 29:203-209.
- BOUTON, P.E., HARRIS, P.V. and SHORTHORSE, W.R. (1984). Electrical stimulation of mutton. *Journal of Food Science*. 49:1011-1017.
- BROWN, T., GIGIEL, A.J., SWAIN, M.V.L. and HIGGINS, J.A. (1988). Immersion chilling of hot cut, vacuum packed pork primals. *Meat Science*. 22:173-188.
- BROWN, T. and JAMES, S.J. (1988). "Process Design Data for Pork Chilling" (AFRC Institute of Food Research, Subject Day, February 23rd 1988, Meat Chilling: Bristol).
- COLLETT, P. and GIGIEL, A.J. (1986). In "Energy Usage and Weight Loss in Beef and Pork Chilling" p. 119, ed. C. Bailey (IIR Commission C2, 'Meat Chilling': Bristol) (International Institute of Refrigeration: Paris).
- DANTZER, R. (1982). In "Transport of Animals Intended for Breeding, Production and Slaughter" pp. 218-231, ed. R. Moss (Martinus Nijhoff: The Hague).
- DEVINE, C. (1988). In "Workshop on the Stunning of Livestock" (Proceedings 34th International Congress of Meat Science and Technology: Brisbane), pp. 50-54, eds. C.E. Devine and F.D. Shaw.
- EIKELENBOOM, G. (1988). "Pig Carcass and Meat Quality" (Proceedings: Universita di Bologna), pp. 199-211.
- FORSLID, A. (1987). Transient neocortical, hippocampal and amygdaloid EEG silence induced by one minute inhalation of high concentration CO₂ in swine. *Acta Physiologica Scandinavica*. 130:1-10.
- FORSLID, A. (1988). In "Workshop on Stunning of Livestock" (Proceedings 34th International Congress of Meat Science and Technology: Brisbane), pp. 8-10, eds. C.E. Devine and F.D. Shaw.
- GALLWEY, W.J. and TARRANT, P.V. (1979). Influence of environmental and genetic factors on ultimate pH in commercial and purebred pigs. *Acta Agricultura Scandinavica*. 21:32-38.
- GERBER, H.D. (1984). "Inaugural Dissertation, Free University of Berlin". Abstracted in *Veterinary Bulletin*. 55:729 (Abstract 5951).
- GIGIEL, A.J. (1984). Energy consumption and weight loss in pig chilling, 2.7 (Proceedings 30th Meeting of the European Meat Research Workers: Bristol), pp. 63-64 (Meat Research Institute: Bristol, UK).
- GIGIEL, A. and BADRAN, R. (1988). Chilling and storage of pig carcasses using high humidity air as produced by an ice bank cooler. *Journal of Refrigeration*. 11:100-104.

- GIGIEL, A.J. and JAMES, S.J. (1984). Electrical stimulation and ultra-rapid chilling of pork. *Meat Science*. 11:1-12.
- GONZALEZ, A.M., VENEGAS, O. and BENCOMO, E. (1987). Effect of preslaughter holding time on pork yield and quality. (Proceedings 33rd International Congress of Meat Science and Technology: Helsinki), pp. 105-107, ed. E. Petaja.
- GRANDIN, T. (1988a). Hog psychology: An aid in handling. *Agri-Practice Behaviour*. 9:4.
- GRANDIN, T. (1988b). In "Workshop on Stunning of Livestock" (Proceedings 34th International Congress of Meat Science and Technology: Brisbane), pp. 13-14, eds. C.E. Devine and F.D. Shaw.
- GREER, G.G. and MURRAY, A.C. (1988). Effects of pork muscle quality on bacterial growth and retail case life. *Meat Science*. 24:61-71.
- GREGORY, N.G. (1987). In "Evaluation and Control of Meat Quality in Pigs" pp. 265-272. eds. P.V. Tarrant, G. Eikelenboom and G. Monin (Martinus Nijhoff: Dordrecht).
- GREGORY, N.G. (1988a). In "Workshop on Stunning of Livestock" (Proceedings 34th International Congress of Meat Science and Technology: Brisbane), pp. 55-58, eds. C.E. Devine and F.D. Shaw.
- GREGORY, N.G. (1988b). In "Workshop on Stunning of Livestock" (Proceedings 34th International Congress of Meat Science and Technology: Brisbane), pp. 59-62, eds. C.E. Devine and F.D. Shaw.
- GUISE, J. (1987). Moving pigs from farm to factory. *Pig International*. December, pp. 8-12.
- GUISE, H.J. and PENNY, R.H.C. (1989a). Factors influencing the welfare and carcass and meat quality of pigs: I The effects of stocking density in transport and the use of electric goods. *Animal Production*. In press.
- GUISE, H.J. and PENNY, R.H.C. (1989b). Factors influencing the welfare and carcass and meat quality of pigs: II Mixing pigs with unfamiliar animals. *Animal Production*. In press.
- GUISE, H.J. and WARRISS, P.D. (1989). The effect of stocking density and temperature on meat quality in pigs. *Animal Production*. 48:In press.
- HEINZ, P.H., GOUWS, P.J. and NAUDE, R.T. (1984). The influence of various factors on the occurrence of high ultimate pH values as an indication of dark, firm and dry (DFD) pork at a South African bacon factory. *South African Journal of Animal Science*. 14:97-104.
- HOENDERKEN, R. In "Stunning of Animals for Slaughter" pp. 59-63, ed. G. Eikelenboom (Martinus Nijhoff: The Hague).
- HONIKEL, K.O. (1987a). In "Evaluation and Control of Meat Quality in Pigs" pp. 273-283, eds. P.V. Tarrant, G. Eikelenboom and G. Monin (Martinus Nijhoff: Dordrecht).
- HONIKEL, K.O. (1987b). In "Evaluation and Control of Meat Quality in Pigs" pp. 129-142, eds. P.V. Tarrant, G. Eikelenboom and G. Monin (Martinus Nijhoff: Dordrecht).
- JAMES, S.J., GIGIEL, A.J. and HUDSON, W.R. (1983). The ultra-rapid chilling of pork. *Meat Science*. 8:63-78
- JAMES, S.J., GIGIEL, A.J., BROWN, T. and BAILEY, C. (1988). A comparison of ultra-rapid and immersion chilling of pork (IIF-IIR, Section B, Commission C, Section D: Wageningen).
- JEREMIAH, L.E. (1984). A note on the influence of inherent muscle quality on cooking losses and palatability attributes of pork loin chops. *Canadian Journal of Animal Science*. 64:773-775.
- JONES, S.D.M., MURRAY, A.C., SATHER, A.P. and ROBERTSON, W.M. (1988a). Weight changes in pigs fasted for different periods of time prior to slaughter. *The Research Bulletin* (Canadian Meat Council: Ontario). 15:7-8.
- JONES, S.D.M., MURRAY, A.C. and ROBERTSON, W.M. (1988b). The effects of spray chilling pork carcasses on the shrinkage and quality of pork. *The Research Bulletin* (Canadian Meat Council: Ontario). 15:9-11.
- KAUFFMAN, R.G., WACHHOLZ, D., HENDERSON, D. and LOCHNER, J.V. (1978). Shrinkage of PSE, normal and DFD hams during transit and processing. *Journal of Animal Science*. 46:1236-1240.
- LAMBOOY, E., GARSSEN, G.J., WALSTRA, P., MATEMAN, F. and MERKUS, G.S.M. (1985). Transport of pigs by car for two days: Some aspects of watering and loading density. *Livestock Production Science*. 13:289-299.
- LAMBOOY, E. and SYBESMA, W. (1988). In "Workshop on Stunning of Livestock" (Proceedings 34th International Congress of Meat Science and Technology: Brisbane), pp. 20-22, eds. C.E. Devine and F.D. Shaw.
- LARSEN, H.K. (1983). In "Stunning of Animals for Slaughter" pp. 73-81, ed. G. Eikelenboom (Martinus Nijhoff: The Hague).
- LAURSEN, A.M. (1983). In "Stunning of Animals for Slaughter" pp. 64-72, ed. G. Eikelenboom (Martinus Nijhoff: The Hague).
- LINDSAY, D.B. (1981). In "The Problem of Dark-cutting in Beef" pp. 101-121, eds. D.E. Hood and P.V. Tarrant (Martinus Nijhoff: The Hague).

- LONDAHL, G. and EEK, L. (1986). "Cooling of Meat Cuts" pp. 243-252, ed. C. Bailey (IIF-IIR, Commission C2: Bristol, UK) (International Institute of Refrigeration: Paris).
- LONG, V.P. and TARRANT, P.V. (1989). The effect of pre-slaughter showering and post-slaughter rapid chilling on meat quality in intact pork sides. *Meat Science*. in press.
- LUNDSTROM, K., HANSSON, I. and BJARSTORP, G. (1987). In "Evaluation and Control of Meat Quality in Pigs" pp. 165-173, eds. P.V. Tarrant, G. Eikelenboom and G. Monin (Martinus Nijhoff: Dordrecht).
- LUNDSTROM, K., BARTON-GADE, P., RUD ANDERSEN, J. and HANSSON, I. (1988). Pale pig meat - relative influence of PSE and low pigment content (Proceedings 34th International Congress of Meat Science and Technology: Brisbane), pp. 584-587, eds. C.S. Chandler and R.F. Thornton.
- MALMFORS, G. (1982). Studies on some factors affecting pigmeat quality. (Proceedings 28th European Meeting of Meat Research Workers), pp. 21-23 (Instituto del Frio, CSIC: Madrid, Spain).
- MAYES, H., HAHN, L., NIENABER, J., JESSE, G.C., ANDERSON, M., BECKER, A., HEYMANN, H., BRYAN, R. and HEDRICK, H.B. (1988). Effect of preslaughter fast and transportation of pigs on weight loss and meat quality (Proceedings International Congress of Meat Science and Technology: Brisbane), pp. 145-147, eds. C.S. Chandler and R.F. Thornton.
- MLC. (1980). "Handling Pigs from Farm to Slaughterhouse" (Technical Bulletin Number 14. Meat and Livestock Commission: Bletchley, UK).
- MLC. (1986). "Meat and Marketing Technical Notes, No. 7" (Meat and Livestock Commission: Bletchley, UK).
- MOLLER, A.J., KIRKEGAARD, E. and VESTERGAARD, T. (1987). Tenderness of pork muscles as influenced by chilling rate and altered carcass suspension. *Meat Science*. 21:275-286.
- MOLLER, A.J. and VESTERGAARD, T. (1988). Effect of temperature conditioning on toughness in hot boned pork loins with high or low initial pH (Proceedings International Congress of Meat Science and Technology: Brisbane), pp. 621-623, eds. C.S. Chandler and R.F. Thornton.
- MONIN, G. and SELLIER, P. (1987). In "Evaluation and Control of Meat Quality in Pigs" pp. 447-458, eds. P.V. Tarrant, G. Eikelenboom and G. Monin (Martinus Nijhoff: Dordrecht).
- MOSS, B.W. (1986). Cut carcass damage. *Farmers Weekly*. 104:17.
- NEWTON, K.G. and GILL, C.O. (1981). The microbiology of DFD fresh meats: A review. *Meat Science*. 5:223-232.
- NIELSEN, N.J. (1982). In "Transport of Animals Intended for Breeding, Production and Slaughter" pp. 115-124, ed. R. Moss (Martinus Nijhoff: The Hague).
- OFFER, G. and TRINICK, J. (1983). On the mechanism of water-holding in meat: The swelling and shrinkage of myofibrils. *Meat Science*. 8:245-281.
- REY, C.R., KRAFT, A.A., TOPEL, D.G., PARRISH, F.C. and HOTCHKISS, D.K. (1976). Microbiology of pale, dark and normal pork. *Journal of Food Science*. 41:111-116.
- RING, C. (1988). In "Workshop on Stunning of Livestock" (Proceedings of 34th International Congress of Meat Science and Technology: Brisbane), pp. 15-19, eds. C.E. Devine and F.D. Shaw.
- RING, C. and BLENDL, H.M. (1984). Containertransport von schlachtschweiner. *Fleischwirtschaft*. 64:1058-1062.
- SAHLIN, K., ALVESTRAND, A., BRAND, R. and HULTMAN, E. (1978). Intracellular pH and bicarbonate concentration in human muscle during recovery from exercise. *Journal of Applied Physiology*. 45:474-480.
- SAINS, A.G. (1980). Deaths in transit: What British surveys show. *Pig Farming*. 28:40-41.
- SCHMITTEN, F., SCHEPERS, K.H., and FESTERLING, A. (1987). In "Control and Evaluation of Meat Quality in Pigs" pp. 191-200, eds. P.V. Tarrant, G. Eikelenboom and G. Monin (Martinus Nijhoff: Dordrecht).
- SEIDLER, D., BARTNICK, E. and NOWAK, B. (1987). In "Evaluation and Control of Meat Quality in Pigs" pp. 175-190, eds. P.V. Tarrant, G. Eikelenboom and G. Monin (Martinus Nijhoff: Dordrecht).
- SMITH, W.C. and LESSER, D. (1982). An economic assessment of pale, soft, exudative musculature in the fresh and cured pig carcass. *Animal Production*. 34:291-299.
- SMULDERS, F.J.M., ROMME, A.M.C.S., WOOLTHUIS, C.H.J., DE KRUIJF, J.M., EIKELBOOM, G. and CORSTIAENSEN, G.P. (1983). In "Stunning of Animals for Slaughter" pp. 90-95, ed. G. Eikelenboom (Martinus Nijhoff: The Hague).
- SOMERS, C., TARRANT, P.V. and SHERINGTON, J. (1985). Evaluation of some objective methods for measuring pork quality. *Meat Science*. 15:63-76.
- SWATLAND, H.J. (1987). In "Evaluation and Control of Meat Quality in Pigs" pp. 143-163, eds. P.V. Tarrant, G. Eikelenboom, G. Monin (Martinus Nijhoff: Dordrecht).
- SWATLAND, H.J., IRVING, T.C. and MILLMAN, B.M. (1989). Fluid distribution in pork, measured by x-ray diffraction, interference microscopy and centrifugation compared to paleness measured by fibre optics. *Journal of Animal Science*. 67:1465-1470.

- TARRANT, P.V. (1988). Le stress du transport chez les animaux de ferme. *Recueil de Medecine Veterinaire*. 164:623-633.
- TARRANT, P.V. and LONG, V.P. (1986). On line and non-destructive methods to measure meat characteristics (Proceedings of the CEC Workshop, November 5-6, 1986: Theix), ed. C. Valin.
- TAYLOR, A.A. and DANT, S.J. (1971). Influence of carcass cooling rate on drip loss in pigmeat. *Journal of Food Technology*. 6:131-139.
- TOPEL, D.G., MILLER, J.A., BERGER, P.J., RUST, R.E., PARRISH, F.C. and ONO, K. (1976). Palatability and visual appearance of dark, normal and pale coloured porcine *M. longissimus*. *Journal of Food Science*. 41:628-630.
- TROEGER, K. and WOLTERS DORF, W. (1989). Measuring stress in pigs during slaughter. *Fleischwirtschaft*. 69:373-376.
- TROY, D.J. and TARRANT, P.V. (1987). Changes in myofibrillar proteins from electrically stimulated beef. *Biochemical Society Transactions*. 15:297-298.
- VAN LOGTESTIJN, J.G., ROMME, A.M.T.C. and EIKELENBOOM, G. (1982). In "Transport of Animals Intended for Breeding, Production and Slaughter" pp. 105-114, ed. R. Moss (Martinus Nijhoff: The Hague).
- VAN PUTTEN, G. (1982). In "Transport of Animals Intended for Breeding, Production and Slaughter" pp. 15-27, ed. R. Moss (Martinus Nijhoff: The Hague).
- VAN DER WAL, P.G. (1976). Bone fractures in pigs as a consequence of electrical stunning. *Proceedings of 22nd European Meat Research Workers*. 1(C3):1-4 (Technical Secretariat, Swedish Meat Research Centre: Kavlinge, Sweden).
- VON MICKWITZ, G. (1982). In "Transport of Animals Intended for Breeding, Production and Slaughter" pp. 45-56, ed. R. Moss (Martinus Nijhoff: The Hague).
- WACHHOLZ, D., KAUFFMAN, R.G., HENDERSON, D. and LOCHNER, J.V. (1978). Consumer discrimination of pork colour at the marketplace. *Journal of Food Science*. 43:1150-1152.
- WARNER, R.D. and ELDRIDGE, G.A. (1988). Preliminary observations of pig meat quality problems in a Victorian abattoir (Proceedings of 34th International Congress of Meat Science and Technology: Brisbane), pp. 573-574, eds. C.S. Chandler and R.F. Thornton.
- WARRINGTON, R. (1974). Electrical stunning: A review of the literature. *The Veterinary Bulletin*. 44:617-635.
- WARRISS, P.D. (1984). The incidence of carcass damage in slaughter pigs. Proceedings of European Meeting of Meat Research Workers. (Meat Research Institute: Bristol, UK). 30:17-18
- WARRISS, P.D. (1987). In "Evaluation and Control of Meat Quality in Pigs" pp. 245-264. eds. P.V. Tarrant, G. Eikelenboom and G. Monin (Martinus Nijhoff: Dordrecht).
- WARRISS, P.D., DUDLEY, C.P. and BROWN, S.N. (1983). Reduction of carcass yield in transported pigs. *Journal of the Science of Food and Agriculture*. 34:351-356.
- WARRISS, P.D. and BEVIS, E.A. (1987). Liver glycogen in slaughtered pigs and estimated time of fasting before slaughter. *British Veterinary Journal*. 143:354-360.
- WARRISS, P.D., BROWN, S.N., FRANCOMBE, M.A. and HIGGINS, J.A. (1987). Effect of preslaughter fasting on the characteristics of pig liver. *International Journal of Food Science and Technology*. 22: 255-263.
- ZELLER, W., SCHATZMANN, U. and IMHOF, A. (1987). *Die Fleischwirtschaft*. 67:1519.

EVALUATION OF NEAR INFRA-RED SPECTROPHOTOMETRY AS A METHOD FOR DETERMINING THE NITROGEN AND ENERGY CONTENT OF PIG CARCASSES

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Near infra-red spectrophotometry has potential as a rapid method for determining the nitrogen (N) and gross energy (GE) content of pig carcasses at slaughter. In a previous experiment (George *et al.*, 1987) a NIR spectrophotometer (Technicon 400 Infraalyzer) was calibrated to predict the N and GE content of both carcass and viscera (empty viscera and blood) samples of known composition. However, these calibrations have not been tested using samples outside of the calibration set. The aim of this experiment was to determine how accurately the calibrated NIR spectrophotometer could predict the N and E content of samples collected from outside the calibration set.

Samples were collected from 44 grower pigs slaughtered at 45 kg live weight and were prepared in the same way as samples used in the calibration (George *et al.*, 1987). They were freeze-dried and chemically analysed for N content by kjeldahl analysis and for GE content by adiabatic bomb calorimetry. Each carcass (3.92-6.85 gN/kg, 27.6-33.2 MJ GE/kg) and viscera sample (6.13-9.5 gN/kg, 23.0-29.8 MJ GE/kg) was then analysed for N and E content using NIR spectrophotometry and the results were compared.

There was a high correlation between the results predicted by NIR spectrophotometry and those obtained by conventional analyses (Table 1). The standard error of prediction (SEP, standard deviation of the differences between laboratory and predicted values) were all less than five percent of the mean.

Table 1. Correlation between laboratory and predicted values for the N and GE content of carcass and viscera samples

Sample type	Mean		Correlation coefficient	SEP	SEP as % of mean
	laboratory	predicted			
Viscera					
N (g/kg)	77.6	81.1	0.98	3.79	4.9
GE (MJ/kg)	26.9	26.9	0.97	0.794	3.0
Carcass					
N (g/kg)	53.2	54.2	0.97	2.03	3.8
GE (MJ/kg)	30.7	31.1	0.95	0.572	1.9

These results confirm that NIR spectrophotometry provides a rapid and accurate method for determining the N and GE content of pigs at slaughter.

References

- GEORGE, S.A., McALPINE, B., ELLIOTT, R. and BATTERHAM, E.S. (1987). In "Manipulating Pig Production" p. 154, eds. APSA Committee (Australasian Pig Science Association, Werribee: Victoria, Australia).

THE DEVELOPMENT OF IMMUNOASSAYS FOR ANTIBIOTIC RESIDUES IN FOODSTUFFS

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Potential effects on human health and the development of international trade have lead to chemical contamination of foodstuffs destined for human consumption becoming an issue of increasing concern throughout the world. Veterinary drugs, including antibiotics, sulphonamides, anthelmintics and hormones are used extensively in animal production to prevent and treat disease and for growth promotion. Currently available methods for detecting and identifying drug residues are inadequate for large scale testing.

We aim to develop a sensitive immunoassay screening test for the penicillins, tetracyclines, sulphonamides and benzimidazoles for on-site use to monitor the extent of chemical contamination and encourage the production of residue-free carcasses.

An essential reagent in any immunoassay is an antibody to the component being measured. The antibiotics, sulphonamides and benzimidazoles are small molecules and have been bound to protein in order to provoke an immunological response when injected into experimental animals. The parent drugs, or drug-haptens (the drug with reactive functional groups attached) have been conjugated to proteins by modifying existing conjugation procedures, taking into account the chemistry and reactivity of the drugs and attempting to retain their existing chemical structures. A summary of successful conjugation procedures and antibodies produced can be seen in Table 1.

Table 1. Drug-protein conjugation procedures and polyclonal antibodies produced

Drug/hapten-protein	Conjugation procedure	Antibody titre
Sulphonamide	Carbodiimide	High
Sulphamethazine	Diazotization	High
	Gluteraldehyde	High
	Diazotization	High
Suphamerazine	Gluteraldehyde	High
	Mixed anhydride	Low
Benzimidazole	Hydrolysis	Moderate

The proteins used in binding were bovine serum albumin, hen egg albumin, keyhole limpet haemocyanin and bovine thyroglobulin. Polyclonal antibodies were produced in rabbits and sheep. Monoclonal antibodies were produced for sulphamethazine. Antibody titres were measured by enzyme-linked immunosorbent assay. The sensitivity and specificity of each antibody were determined by an indirect competitive immunoassay.

To date the polyclonal antibodies have not demonstrated sufficient affinity for the parent drugs to detect them at levels approaching the maximum residue limit. Alternative binding procedures, use of monoclonal antibodies and homogeneous assays are other approaches being investigated.

Work is proceeding to develop sensitive tests for antibiotic and sulphonamide residues in Australia. Immunoassay technology should provide more effective tests for chemical residue monitoring.

EFFECT OF AMOUNT OF FEEDING ON PERSISTENCE OF DIELDRIN RESIDUES IN PIGS

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Piggeries have been quarantined because of dieldrin residues. Dieldrin is stable in most environments and is readily absorbed via the skin or gut and stored in fatty tissue. A strategy was required to reduce dieldrin concentrations. Pesticide released by fat catabolism is actively reabsorbed and stored (Hayes, 1965). Only minute amounts are excreted as bile salt conjugates in the faeces. The successful use of barbiturates as detoxicants has been described by Dobson and Baugh (1976) but would not be a suitable technique in piggeries. Therefore, it was postulated that fat deposition in the absence of further pesticide uptake should lead to a dilution effect; a feeding strategy was used to test this hypothesis.

Six baconer pigs from a quarantined batch were individually housed on concrete and fed a low protein, low energy (12.9 MJ DE/kg) dry sow diet either *ad libitum* or restricted to 1.5 times maintenance. The pigs were weighed weekly and fat biopsies from the perianal fat pad were residue tested four-weekly.

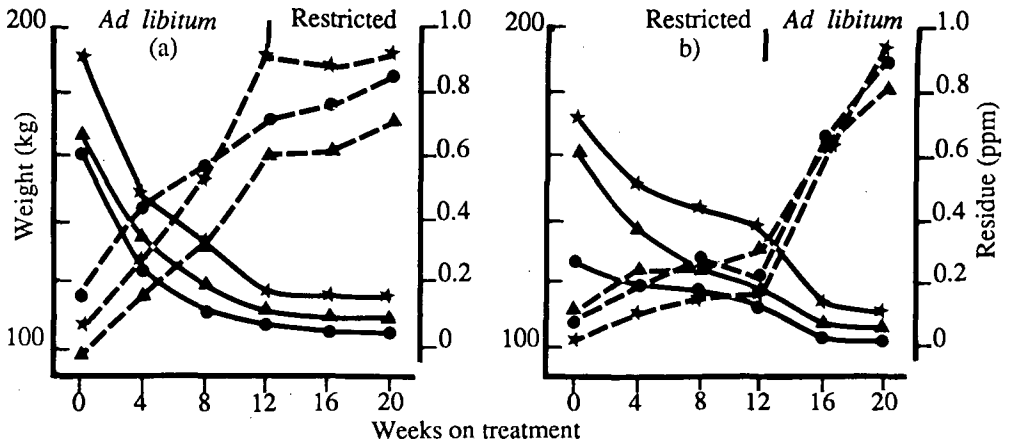


Figure 1. Live weight (broken lines) and residue (solid lines) changes for pigs (1-6) fed (a) *ad libitum*/restricted and (b) restricted/*ad libitum*.

The results demonstrate the desirable effect of *ad lib* feeding and establish a "half-life" of approximately four weeks for dieldrin residues. When pigs were swapped from one feeding method to the other, there was an immediate response seen at the next biopsy. Because the restrictively fed pigs still gained considerable weight in the first four week period, the feed allowance was reduced to 1.2 times maintenance. At slaughter, seven fat depot sites were biopsied. For each pig, the residue at each site was the same indicating even distribution in the body fat. Because minute residues are detectable, a nil level is almost impossible to achieve following contamination.

References

- DOBSON, R.C. and BAUGH, E.R. (1976). *Bulletin of Environmental Contamination Toxicology*. 16:567-571.
 HAYES, W.J. Jr. (1965). *Annual Review of Pharmacology*. 43:505-516.

RESTRICTION FRAGMENT LENGTH POLYMORPHISM (RFLP) IN THE DETECTION OF PORCINE STRESS SYNDROME (PSS) SUSCEPTIBLE AND CARRIER PIGS

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Porcine Stress Syndrome (PSS) is a genetically determined hypermetabolic state that follows a stress such as transportation, or anaesthesia with halothane. PSS often culminates in death, while additional economic losses occur due to the poor meat quality generally seen in PSS affected pigs (Chambers and Hall, 1987).

PSS has been described as an autosomal recessive trait with variable penetrance (Southwood *et al.*, 1986). The most widely used test for PSS at present is the halothane test, which is largely able to detect homozygous recessive (Hal^{nn}) pigs but not heterozygotes (Hal^{nN}). Thus eradication programs based on this test are of only limited efficiency.

The analysis of Restriction Fragment Length Polymorphisms (RFLPs) using *Hal* or closely linked genes as "probes", allows the potential for identification of both Hal^{nn} and Hal^{nN} pigs. This molecular genetic approach is based on the use of enzymes, called restriction enzymes, that cut DNA at specific nucleotide sequences. DNA is extracted from a blood sample and then "digested" by one of these enzymes. The lengths of the fragments produced are determined by the distances between cutting sites. An individual fragment may be detected, following gel electrophoresis and Southern transfer, with a radioactively-labelled segment of DNA (probe) that is homologous to the fragment of interest. If a mutation occurs in this fragment that destroys/creates one of the specific nucleotide sequences, the detected fragment will be longer/shorter. This change in length can be detected, and is termed an RFLP.

Glucosephosphate isomerase (*GPI*) is a gene in close linkage with *Hal*, and has recently been cloned by Davies *et al.* (1987). Using *GPI* as a probe, Davies' team was able to demonstrate RFLPs in Norwegian pigs, with enzymes *SacI* and *PvuII*. *GPI* has now been used in a similar, collaborative, study of Australian pigs. Ten different enzymes were tested on eight families from three populations of Large White or Belgian Landrace in which *Hal* is segregating. Using *PvuII*, we have detected an RFLP (6.0 or 6.6 kb) that appears to correspond well with the alleles observed by Davies (5.9, 6.1, 6.3, 6.9, and 7.2 kb). In addition we have seen RFLPs with enzymes *TaqI* (6.6 or 7.8 kb), and *MspI* (6.1 or 7.7 kb). In almost all families at least one RFLP has been useful in tracking the segregation of *Hal*. At this stage we have not detected any RFLPs with *SacI* although our single *SacI* band (9.6 kb) is in the vicinity of Davies' *SacI* allelic bands (8.1, 8.6, 8.8, 9.4, and 9.7 kb).

These preliminary results demonstrate the applicability of this technique, using a linked probe, to PSS heterozygote detection. The technique also provides a technical base from which further collaborative work, with a view to cloning *Hal* itself, will proceed.

References

- CHAMBERS, J. and HALL, R.R. (1987). *Compendium on Continuing Education for the Practicing Veterinarian*. 9:F317-326.
- DAVIES, W., HARBITZ, I., and HAUGE, J.G. (1987). *Animal Genetics*. 18:233-240.
- SOUTHWOOD, O.I., SIMPSON, S.P., and WEBB, A.J. (1986). In "Third World Congress on Genetics Applied to Livestock Production" volume XI pp.401-406, eds. G.E. Dickerson and R.K. Johnson (University of Nebraska: Lincoln, Nebraska).

A SYMPOSIUM - FACTORS AROUND SLAUGHTER AFFECTING THE QUALITY OF PIG MEAT

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Symposium introduction

In August 1988 the 34th International Congress of Meat Science and Technology was held in Brisbane. This was the first time this meeting (formerly the European Meat Workers Conference) had been staged in the southern hemisphere. This meeting, together with the associated workshops entitled "Dark-cutting in Cattle and Sheep", "Stunning of Livestock" and "Pig Meat Quality", focussed considerable research attention on meat quality as distinct from meat production for which Australia is world renowned. At about that time the Pig Research Council of Australia added further research focus to this area by commissioning a consultant to review pigmeat science and education in Australia.

Quality attributes of Australian pig meat have barely been researched and rarely have research workers in the field of pig meat production paid attention to quality aspects of the meat being produced. A major purpose of this symposium is to put the background and most recent findings of pig meat quality before the research and industry community interested in Australian pig meat production. Therefore, contributions to the symposium will cover the areas of microbiological contamination, storage life of vacuum packaged meat, factors which affect the manufacturing properties of pig meat and the influence of growth promotants/repartitioning agents on pig meat quality. Dr. V. Tarrant has been invited to present a review entitled "The effects of handling, transport, slaughter and chilling on meat quality and yield in pigs". This symposium will complement the review.

Thus, the symposium is broad in scope, ranging from reporting the latest findings of overseas researchers, to consideration of aspects that are of special concern to the Australian pig meat industry. The latter arise mainly from differences in husbandry practices, the relatively large distances that animals are often transported to slaughter and remoteness from export markets for pig meat.

Symposium continued on next page

GROWTH PROMOTANTS, REPARTITIONING AGENTS AND PIG MEAT QUALITY

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Introduction

In the 1980s there has been a surge of interest in the efficient production of lean meat from domestic livestock. This interest has been stimulated by the development and application of biologically-active pharmacological compounds, be they chemically synthesised, biologically synthesised (conventionally or by recombinant DNA techniques) or naturally occurring, e.g. porcine growth hormone (pGH). Some of these compounds have been shown to promote fast, efficient lean meat production in a variety of domestic livestock. Thus the current emphasis in research on the manipulation of growth in domestic livestock is not on growth rate per se, but on the differential rates of deposition of lean (muscle) and fat (adipose) in the efficient, rapidly growing animal.

However, fast efficient lean meat production is only worthwhile if the quality (in terms of flavour, tenderness, colour, texture, water holding capacity etc.) of the meat is highly desirable in the market place, i.e. meat quality should not be sacrificed for rapid growth rates of lean meat. Although many surveys of consumer preferences in the western world have indicated preference for meat with less fat from sheep, cattle and pigs, lean carcasses and lean meat can have unattractive appearances in display cabinets due to surface drying and discolouration. Kempster (1987) believes that some fat (2-3% in beef) is desirable for optimum meat quality and acceptability. Very lean pig carcasses have been criticised by both butchers and consumers for having soft fat, subcutaneous fat tissue separation, and meat lacking in juiciness and flavour (Kempster, 1987; Wood *et al.*, 1988). Such observations have led to the potential use of the Duroc breed of pigs, noted for its higher intramuscular fat (marbling) levels (Barton-Gade, 1988; Wood and Bichard, 1988).

A comprehensive review of the American literature on the influence of repartitioning agents and pGH on pig meat quality has recently been presented by Merkel (1988). In our paper, we integrate some of our data on this topic with that presented by Merkel (1988) and speculate on mechanisms by which the administration of such compounds could affect pig meat quality.

Porcine growth hormone (pGH) (\approx Porcine somatotrophin {pST})

Fat deposition and marbling

There is now good evidence that pGH (native or recombinant) treatment of pigs results in increased growth, food conversion, and muscle and protein deposition, and decreased food consumption and fat deposition. These responses are dose dependent (see Thornton and Tume, 1988). Although fat thickness at the P₂ site of carcasses in the 63-78 kg range is dramatically reduced, only high doses of pGH (9 mg/day) produced P₂ values of the order of 11 mm (see Table 1). This compares with 8 mm at the P₂ site which Wood *et al.* (1988) describe as "lean" and which had some deleterious effects. In the study of Hertzman *et al.* (1989), pGH treatment dramatically reduced subcutaneous fat deposition (which accounted for some 76% of the total reduction in carcass fat deposition) but subcutaneous fat consistency and marbling levels were unaffected. In the experiment of Bechtel *et al.* (1988), marbling levels were

reduced but remained "acceptable". However, very high doses of pGH, used by Beermann *et al.* (1988) (200 $\mu\text{g}/\text{kg}$ live weight/day) could produce carcasses with an unacceptable fat cover and texture for existing markets.

Objective meat quality attributes

There is no evidence that treatment with pGH (native or recombinant) has any deleterious effects on objective meat quality attributes (see Table 2). Muscle pH and shear force are unaffected by pGH treatment. Small changes in colour (slightly darker and less red) of *longissimus dorsi* muscle reported by Beermann *et al.* (1988) were visually not perceptible. Hertzman *et al.* (1989) found no differences between pGH treated and control animals in the colour attributes (L, a and b values) of *longissimus dorsi* or *semimembranosus* muscles.

Subjective, sensory attributes of meat quality from animals treated with pGH

Taste panel evaluation of samples of *longissimus dorsi* muscles have shown no differences in any sensory attributes of meat quality (flavour, tenderness, juiciness, aroma and overall acceptability) that have been examined (see Table 3).

Table 1. Responses of carcass variables to pGH treatment

Native pST treatment (mg/day)	0	3.0	6.0	9.0
Carcass weight (kg)	78.1	74.6	74.4	77.5
Loin eye area (cm ²)	34.7	39.3	37.7	38.5
P ₂ fat thickness (mm)	25.8	14.1	13.3	10.7
Marbling score (1-5)	3.0	2.4	2.3	2.6
(Bechtel <i>et al.</i> , 1988)				
Recombinant pGH treatment ($\mu\text{g}/\text{kg}$ live weight/day)	0			90
Carcass weight (kg)		63.6		68.1
Loin eye area (cm ²)		34.4		38.6
P ₂ fat thickness (mm)		16.1		13.2
P ₂ (50-90 kg)		4.6		1.8
Marbling (%) LD		1.2		1.1
SM		2.0		2.1
Fat consistency (N x 100)		13.5		12.0
Carcass muscle (%)		52.7		58.3
Carcass fat (%)		30.9		24.0
(Hertzman <i>et al.</i> , 1989)				
Native pST treatment ($\mu\text{g}/\text{kg}$ live weight/day)	0	60	120	200
Carcass protein (%)	14.7	17.0	18.1	18.8
Carcass lipid (%)	32.1	21.6	17.2	13.8
(Beermann <i>et al.</i> , 1988)				

LD= *longissimus dorsi*; SM= *semimembranosus*

General considerations

It is evident that, potentially, the administration of pGH can result in the production of lean meat without compromising table meat quality. However, high doses of pGH could result in very lean carcasses which may be unattractive to butchers. There have been no studies done on the processing attributes of meat from pGH treated pigs. There seems little reason to suspect that manufacturing properties, e.g. water holding capacity, curing ability etc., would be adversely affected provided there was adequate fat deposition for the target market. Thus it appears that pGH treatment

does not adversely influence pre- and post-slaughter factors known to affect meat quality of pigs (Tarrant, 1989).

Table 2. Objective meat quality attributes of animals treated with pGH

Native pST treatment (mg/day)	0	3.0	6.0	9.0	
Shear force (kg) LD	3.2	3.4	3.6	3.8	
(Bechtel <i>et al.</i> , 1988)					
Recombinant pGH treatment (μ /kg live weight/day)	0			90	
¹ pH ₂₄ LD	5.6			5.6	
SM	5.6			5.6	
ST	5.7			5.7	
Peak shear force (kg) LD	5.5			5.0	
SM	6.2			6.5	
ST	3.8			3.6	
Instron compression (kg) LD	1.4			1.4	
SM	1.7			1.8	
Colour (L ²) LD	54			54	
SM	54			53	
Colour (a ³) LD	5			5	
SM	7			7	
Colour (b ⁴) LD	4			5	
SM	7			6	
Cooking loss (%)LD	33			33	
SM	30			29	
ST	27			27	
(Hertzman <i>et al.</i> , 1989)					
Native pST treatment (μ g/kg live weight/day)	0	60	120	200	
pH ₂₄ LD	5.3	5.4	5.5	5.5	
Shear force (kg) LD	3.2	3.6	3.0	3.8	
Colour (Gardner Rd) LD	26	24	24	23	
(Gardner a)	13	12	12	11	
Cooking loss LD	20	23	22	21	
(Beermann <i>et al.</i> , 1988)					
Recombinant pST treatment (μ /kg live weight/day)	Control	22 Kd	Form	21 Kd	Form
	0	60	90	60	90
Shear force (kg) LD	5.2	5.5	5.3	4.7	5.2
Cooking loss (%)LD	26	26	25	25	26
(Beermann <i>et al.</i> , 1988)					

¹pH at 24 h; ²lightness/darkness; ³yellowness/greenness; ⁴redness/greenness; LD= *longissimus dorsi*; SM= *semimembranosus*; ST= *semitendinosus*

Administration of growth hormone has produced greater responses in lean meat production in pigs than in other domestic species (Thornton and Tume, 1988). It has also produced large (+40%) responses in milk production from high producing dairy cows (Baumann *et al.*, 1985). In the dairy cow, these responses have been interpreted as an extension of the natural regulating mechanism(s) as high producing cows have high levels of growth hormone (see Thornton, 1987). It remains to be established whether or not this response (lean meat production) of pigs to growth hormone administration can also be regarded as an extension of natural regulatory processes in

response to higher levels of circulating pGH and greater binding site numbers or affinity at the cellular level. There is preliminary evidence that muscle fibre type is not affected (Beermann, personal communication) and that the crosslink concentration in intramuscular collagen is unaffected (Horgan, Kuypers and Kurth, personal communication) by pGH treatment. The fact that the EEC ban on meat from livestock treated with growth promotants excludes growth hormone, gives political recognition to the concept that treatment with growth hormone can be regarded as an extension of a natural phenomenon.

Table 3. Sensory meat quality attributes of *longissimus dorsi* muscle from pGH treated animals

Native pST treatment (mg/day)	0	3.0	6.0	9.0	
Tenderness ¹	9.3	8.6	8.7	9.6	
Off-flavour ¹	14.0	13.7	13.8	14.2	
Colour ²	2.9	2.6	2.5	2.6	
(Bechtel <i>et al.</i> , 1988)					
Recombinant pGH treatment (μ g/kg live weight/day)	0	90			
Tenderness ³		5.4		5.2	
Meat flavour ³		5.0		5.1	
Off-flavour ³		2.0		1.6	
Juiciness ³		5.4		4.7	
Acceptability ³		5.3		5.4	
(Hertzman <i>et al.</i> , 1989)					
Recombinant pST treatment (μ g/kg live weight/day)	Controls	22 Kd	Form 90	21 Kd	Form 90
	0	60	90	60	90
Tenderness ⁴	4.8	4.5	5.2	5.1	4.6
Flavour ⁴	5.3	4.7	5.3	5.0	5.1
Aroma ⁴	5.1	5.0	5.0	5.0	5.0
Juiciness ⁴	4.6	4.2	4.9	4.8	4.5
(Beermann <i>et al.</i> , 1988)					

¹Scale 0-15; ²scale 1-5; ³scale 0-9; ⁴scale not given

Repartitioning agents

The term repartitioning agent has been applied to a spectrum of compounds with β -adrenergic agonist activity which have been shown to promote muscle deposition and reduce fat deposition in domestic livestock and laboratory animals. The effects of β -adrenergic agonist (BAA) on animal growth and carcass quality have been reviewed (Hanrahan, 1987). In general, these compounds have more pronounced effects in the body composition and meat quality of ruminants, particularly lamb, than on pigs (Thornton and Tume, 1988; Merkel, 1988). However between species comparisons are confounded by the different BAAs used, the levels and mode of treatment employed, and other experimental variables.

Fat deposition

There is abundant evidence that all the BAAs reduce subcutaneous fat deposition in pigs. The magnitude of these reductions is, in general, dose dependent (see Hanrahan, 1987) but there is no evidence that any of the BAAs reduced fat cover to a level (<8 mm; Wood *et al.*, 1988) which may affect carcass quality. As with pGH

treatment there would be little point in commercially applying BAAs at levels which result in undesirable carcass traits.

Marbling and intramuscular fat

Visual levels of marbling (Table 4) and chemically determined intramuscular fat contents (Table 5) of *longissimus dorsi* muscles are in general unaffected by BAA treatment.

Table 4. Effect of β -adrenergic agonists on marbling in *longissimus dorsi* muscle

Agonist	Level fed (ppm)	Observation	Source
Cimaterol	.25, .5, 1	ND	Jones <i>et al.</i> (1985)
	.25, .5, 1	Linear increase	Moser <i>et al.</i> (1986)
	.5, 1	ND	Thornton <i>et al.</i> (1987)
Clenbuterol	.05, .1, 1	Increase	Ricks <i>et al.</i> (1984)
	L-644, 969	NS trend toward decrease	Wallace <i>et al.</i> (1987)
Ractopamine	2.5, 5, 10, 20, 30	ND	Hancock <i>et al.</i> (1987)
	20	ND	Merkel <i>et al.</i> (1988)

ND= not different

Table 5. Effect of β -adrenergic agonists on intramuscular fat content of *longissimus dorsi* muscle

Agonist	Level fed (ppm)	Observation	Source
Cimaterol	.05, 1	ND	Thornton <i>et al.</i> (1989)
Clenbuterol	1	ND	van Weerden (1987)
Ractopamine	5, 10, 20	ND	McKeith <i>et al.</i> (1988)
	20	ND	Merkel <i>et al.</i> (1988)
Salbutamol	4	ND	Cole <i>et al.</i> (1987)
	4	ND	Wood <i>et al.</i> (1987)

ND= not different

Tenderness

Shear force values tended to be increased slightly by BAAs in some studies but not in others (see Table 6). Increases were marginal and are probably too small to be detected by taste panels. This finding contrasts with reports from studies on cattle and sheep which have shown significant increases in shear force values (see Merkel, 1988).

pH

Muscle pH values (over a variety of times post-slaughter) were unaffected by any of the BAAs or the levels at which they were fed, clenbuterol at 1 ppm (Bekaert *et al.*, 1987; van Weerden, 1987); cimaterol at 0.5 and 1 ppm (Thornton *et al.*, 1989); ractopamine at 5, 10 and 20 ppm (Merkel, 1988); salbutamol at 2, 4 and 8 ppm (Cole *et al.*, 1987). It appears that the administration of BAAs does not predispose pig meat to high ultimate pH values. Again, these responses in pig muscle contrast to the often reported increased ultimate pH of muscle from sheep and cattle treated with BAAs (see Merkel, 1988). However, the ultimate pH of *longissimus dorsi* muscle from lambs treated with cimaterol (Allen *et al.*, 1985) and clenbuterol (Thornton *et al.*, 1988), but slaughtered immediately, was not different from that of controls.

Table 6. Effect of β -adrenergic agonists on shear force

Agonist	Level fed (ppm)	Muscle	Response	Source
Cimaterol	.25, .5, 1	LD	>	Jones <i>et al.</i> (1985)
		LD	>	Thornton <i>et al.</i> (1988)
	.5, 1	SM	ND	
		ST	ND	
Clenbuterol	.7, 1.4	LD	ND	Merkel (1988)
	1	LD	ND	van Weerden (1987)
Ractopamine	5, 10, 20	LD	ND	Merkel (1988)
		Ham	ND	
	20	LD	ND	Merkel <i>et al.</i> (1988)

LD= *longissimus dorsi*; SM= *semimembranosus*; ST= *semitendinosus*; ND= not different

Colour

Several studies have found no differences in colour of the *longissimus dorsi* muscle when pigs have been fed BAAs (see Table 7).

Table 7. Effect of β -adrenergic agonists on *longissimus dorsi* muscle colour

Agonist	Level fed (ppm)	Observation	Source
Cimaterol	1,	ND	Bekaert <i>et al.</i> (1987)
	.25, .5, 1	ND	Jones <i>et al.</i> (1985)
	25, .5, 1	ND	Moser <i>et al.</i> (1986)
	.05, 1	ND	Thornton <i>et al.</i> (1989)
Clenbuterol	.05, .1, 1	ND	Ricks <i>et al.</i> (1984)
L-644, 969	.25, 1, 4	ND	Wallace <i>et al.</i> (1987)
Ractopamine	2.5, 5, 10, 20, 30	ND	Hancock <i>et al.</i> (1987)
Salbutamol	2, 4, 8	ND	Cole <i>et al.</i> (1987)
	4	ND	Wood <i>et al.</i> (1987)

ND= not different

Taste panel score

There appears to be only one published study of organoleptic attributes of pork from pigs fed BAAs, 0, 5, 10 and 20 ppm ractopamine (Merkel, 1988, including data of McKeith *et al.*, 1988). In this study, both loin chops and cured hams were assessed and no significant differences, between treated and control animals, were found for any palatability trait examined (juiciness, tenderness, flavour and "off" flavour and overall acceptability).

Processing qualities

Attributes of meat described as muscle firmness, muscle wateriness, water holding capacity and drip loss have not been affected by feeding BAAs to pigs (see Merkel, 1988 including data of McKeith *et al.*, 1988).

General considerations

The generalisation which can be drawn from this information is that BAAs have little or no effect on meat quality of pigs. This contrasts with the information from ruminant studies which indicate detrimental effects of BAAs on meat quality of both lambs and cattle. There is no clear explanation of this species difference.

Large (1.5 mg) doses of clenbuterol resulted in increased heart rate and body temperature and decreased blood pressure in lambs (Herbert *et al.*, 1985; Brockway

et al., 1987) but these effects persisted for less than 24 h. Rather than anticipate similar such effects, we suggest that similar experiments should be done with pigs.

The physiological/biochemical effects of BAAs are different between ruminants and pigs, but these specific differences appear greater in *in vitro* studies on tissues than in *in vivo* experiments on animals (see Thornton and Tume, 1988). *In vivo* studies have shown that BAAs also affect regulatory hormone levels of sheep, e.g. decreased insulin and increased growth hormone (see Thornton *et al.*, 1987). Although these experiments have not been done in pigs, there seems little reason to suspect that such metabolic responses and differences between species are responsible for the observed differences in meat quality between ruminants and pigs treated with BAAs.

In lambs fed cimaterol the incidence of type I muscle fibres was less and the frequency distributions of both type I fibres and type II fibres were markedly different from those of controls (Beermann *et al.*, 1987). Similar responses in type II fibres in the *longissimus dorsi* muscles of pigs treated with salbutamol have been demonstrated by Oksbjerg, Blackshaw and Fernandez (personal communication). Apparently such BAA induced changes in muscle fibre type incidence and distribution have little effect on meat quality.

Treating lambs with BAAs has resulted in decreased activity of the calcium dependent proteinase enzymes (CDP-I and cathepsin B) and increased activity of CDP-II and calpastatin of muscles (see Merkel, 1988). These changes in enzyme activity may influence meat tenderness. Interestingly Merkel *et al.* (1988) found no differences in total CPD activity or cathepsin B, H or L activities between ractopamine fed pigs and controls.

Conclusions

Both pGH and some of the BAA have the ability to promote rapid growth of lean without detrimental effects on meat quality of pigs.

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MICROBIOLOGICAL CONTAMINATION OF PIG CARCASSES

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Introduction

The keeping quality of meat is largely determined by the numbers and types of micro-organisms present on carcass surfaces after slaughter, and the temperature of storage (Ingram, 1972; Sheridan, 1982). Pig carcasses usually carry considerably more bacteria on their surfaces than lamb or beef (Johanson *et al.*, 1983; Roberts, 1980), because the numbers of bacteria present on pig carcass surfaces are influenced to some degree by the slaughter and dressing processes (Roberts, 1980; Gerats *et al.*, 1981).

Materials and methods

A survey of bacterial contamination of pig carcasses on the slaughterline was conducted at eight abattoirs which had different scalding and dehairing systems and dressing procedures. Some characteristics of these abattoirs are given in Table 8.

With the exceptions noted below, each abattoir was visited on five occasions, and 10 pigs were sampled at four separate locations on the slaughter chain. The first pig to be sampled was selected at random and then every fifth pig presented on the slaughter chain was selected for sampling. The same batch of pigs were followed through the scalding and dehairing process. Pigs sampled at site B were permanently identified so that the same pigs were serially sampled at sites B, C and D.

The samples collected from each pig consisted of swabs taken from two 20 cm² areas which were defined by sterile templates using the wet-dry swab technique of Kitchell *et al.* (1973). The two areas selected for swabbing were the ham and mid-back regions of each carcass. These areas were selected on the basis of previous work by Morgan *et al.* (1987).

Pigs were swabbed before they were scalded (site A), after being scalded and dehaired (site B), after evisceration (site C), and as they were placed in chiller storage (site D). Because of the physical restraints on sampling in the slaughter chains the sampling points differed slightly at some abattoirs.

Because of the difficulty in gaining access to stunned pigs, prior to their entering the scalding tank, pre-scald swabs were collected from pigs at abattoir III on a single visit. Thus the bacterial counts from this site at this abattoir are not distributed over five visits like the swabs from sites B, C and D. Because of travel constraints, pigs at abattoir VIII were swabbed over three visits with 17 pigs being sampled at each visit. In addition to sampling along the slaughterline, specific investigations were conducted at abattoir I into the effect of time on the contamination of carcasses and scald tank water. Approximately 45 min after the commencement of the day's slaughter run five carcasses were swabbed, at 15 min intervals, as they were placed in the chiller. This procedure, with a break for morning "smoko", was continued until 4 h had elapsed from the commencement of the day's slaughter. At the same visit, scald tank water samples were collected at approximately 45, 165 and 240 min usage. The second sample was collected after the scald tank temperature had stabilized during the morning "smoko" break when no pigs entered the tank. Throughput during these visits was approximately 120 pigs/h. This sampling procedure was conducted on five occasions.

Table 8. Characteristics of abattoirs surveyed

	Abattoir							
	I	II	III	IV	V	VI	VII	VIII
Pre-sticking wash	-	+	-	-	+	+	+	+
Sticking site	Pen	Pen	Pen	Pen	Rail	Rail	Rail	Rail
Pre-scald wash	-	-	-	-	-	+	-	-
Scald ¹	T	R	T	T	T	Tun	T	Tun
Dehairing systems ²	1	2	2	1	1	Tun	1	Tun
Singeing ³	M	M	M	M	M	T	M	T
Wash stations ⁴	H	A	H	H	A	A	A	A
Final wash ⁴	H	H	H	A	A	A	A	A
Operatives ⁵	B	L	L	B	L	L	L	L
Rail stations ⁶	L	H	M	L	H	M	M	M
Throughput/day	600	1000		250	800	800	500	1000
Chain ⁷	M	A	A	M	A	A	A	A

¹scald: T - tank scald; R - on-rail scalding; Tun - tunnel scald

²dehairing: 1 - single beater/dehairer; 2 - twin in-line beater/dehairer

³singeing: M - hand-held gas torch; T - flame tunnel

⁴wash: H - hand hosing; A - wash tunnel stations

⁵operatives: B - butcher; L - labourer

⁶rail stations: L - low; M - medium; H - high

⁷chain: M - manual; A - mechanical

Bacteriological procedures

Carcass swabs were stomached in a Colworth Stomacher (Seward, England) with the 20 ml of peptone water used to transport the swabs to the laboratory. The homogenate was then used to conduct total viable counts and *Escherichia coli* counts for all carcasses. In addition, at some abattoirs *Campylobacter* and *Clostridium perfringens* counts were also performed on some or all the site swabs. The techniques for bacterial isolation have been described by Morgan *et al.* (1987).

Data analysis

All counts were converted to logarithmic values to the base 10 for calculation of means and standard errors for counts by site and by visit. To overcome the problem of taking the logarithm of zero when analyzing *E. coli* counts, one was added to all counts before conversion.

Results

Abattoir procedures

All abattoirs electrically stunned their pigs before bleeding but the handling procedures differed between abattoirs. At some abattoirs pigs were loaded into a squeeze conveyor in which they were stunned before being shackled and bled. Others herded a group of pigs into a pen where once they were stunned they fell to the floor of the pen from which they were hoisted onto a rail for bleeding.

Three methods for scalding and dehairing pig carcasses were encountered during this study. The first method, tank scalding, involved the immersion of the whole carcass in a trough of hot water, kept at 58-65°C, for approximately 3-5 min. Carcasses then passed to the dehairing machine where they were rotated with other pigs while in a horizontal position. The rubbing of the carcasses together helped to remove the softened hairs and other skin debris. In addition, the carcasses are scraped by rubber tongues which flail the carcass surfaces dislodging hair and other skin debris, including the outer layer of the epidermis. Water is sprayed onto the carcasses whilst they are in the dehairing machine to wash away the hair and other debris. The water may be

at ambient temperature, or heated, and is often recirculated in the dehairing machine. Following this process, carcasses are usually scraped by hand to remove residual hair and other debris missed by the dehairing machine process. Finally carcasses were singed by a hand-held gas flame to remove any hair remaining. Occasionally this was followed by a second hand scraping to remove charred hair and skin but, in all cases carcasses were washed by some means before progressing further along the slaughterline.

The second method of scalding, which was in effect at abattoir II, involved carcasses being scalded on the hanging rail with wet steam as they passed through a chamber. Carcasses spent approximately 15 min passing through this chamber before being placed in the dehairing machine. At this abattoir carcasses after initial dehairing were transferred to a second dehairing machine for a final clean-up before undergoing similar processes to those described for dehairing after tank scalding.

In the last method, found at abattoirs VI and VIII, pigs were scalded and dehaired in a long tunnel. The pigs were conveyed along the small diameter tunnel in a horizontal position while being rotated. They were bathed in hot water at 60-65°C whilst in the tunnel. The rubbing of the carcasses together assisted in the removal of hair and other debris from the carcass. The speed of the carcasses passing through the tunnel and the water temperature is adjusted to produce adequately dehaired carcasses. These systems also recirculate some of the scald water to reduce energy losses. Separate dehairing machines were not used at these two abattoirs but hand scraping was performed to give carcasses a final clean-up. At abattoir VI pigs then passed through a flame tunnel emerging with a slightly charred appearance. The flaming apparatus at abattoir VIII was less fierce producing minimal charring.

Total viable counts

Means and standard errors for total viable counts (TVC) at each site, on each visit, at each abattoir are shown in Table 9.

Table 9. Total viable count means and standard errors for pig carcasses sampled at four sites on the slaughter chain

Abattoir	Pre-scald		Post-dehair		Post-evisceration		Chiller	
	mean	SE	mean	SE	mean	SE	mean	SE
I	5.484	0.049	4.224	0.043	3.519	0.050	3.512	0.061
II	4.744	0.043	3.354	0.045	3.151	0.035	3.229	0.045
III	4.844	0.076	3.283	0.058	3.133	0.036	3.086	0.034
IV	5.466	0.035	3.688	0.073	3.818	0.066	3.449	0.064
V	5.010	0.099	3.449	0.089	3.271	0.095	3.114	0.072
VI	4.513	0.046	2.863	0.039	2.879	0.046	2.691	0.062
VII	4.458	0.067	3.045	0.060	3.206	0.056	2.897	0.055
VIII	5.113	0.055	2.331	0.016	2.664	0.110	2.395	0.047

In general, the differences in the mean visit TVC for a particular site at individual abattoirs were small. This was mainly due to the variability of the TVC on individual pigs which resulted in wide confidence limits to the estimated mean TVC. When all TVCs for a particular site were used to calculate a mean TVC for a site at an abattoir there were some patterns evident. In general, the abattoirs which used tank scalding (I, III, IV, V, VII) tended to have higher final TVC at the three sites sampled after the scalding and dehairing process than those which used on-rail or tunnel scalding.

For tank-scalded pigs, the TVC on carcasses at the end of the slaughter process appeared to be related to the average TVC on pigs at the commencement of scalding and dehairing. It was not possible to establish a statistical relationship between site A counts and site D counts since different groups of pigs were involved at each site.

However, increasing mean TVC values at site A were paralleled by increasing mean TVC values at site D. This relationship did not appear to hold for pigs scalded by methods other than tank scalding.

The scalding and dehairing process reduced the mean TVC significantly, so that after this process they were at approximately the levels found on carcasses at the end of the slaughter chain, as is shown in Table 9. The change in TVC from site A to site B was in the order of 1.5 logs reduction. However, the reduction in TVC from site B to site D was only of the order of 0.2 log.

However, the TVC on individual pigs was not always reduced by the washing steps between the end of scalding and dehairing and the final placement of carcasses into the chiller. In general the carcasses with the lower counts at site B tended to have increased counts at site D, while those with higher site B counts tended to show a decrease in bacterial counts at site D. However, the major trend was for a decrease in TVC on individual pigs from site B to site D with the average decrease being approximately 15% reduction in the TVC level recorded at site B.

The reduction in TVC between site C, which was immediately before the final wash, and site D was very small, with the average decrease being approximately 4% reduction in the TVC recorded before the final wash.

Escherichia coli counts

Means and standard errors for *E. coli* counts at each site, on each visit, at each abattoir are shown in Table 10.

Table 10. *Escherichia coli* count means and standard errors for pig carcasses sampled at four sites on the slaughter chain

Abattoir	Pre-scald		Post-dehair		Post-evisceration		Chiller	
	mean	SE	mean	SE	mean	SE	mean	SE
I	2.906	0.067	1.879	0.064	1.539	0.088	1.396	0.084
II	2.841	0.065	0.924	0.081	0.582	0.078	0.866	0.084
III	2.046	0.128	1.152	0.071	0.767	0.066	0.636	0.079
IV	3.230	0.066	1.083	0.088	1.029	0.087	0.589	0.061
V	2.179	0.122	0.991	0.082	0.852	0.084	0.644	0.077
VI	2.910	0.067	0.213	0.047	0.269	0.060	0.258	0.055
VII	2.774	0.062	0.491	0.061	0.536	0.068	0.599	0.074
VIII	2.803	0.067	0.092	0.026	0.236	0.055	0.142	0.039

Abattoirs VI and VIII, which used a tunnel scalding and dehairing process, had significantly lower mean *E. coli* counts on carcasses after scalding and dehairing than other abattoirs.

The *E. coli* counts on carcasses at the end of the slaughterline were low, these being less than 10 organisms/cm² at all abattoirs except at abattoir I. At this abattoir carcasses at site D had a mean *E. coli* count of 25 organisms/cm². Mean *E. coli* counts at other points on the slaughterline were significantly higher at abattoir A also, although pigs entering the scalding tank at this works had no higher *E. coli* counts than pigs at other abattoirs.

There appeared to be no relationship between *E. coli* counts on pigs entering the scalding process and the counts obtained at the end of the slaughterline.

Campylobacter

Swab samples were examined for the presence of *Campylobacter* organisms at site D at abattoirs I, II, III, VI, VII, and VIII but the numbers of organisms recovered from the carcasses was very low, in the order of 1 organism/cm². The mean percentage of

carcasses carrying *Campylobacter*, at each abattoir, ranged from 0-42.5% with the overall average for all abattoirs being 14%.

At abattoirs III, VI, VII and VIII most carcasses, entering the scalding process, were contaminated with *Campylobacter* with 63% of 157 carcasses tested showing evidence of contamination with this organism. At individual abattoirs, the contamination rate ranged from 32.5-76%. The *Campylobacter* counts on ranged from 0->300 organisms/cm² with an average count of 7 organisms/cm².

Clostridium perfringens

At site A, 95% of the 176 carcasses examined at abattoirs I, II, III, VI, VII, and VIII, were contaminated with *Clostridium perfringens*. Mean *Clostridium* counts for individual visits ranged from 5-7050 organisms/cm². Scalding and dehairing resulted in a significant fall in the numbers of *Clostridium perfringens* on carcasses and also in a fall in numbers of carcasses contaminated to 26% of carcasses sampled at site B. The *Clostridium perfringens* counts on carcasses at site B ranged from 0-20 organisms/cm² with most carcasses having counts of 1 organism/cm². The rate of contamination of carcasses with *Clostridium perfringens* fell throughout the slaughter and dressing process with only 9.8% of carcasses being contaminated at site D.

Tank scalding

Mean total viable counts and their standard errors for each time of sampling at each visit are shown in Table 11. An inspection of these results shows that there was no significant increase or decrease in bacterial contamination on carcasses over a 3 h period. There was also no significant change in *E. coli* counts on carcasses over the 3 h sampling period.

Table 11. Effect of time on pig carcass bacterial counts

Time of sampling (min)	TVC ₂₁		<i>E. coli</i>	
	mean	SE	mean	SE
0 ¹	3.597	0.092	1.376	0.064
15	3.599	0.115	1.209	0.061
30	3.472	0.105	1.050	0.054
45	3.497	0.111	1.173	0.048
60	3.455	0.097	1.060	0.041
75	3.474	0.097	1.031	0.043
90	3.522	0.097	1.275	0.051
105	3.469	0.109	1.279	0.062
145	3.535	0.082	1.298	0.069
160	3.349	0.076	1.136	0.064
175	3.440	0.085	1.052	0.074

¹Time 0= 0745 h

The results of TVC at 37°C shows an increasing level of contamination of the scald tank water over the sampling period (Table 12). Counts showed increases in the order of 10-100 times the level measured approximately 45 min after the commencement of the day's kill. Results of TVC at 21°C show a different pattern with the second sample, taken at "smoko", being lower than the initial sample. There was not a significant increase in numbers of bacteria, able to grow at 21°C, between the first sample and the last sample some 3 h later.

Table 12. Microbiological and other characteristics of scald tank water at abattoir I

Visit	Time since start of day's kill	pH	Temperature	<i>E. coli</i> ¹	TVC ₃₇ ¹	TVC ₂₁ ¹
1	0:50	7.69	58°C	0	31,500	5,350
	2:40	7.58		0	3,550,000	1,220
	3:50	8.00		0	4,600,000	2,600
2	0:45	8.14	58°C	14	18,350	1,750
	2:40	8.11		1	28,300	1,130
	3:50	8.18		0	183,000	1,890
3	0:45	7.49	57°C	0	28,000	5,050
	2:45	7.70		0	>30,000	n.s.
	4:00	7:40		0	>30,000	8,100
4	0:45	n.d.	n.d.	0	19,150	730
	2:45	n.d.		0	4,200,000	760
	3:50	n.d.		0	10,150,000	3,700
5	0:45	n.d.	n.d.	0	23,200	2,230
	2:45	n.d.		0	38,500	810
	3:50	n.d.		3	83,000	2,750

¹Number of organisms/ml of scald water; n.d.= not done; n.s.= no sample. Note: pH measured at laboratory because fresh sample too hot.

Discussion

The primary hygienic concern in slaughtering of any animal for human consumption is the prevention of microbial contamination of the carcass meat from the exterior skin surfaces and alimentary tract. Pig slaughtering presents different problems to those experienced in sheep and cattle because of the treatment of the exterior skin surfaces. One reason for this is the process for removal of hairs from the hide, which remains on pig carcasses whereas with sheep and cattle the hide is totally removed.

In the scalding process faeces escapes from the anus, due to muscle relaxation, and blood oozes from the sticking wound. These excretions together with saliva all contribute to the biological and bacterial load of the scalding water. The time and temperature to which carcasses are exposed to the scald water are insufficient to destroy all bacteria on the carcass surface. In addition, bacteria from the carcass surfaces survive readily in scald tank water as shown by the results obtained in our second experiment conducted at abattoir I. These results showed that bacterial numbers increased during the day. The greatest concentration of bacteria were those which grew at 37°C, rather than those growing at lower temperatures. However, both groups of bacteria counted showed an increase in concentration with time, and increasing contamination of the water. The scald tank water could therefore be a source of bacteria for contaminating carcass surfaces. However, we found that increasing bacterial concentrations in the scald tank water did not appear to be reflected in increasing levels of bacterial contamination on carcasses at the end of the slaughterline. Snijders (1975) found that bacterial levels in scald tank water rose in the first 60 min of production and then remained almost constant for the rest of production. He found that TVC, at 30°C, reached average levels of log 3.5-4.5 bacteria/ml of tank water. As a result of these studies, Snijders (1975) concluded that tank scald water needed to be of high pH and at a temperature of greater than 60°C to achieve low bacterial counts in the water.

Bacterial contamination of scald tank water may be more important as a source of contamination for deep muscle tissues rather than for the external carcass surface. Jones *et al.* (1979; 1984) have shown that there is uptake of colloidal particles from

scald tank water through the stick wound. Residual heart action is sufficient to circulate these particles throughout the circulatory system and could result in deep muscle contamination with bacteria.

Dehairing machines also contribute to the bacterial load on carcasses by recontaminating them with faeces and blood which continues to leak from carcasses as they are rubbed and buffeted together. These excretions are further spread by the scrapers or flails in the dehairing machine. A spray or mist of contaminated water droplets ejected from the machine as the carcasses are tumbled may also contribute to the spread of contamination between carcasses.

The singeing process can also influence the bacterial flora on pig carcasses as shown by work done by Rasch *et al.* (1978). They demonstrated a 1.3 log difference in bacterial counts on pig carcasses singed in a flame tunnel/oven when compared to those singed manually. The only two abattoirs in our survey which used flame tunnel/oven singeing, abattoirs VI and VIII, also had tunnel scalding and dehairing machines and produced carcasses with the lowest counts, after dehairing, of all abattoirs surveyed. It is likely that a combination of the singeing technique and the scalding and dehairing process was responsible for this effect since abattoir VI singed their pigs more severely than abattoir VIII but did not produce carcasses with lower counts. At abattoir VI pigs emerged from the singeing process with mahogany coloured skin due to the heat whereas at abattoir VIII carcasses were not so affected.

We found that mean total bacterial counts fell by at least 1.2 log counts, with decreases ranging from 1.2 log to 2.8 log. Two abattoirs with tunnel scalding systems produced the least contaminated carcasses after scalding and dehairing, suggesting that the more recent developments in scalding and dehairing are more hygienic. *E. coli* counts also showed a marked decline after scalding and dehairing with the two tunnel scald abattoirs also performing best at removal of bacteria from carcasses. Contamination of carcasses with *Clostridia* also showed a marked decline after scalding and dehairing with a 50-70% decline in the number of carcasses detected with these organisms. A similar pattern was observed by Baltzer and Wilson (1965) at both Danish and Irish bacon factories. At all abattoirs there was an increase in the contamination of scald tank water with *Clostridia* with increasing numbers of pigs processed during a production run.

Troeger and Woltersdorf (1986) have demonstrated that meat from skinned pig carcasses have lower bacterial counts than that from scalded and dehaired carcasses. They suggest that scalding and dehairing may also adversely affect the quality of pigmeat as well as being responsible for the high bacterial content of pigmeat.

Our results suggest that the scalding and dehairing process, while contributing to a marked decline in bacterial numbers on carcasses, is a major impediment to the hygienic production of pigmeat. Some steps which can be taken to improve hygienic slaughter include thorough washing of pigs before they enter the slaughterhouse and attention to lairage pen hygiene and design to reduce the bacterial load on the skins of pigs coming onto the slaughter floor. The sticking/bleeding process may also contribute to the spread of contamination if this operation is carried out in pens as these areas rapidly become soiled with blood and faeces. If there is no washing step after stunning and sticking then bacteria will be spread from carcass to carcass in the stunning area. Abattoirs where stunning was conducted in a conveyor system appeared to produce visually cleaner pigs prior to scalding. Recirculation of water in dehairing machines also appeared to have a negative effect on carcass contamination with bacteria.

Snidjers *et al.* (1977) examined the use of chlorinated water in dehairing machines and found that it could give a further reduction of 0.5-1.0 log in total bacterial counts. This effect was dependent on the chlorine concentration. They also found that when 75 ppm chlorinated water was used in the dehairing process that there was a fall in the

TVC during chilling, which was in contrast to the rise in TVC on control carcasses.

The average reduction in mean TVC from bleeding to final wash, taken over all abattoirs, was 1.9 log. Except for abattoir VIII which showed a reduction of 2.7 log all abattoirs showed average reductions ranging from 1.5 log-2.0 log, irrespective of the average initial carcass TVC. This suggests that processing has only a finite effect on carcass contamination. The reduction in bacterial levels compare with those obtained by Snijders and Gerats (1976) who found an average reduction in TVC from bleeding to final wash of 2.2 log.

Snijders and Gerats (1976) also found small reductions in TVC after the scalding and dehairing operation similar to that observed in our survey. However, in our survey not all carcasses showed reductions in TVC suggesting that there is still potential for slaughterline operations to recontaminate carcasses. One of the obvious sites where this could occur is the evisceration process. If the gastrointestinal tract is punctured during removal the carcass, and succeeding ones, can have their bacterial levels augmented by organisms transferred from contaminated equipment or operator's hands.

We found that the final washing of carcasses produced only minor decreases in the bacterial levels on carcasses. This may be the result of inadequate washing techniques. To be effective carcass washing needs an adequate water pressure and effective direction otherwise bacteria may only be moved from one site to another on the carcass (Nortje *et al.*, 1979). Hot water, at a temperature of at least 80°C, is also needed to reduce bacterial numbers on carcasses (Kelly *et al.*, 1981). It has been suggested by Nortje *et al.* (1979) that low degree singeing of carcasses prior to chilling might be used as an alternative to washing as a method of reducing bacterial numbers on carcasses.

EXTENDING THE STORAGE LIFE OF CHILLED PORK

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Introduction

There are three major factors which control the storage life of fresh meat stored at refrigeration temperatures. These are microbial growth, colour stability and chemical changes. Depending upon the conditions of storage either one or a combination of these factors will limit the storage life of meat. When fresh carcass meat is stored in air, storage life is usually terminated as a result of excessive microbial growth. However under alternative conditions of storage such as occur in modified gas atmospheres or vacuum packs, chemical changes in the muscle and/or colour instability can also be limiting factors.

Apart from modifying the atmosphere surrounding the meat by either vacuum-packaging or modified atmosphere packaging, irradiation may also be used to extend storage life. These procedures may be combined with decontamination of the meat to retard bacterial spoilage.

This paper reviews methods of extending the storage life of chilled pork and presents the results of research carried out at the CSIRO Division of Food Processing, Meat Research Laboratory. These studies were aimed at identifying the factors controlling the storage life of pork and investigating mechanisms by which this storage life could be extended.

Spoilage

When meat is stored in air under conditions of high relative humidity, it spoils rapidly due to the growth of *Pseudomonas* bacteria. The primary aim of any attempt to extend the storage life of fresh meat must be to prevent or retard the growth of this organism.

This is one of the main reasons why carcasses are chilled under controlled conditions which reduce the available water at the carcass surface. The results in Table 13 show that varying air speed in the chiller can bring about a significant reduction in the growth rate of *Pseudomonas*. The effectiveness of this technique is obviously determined by what is considered to be an economically acceptable weight loss during this procedure.

Table 13. The effect of chiller air speed on the growth of *Pseudomonas* on beef carcasses stored at 0°C (Scott and Vickery, 1939)

Average air velocity (m/sec)	Increase in bacterial numbers over a 48 h period
0.15	4 fold
0.45	2 fold
0.75	nil

However when meat is stored packaged, controlled drying of the meat surface does not occur because one of the prime criteria of any material used for packaging

fresh meat is that it must be impermeable to moisture. *Pseudomonas* is a strict aerobe and as such requires oxygen for growth. One commonly used and very successful technology for extending the storage life of fresh meat, is vacuum-packaging. This technique involves placing the meat in a bag made from a plastic film of low oxygen permeability, removing the air by the process of evacuation and sealing the bag under vacuum. This drastically alters the gaseous environment surrounding the meat. It becomes devoid of oxygen and there is an increase in the carbon dioxide level due to consumption of residual oxygen by respiration of the meat. To maximise storage life the permeability of the film should be as low as possible and films with oxygen permeabilities $<1 \text{ ml/m}^2/24 \text{ h/atm}$ (measured at 25°C and 98% RH) are now available.

Vacuum-packaged beef is a major export commodity for Australia. Trade in this product is possible because of a storage life under commercial conditions of ≈ 10 weeks at $0-1^\circ\text{C}$. This can be achieved provided plastic films with oxygen permeabilities less than $50 \text{ ml/m}^2/24 \text{ h/atm}$ are used. Under laboratory conditions storage lives of up to 15 weeks have been reported. (Newton and Rigg, 1979; Egan, 1983). To achieve maximum shelf life only meat of low pH (<6.0) must be packaged and the packaging material must be of low gas permeability. When spoilage does occur under these conditions, it is manifested by the development of flavours and/or odour commonly described as sour acid or cheesy. These are caused by the growth of lactic acid bacteria (Dainty *et al.*, 1979; Egan and Shay, 1982; Egan, 1983).

The environmental conditions existing in a vacuum pack, namely very low oxygen tension, high carbon dioxide concentration, low storage temperature and low pH, result in a bacterial flora consisting almost entirely of psychrotrophic lactic acid bacteria. If any of the aforementioned conditions are not adhered to, storage life will be reduced as a result of the growth of certain other types of bacteria. For example, if meat of high pH (>6.0) is packaged, spoilage is usually more rapid and may be characterized by changes in appearance, especially greening of the weep. Spoilage of this type is caused by the growth of Gram-negative bacteria especially *Alteromonas putrefaciens* and psychrotrophic *Enterobacteriaceae*, which are able to grow in the less selective environment (Nicol *et al.*, 1970; Bem *et al.*, 1976; Taylor and Shaw, 1977; Patterson and Gibbs, 1977; Gill and Newton, 1979; Newton and Gill, 1980; Erichsen *et al.*, 1981).

There is no significant export trade in vacuum-packaged pork, but limited amounts are used in the local hotel and restaurant trade. Processors have reported a shelf life of as little as two weeks under commercial conditions and this is considered inadequate even for local use.

Since there is a higher incidence of high pH meat with Australian pork than there is with beef, the packaging of high pH meat appears to be a major factor causing the inadequate shelf life. Additionally and in contrast to beef, multiple muscles are often packed together in the same bag resulting in an increased likelihood of packs containing high pH meat.

The microbiology of vacuum-packaged beef has been thoroughly investigated but there have been very few studies of vacuum-packaged pork. Hermansen (1980) reported that the spoilage of high pH pork was putrefactive and caused by *Alteromonas putrefaciens*.

In the course of our investigations we have examined the growth of the microbial flora on the lean, fat and skin surfaces of both high and low pH vacuum-packaged pork stored at 0°C and 5°C . Using the "laboratory analytical taste panel", the storage life of vacuum-packaged low pH pork has been estimated and found to be only about half of that of beef stored under similar conditions. We have also investigated the use of a number of methods to extend the storage life of high pH pork to that of normal pH pork. These have included treatment with organic acids just prior to vacuum-packaging, irradiation, storage under high levels of carbon dioxide and storage at -1°C (just above the freezing point of meat).

Microbiology and storage life of pork

When fresh meat is stored at temperatures of 0-5°C, the types of micro-organisms that grow, the nature of the spoilage that occurs and the storage life that results are all effected by the pH of the meat. The other important factor influencing microbial growth is the composition of the gaseous atmosphere in which the meat is stored. Table 14 shows the effect of oxygen availability and pH on the growth on lean fresh meat of the major types of spoilage bacteria at 0-5°C.

Table 14. Effect of oxygen and pH on the growth of the major types of meat spoilage bacteria at 0-5°C on meat

Organism	pH 5.5-5.7		pH 6.0 or higher	
	oxygen	no oxygen	oxygen	no oxygen
<i>Pseudomonas</i>	+	-	+	-
Enterobacteriaceae	+	- ¹	+	+
<i>Brochothrix thermosphacta</i>	+	- ¹	+	+
Lactic acid bacteria	+	+	+	+
<i>Aeromonas</i>	-	-	+	+
<i>Alteromonas putrefaciens</i>	-	-	+	+

¹Some growth may occur, especially at intermediate pH values (5.8-5.9) and at 5°C, but this is usually not sufficient to contribute significantly to spoilage.

The growth of the microbial flora on the lean and skin surfaces of vacuum-packaged pork of low pH (5.5-5.8) stored at 0°C is exemplified by the results shown in Figure 1. Lactic acid bacteria were the dominant component of the fat as well as the skin and lean surfaces. On the lean surfaces these organisms reached a maximum population typically in the range of $2.5 \times 10^7/\text{cm}^2$ after 4-5 weeks of storage and comprised more than 99% of the organisms present. Whilst *Brochothrix thermosphacta* reached a maximum population of only $\approx 10^4/\text{cm}^2$ on the lean (Figure 1b), it reached about $5 \times 10^5/\text{cm}^2$ on the fat (data not shown) and exceeded $10^6/\text{cm}^2$ on the skin (Figure 1a). The population of Gram-negative bacteria remained low on all three types of tissue ($\approx 10^4/\text{cm}^2$) but typically showed quite large fluctuations.

The development of the microbial flora on vacuum-packaged pork of high pH was then studied. Results demonstrating growth on lean surfaces are shown in Figure 2. The total number of viable organisms present on pork of pH 6.1-6.7 stored for 4-5 weeks at 0°C was typically somewhat higher than found for meat of low pH. Lactic acid bacteria were again the dominant component of the flora reaching approximately $10^8/\text{cm}^2$. *B. thermosphacta* and the Gram-negative bacteria were also present in higher numbers than on meat of low pH.

When high pH meat was stored at 5°C, not only did the bacteria grow more rapidly but the total count was noticeably higher than at 0°C (typically $2.5 \times 10^8/\text{cm}^2$). There was also significant increases in the population reached by the Gram-negative bacteria and *B. thermosphacta*, which grew to $10^8/\text{cm}^2$ and $10^6/\text{cm}^2$ respectively. A considerable proportion of the Gram-negative isolates were capable of producing hydrogen sulphide. Table 15 lists the maximum population of the various groups of bacteria found on the lean surfaces of vacuum-packaged pork.

In most experiments, spoilage of high pH vacuum-packaged pork was due to the development of undesirable colour defects. Greening of the weep often occurred, and this resulted in a general discolouration which was most noticeable on the fat surfaces. When green packs were opened a smell of hydrogen sulphide was usually noted. Greening of high pH pork usually occurred after 4-5 weeks when stored at 0°C and after 2-3 weeks at 5°C.

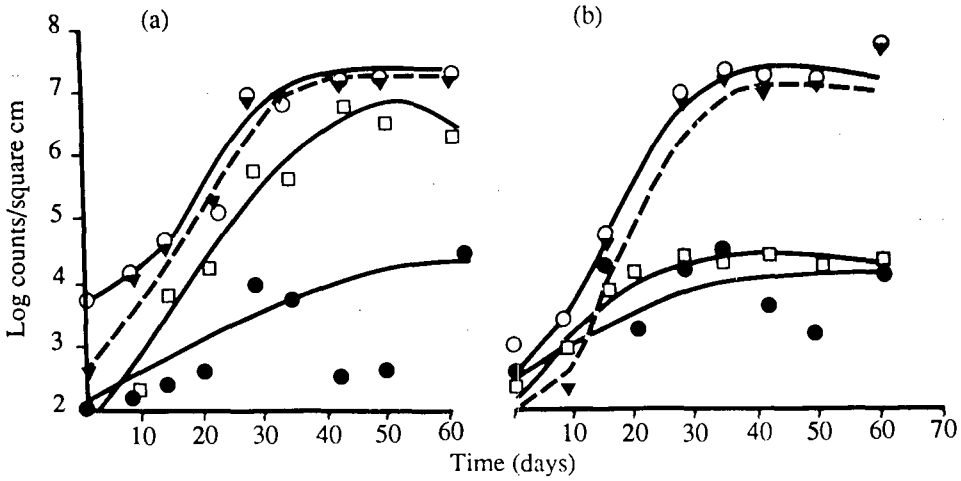


Figure 1. Growth of the bacterial flora on the skin (a) and lean (b) surfaces of low pH (5.5-5.8) vacuum-packaged pork stored at 0°C. Total counts (open circles); lactic acid bacteria (solid triangles); *Brochothrix thermosphacta* (open squares); Gram-negative bacteria (solid circles).

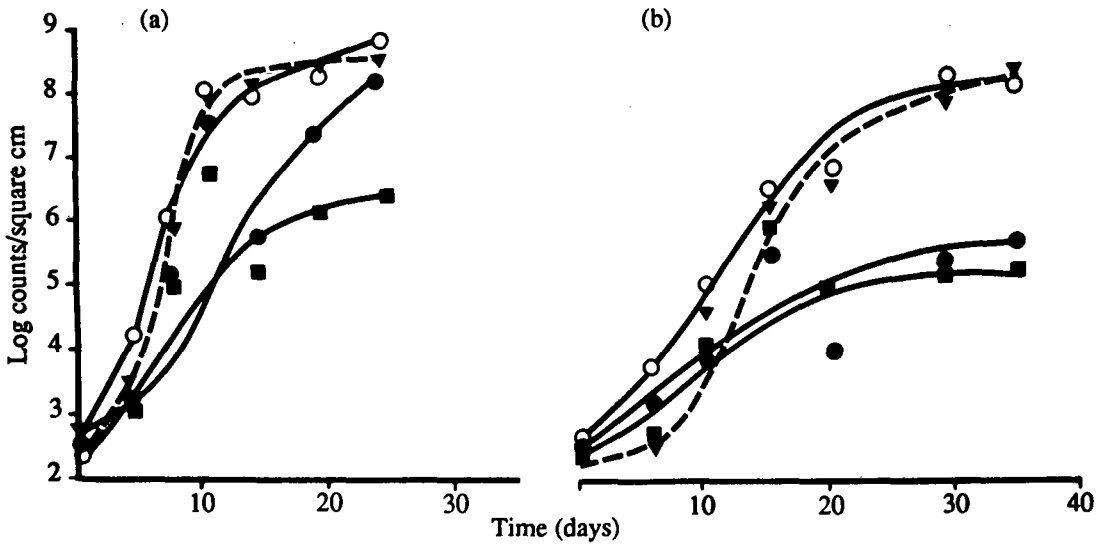


Figure 2. Growth of the bacterial flora on the lean surface of high pH (6.1-6.7) vacuum-packaged pork stored at 5°C (a) and 0°C (b). Total counts (open circles); lactic acid bacteria (solid triangles); *Brochothrix thermosphacta* (open squares); Gram-negative bacteria (solid circles).

Table 15. Highest numbers of bacteria on the lean surface of vacuum-packaged pork¹ stored for up to six weeks

pH	Temperature (°C)	Total viable count ¹	Lactic acid bacteria	<i>B. thermosphacta</i>	Gram-negative bacteria
5.4-5.8	0	7.9	7.9	4.2	4.8
	5	8.0	7.9	4.5	6.2
6.1-6.7	0	8.7	8.5	7.5	6.7
	5	8.6	7.9	7.2	8.0

¹Log₁₀ number/cm²

In the case of vacuum-packaged pork with a normal pH (5.4-5.8), flavour changes limited storage life. A trained analytical taste panel of 15 members was used to determine the shelf life at 0°C and the results of a typical experiment are shown in Figure 3. The pork samples gradually deteriorated during storage mainly due to the development of a changed or "off" flavour which first became apparent after four weeks. This off flavour increased in intensity during further storage and the flavour of the stored samples became significantly different from that of the frozen controls after 6 weeks ($P < 0.01$). At this time the flavour of the meat was variously described as bitter, sour or acid. A changed or "off" aroma was also noted by the tasters, but the flavour change was regarded as the major defect.

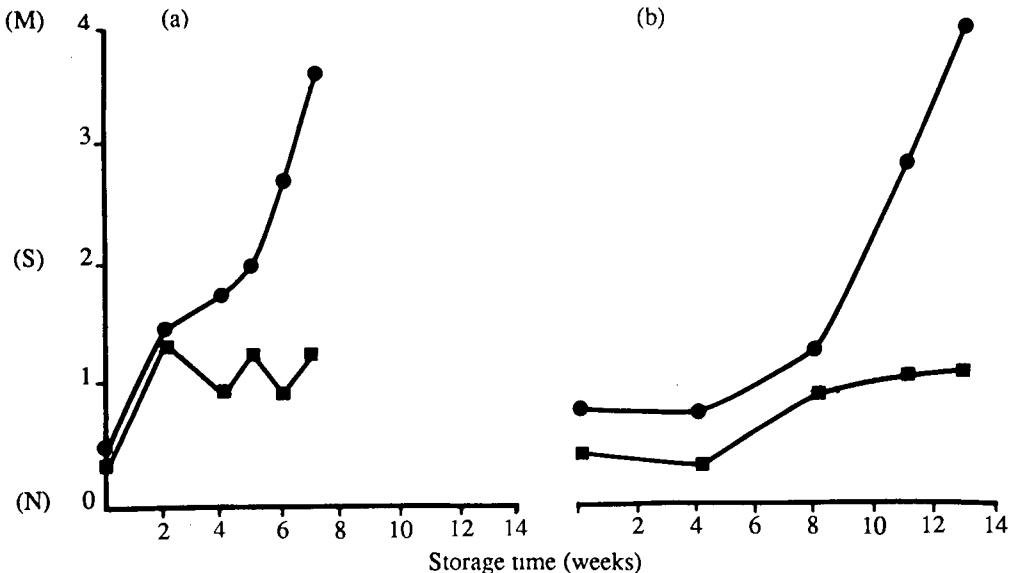


Figure 3. Taste panel evaluation of the development of changed or "off" flavour in low pH (5.5-5.8) vacuum-packaged pork (a) and beef (b) stored at 0°C; (M) = moderate; (S) = slight; (N) = none. Frozen control (solid squares); stored samples (solid circles).

Beef of pH 5.5-5.8 vacuum-packaged and stored under similar conditions was presented to the same taste panel for evaluation. Again spoilage was predominately due to a change in meat flavour but with the beef it only became significant after 11 weeks storage at 0°C ($P < 0.05$).

Whilst the microbiology of vacuum-packaged beef and pork appears to be similar both quantitatively and qualitatively, the storage life of pork is only about half that of beef. Endogenous muscle changes unrelated to bacterial growth may be limiting storage

life, however additional research is necessary to clarify this.

Further experimental work carried out at the Meat Research Laboratory has concentrated on devising procedures that will ensure a storage life of six weeks for pork regardless of pH.

Irradiation of pork

Irradiation is a powerful tool for the destruction of parasites and micro-organisms in meats. A dose of 2.5 kGy destroys vegetative bacteria and so is effective in reducing the populations of pathogens and spoilage organisms. To destroy spores and viruses or to sterilise meat much higher doses are needed.

Following irradiation, meats can be readily contaminated with bacteria. This problem can be overcome by packaging prior to treatment. Vacuum-packaged primal cuts of beef have a storage life of 10-12 weeks at 0°C and there appears to be little scope for further extension using irradiation. However it is beneficial for the extension of storage life at 0-5°C of other types of fresh meats such as vacuum-packaged pork and sheep meats where the increased incidences of high pH meats result in reduced shelf lives.

In 1980, following more than 30 years of research, the World Health Organization Joint Committee on Food Irradiation concluded that foods irradiated with an average dose of up to 10 kGy posed no nutritional, microbiological or toxicological problems. In 1983, the Codex Alimentarius Commission adopted a revised "Recommended International Standard and Code of Practice for Irradiated Foods" which listed low dose treatments for a variety of foods. The flesh foods included chicken and dried fish. The commercial application of this technique to red meats has not progressed as far as it has with poultry, but studies have clearly demonstrated that irradiation can increase the storage life and reduce the public health hazard sometimes associated with red meats (Dempster, 1985). Our work with pork has investigated the possibilities of using low doses (< 5kGy) of radiation to extend the storage life of high pH pork to at least the same as that of normal pH pork.

Shay *et al.* (1988) treated vacuum-packaged high pH pork striploins (pH 6.2-6.6) to a dose of 2.5 kGy, which caused a reduction of 3 log₁₀ units in the number of viable bacteria present. At 4.3 kGy the reduction was greater than 5 log₁₀ units. However both these dose levels caused organoleptic changes. Colour was affected: Lean surface was brighter (pink-red), the skin was pink and there was noticeable bleaching of the fat. Odour was also affected: On opening the packs, an atypical odour described by some panellists as similar to that of a "wet dog" was noted.

The group of organisms causing premature spoilage of vacuum-packaged, high pH pork (namely Gram-negative bacteria) are more sensitive to low dose irradiation treatment than are other spoilage organisms commonly occurring on packaged meats. When the dose was reduced to 1.0 kGy the reduction to the bacterial population was 2 log₁₀ units and the storage life at 0°C was increased from 3-4 weeks to six weeks. However even these low doses caused organoleptic changes that were considered significant by the taste panel (Miller, 1987). The study of Mattison *et al.* (1986) found that irradiation of vacuum-packaged pork at a dose of 1 kGy produced significant organoleptic changes but that these dissipated during storage for 14 days at 4°C. Similar studies carried out by us have confirmed this effect. Further studies of the chemical and biochemical basis of the effect of storage on the organoleptic properties of irradiated pork are needed.

The potential of irradiation cannot be realised at present in commercial practice because its use is either not legal in a number of countries or results in meat being considered as processed. Recently, an all-party House of Representatives Standing Committee on Environment, Recreation and the Arts recommended that food

irradiation should not be introduced to Australia until such time as a routine method of detecting irradiated foods had been developed (December, 1988).

Carbon dioxide storage

The anti-microbial effects of carbon dioxide have been known for a long time. In spite of this, the mechanism by which carbon dioxide inhibits cell activity and microbial growth is poorly understood. Further, there is minimal detailed data concerning the extent to which carbon dioxide influences the composition of the microbial flora of meat. However, it has been clearly established that bacteria vary markedly in their sensitivity to carbon dioxide and that the Gram-negative bacteria which spoil meat stored in air are relatively sensitive (Haines, 1933). As a result, when meat is stored under carbon dioxide the usual Gram-negative flora is replaced by the more CO₂-resistant organisms such as lactic acid bacteria and *Brochothrix thermosphacta* (Seideman *et al.*, 1976; Newton *et al.*, 1977).

Enfors *et al.* (1979) demonstrated that the time required for the total count to reach $5 \times 10^6/\text{cm}^2$ under carbon dioxide storage was seven times longer than in air. Our experimental work (Egan and Shay, 1984) has demonstrated that it is the Gram-negative group of bacteria that cause the early spoilage of vacuum-packaged pork. In a typical experiment high pH pork was stored at 0°C in a modified atmosphere containing greater than 90% carbon dioxide. The microbial growth on the lean surface was compared with that of vacuum-packaged high-pH pork (Figure 4). The carbon dioxide essentially prevented the growth of the Gram-negative bacteria and *B. thermosphacta*. Whilst it slightly inhibited the rate of growth of the lactic acid bacteria, these organisms grew to a population of almost $10^8/\text{cm}^2$ after six weeks storage. Since the growth of the putrefactive Gram-negative bacteria was suppressed, this technique has the potential for considerably extending storage life.

Whilst storage in a carbon dioxide atmosphere reduces significantly the rate of bacterial spoilage of pork there are a number of practical problems that complicate the usefulness of this technique. The gas dissolves in the water phase of the meat, the amount going into solution being dependent of the temperature and (to some extent) the degree of fat cover. The amount of carbon dioxide required to maintain a concentration adequate to cause inhibition of bacterial growth during storage is about 1-1.5 l/kg of meat. This means that packs are moderately inflated which leads to problems in handling. Further if there are fluctuations in temperature the amount of gas in solution in the meat changes, and the volume of the pack alters.

Recent developments in packaging technology have resulted in the availability of equipment which largely overcomes these problems. This equipment should enable pork to be stored for periods of up to three months and hence transported to markets anywhere in the world without spoilage. However to achieve this a temperature no higher than -1°C is required during storage and the oxygen concentration must be very low.

Treatment with organic acids prior to packaging

By far the most important factors in controlling the degree of initial contamination of fresh meat are the practices used during slaughtering and dressing procedures. However in spite of increased attention being given to the use of hygienic practices, carcasses still become contaminated with bacteria. Additional processes can be used to reduce the contamination of carcasses, or (after boning) of primal cuts.

Organic acids, such as lactic acid and acetic acids, occur naturally in a variety of food and have anti-microbial effects that are well documented. Dilute solutions of lactic acid (\approx 1-2%) have been recommended for the decontamination of carcasses and

offal meats (reviewed by Smulders *et al.*, 1986). The use of a dilute solution of acetic acid to decontaminate sheep carcasses prior to vacuum-packaging, has been shown to result in an increase in storage life (Eustace, 1984).

Little information is available on the effect of treatment with organic acids on the storage life of pork. We have investigated the effectiveness of treatment with lactic and acetic acid for this purpose (Shay *et al.*, 1988). The target organisms for these treatments were, once again, the Gram-negative bacteria and our studies have concentrated on the effects of the acid treatments on this group.

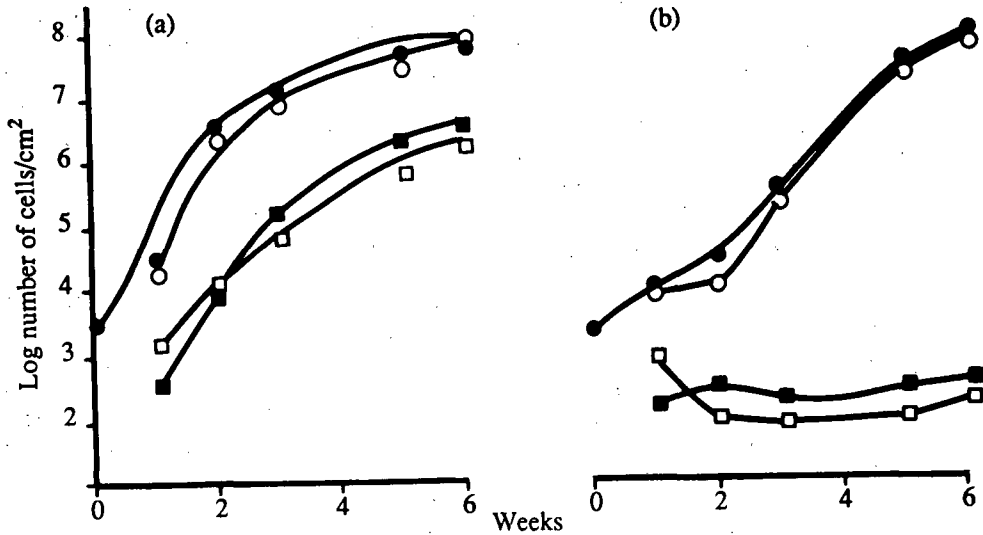


Figure 4. Effect of vacuum-packaging (a) and carbon dioxide (b) on the microflora of high pH (6.0-6.5) pork stored at 0°C. Total count (solid circles); lactic acid bacteria (open circles); *Brochothrix thermosphacta* (solid squares); Gram-negative bacteria (open squares).

Experiments were designed to compare both lactic and acetic acid treatments. Portions of pork loins from the same muscle were dipped in solutions of either acetic (1.5%) or lactic acid (2%) held at 55°C. The exposure time in both cases was 10 sec. A portion of each striploin was kept aside as a non-treated control. Following treatment the meat was drained on a wire rack for 1-2 min prior to vacuum-packaging in the normal manner.

In the first series of experiments, the reductions in the population of Gram-negative bacteria produced by the acetic acid and lactic acid treatment were 50% and 85%, respectively. Gram-negative bacteria grew most rapidly on the vacuum-packaged pork which had not been treated with acid and reached a population in excess of $10^6/\text{cm}^2$ after about two weeks storage (Figure 5). The growth of the Gram-negative bacteria was inhibited by the acid treatments, and these organisms reached a population of about $10^6/\text{cm}^2$ after about two and a half and four weeks storage for the lactic and acetic acid treatments respectively.

The meat which had not been acid treated spoiled the most rapidly. Putrid odours were detected after three weeks. After four weeks storage the meat was slightly green due to sulphmyoglobin formation. Meat treated with lactic acid had a putrid odour after five weeks storage and greening had occurred after six weeks. No putrid odour or green discolouration was detected in the case of meat treated with acetic acid, even after six weeks storage.

Treatment with acetic acid extended the storage life of vacuum-packaged high pH pork from about three weeks to six weeks at 0°C. In the case of meat treated with lactic acid, the storage life was about five weeks. Acetic acid seems to be somewhat more effective for this purpose than lactic acid, since this result was obtained in two independent storage trials. Acetic acid treatment resulted in a more prolonged inhibition of the growth of the Gram-negative bacteria than did lactic. The pH of the meat used in these experiments was 6.2-6.5. The acid treatment is likely to be less effective if meat pH is even higher. In addition if storage temperature is not maintained at 0°C, the effectiveness of this treatment will be reduced. At a storage temperature of 5°C very little benefit is obtained from acetic acid treatment in terms of storage life extension (Shay and Egan, 1986). Acetic acid treatment of pork just prior to vacuum-packaging appears to be a simple effective and inexpensive treatment to extend the storage life of high pH pork such that all pork would have a life of at least six weeks at 0°C.

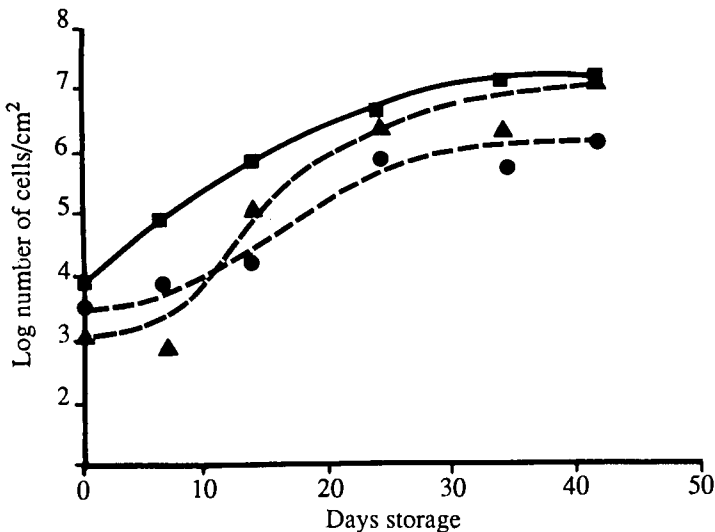


Figure 5. The effect of treatment with dilute acids on the growth of Gram-negative bacteria on the lean surfaces of vacuum-packaged high pH (6.2-6.5) pork stored at 0°C. Untreated (solid squares); treated with acetic acid (solid circles); treated with lactic acid (solid triangles).

Recently a trial commercial shipment of pork which had been acetic acid treated was transported to Japan by sea. The primal cuts treated were collars and strip loins. The pH values of the meat ranged from 5.5-6.7. Delivery air temperature of the refrigeration unit of the container was set accurately at -1.5°C. Temperature monitoring devices placed in the container supplied data which demonstrated that after equilibration the coolest meat was at -1.2°C and the warmest meat was kept below 0.8°C. Detailed microbiological analyses showed that the meat regardless of pH value was in a sound microbiological condition, with Gram-negative numbers on acetic acid treated meat not exceeding $10^4/\text{cm}^2$.

Some untreated control packs of pork included in this trial exhibited putrefactive and hydrogen sulphide odour upon opening. All treated meat was acceptable and this method provides a commercially practical and effective method of achieving a storage life sufficient to export pork to some markets using sea transport.

Conclusions

- (1) The incidence of meat of high pH (>6.0) is a major factor limiting the storage life of vacuum-packaged pork stored at refrigeration temperatures.
- (2) The storage life achieved by conventional vacuum-packaging procedures is too short to permit export to markets in South-East Asia by sea transport, for which a minimum storage life of six weeks is needed.
- (3) Increased storage life can be achieved by a combination of good manufacturing practice, treatment of the meat with a dilute solution of acetic acid prior to packaging, the use of packaging films of very low permeability and storage at temperatures of 0-2°C.
- (4) Storage in an atmosphere of carbon dioxide at similar temperatures can also be used to store pork for prolonged periods, however the oxygen concentration must be very low.

FACTORS AFFECTING THE MANUFACTURING PROPERTIES OF PIG MEAT

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Introduction

It is estimated that approximately 55% of the pigmeat produced in Australia in 1986 was used in the manufacture of smallgoods. Between 30-40% of this meat was used in the production of ham and bacon. In these products the structure of muscle is still readily identifiable. Meat for them is generally obtained from carcasses weighing below 80 kg. The remaining 60-70% of the meat is used for the production of sausage type products in which the meat is usually comminuted to the degree that the original structure of muscle is no longer apparent. Such comminution, and the addition of spices that enable undesired flavours to be masked, means that meat for this purpose can be derived from larger pigs, as well as sows and boars.

The aim of this paper is to define for the various product groups, the properties desired in the pigmeat employed. Factors such as water holding capacity, colour and pH are considered. The type of pig best suited as a source of pigmeat for manufacture is discussed.

Manufactured meats

Constraints imposed by product range

In Table 16 the main groups of products manufactured using pigmeat are listed.

Table 16. Pigmeat used in manufacturing in 1987

	Meat used	% of total manufacturing pigmeat
Ham and bacon ¹	Carcass less than 80 kg and backfat less than 20 mm	40-45
Fermented sausage	From sow or boar or carcass generally greater than 80 kg and 20 mm backfat	5-10
Cooked sausage	Any pigmeat	30-45
Fresh sausage	Any pigmeat	10-15

¹Derived from Australian Bureau of Statistics, catalogue number 72150 (the percentage of ham and bacon is accurate but other figures are conjectural).

Ham

Two main types of ham are produced, ham on the bone and reformed ham. With regard to ham on the bone, weight is a restriction, both from the point of view of cost to the consumer and the ability of the consumer to consume the product. Generally hams in excess of 10 kg have limited market appeal. In addition, dark colour, together

with the presence of fat and gristle, can reduce the appeal of these products to consumers. These constraints tend to preclude the use of hindquarters from older and heavier animals. Generally hindquarter and forequarter hams on the bone or formed hams are derived from pigs with carcass weights below 80 kg and backfat thickness less than 20 mm.

Bacon

There are essentially two types of bacon, namely rind-on and rindless bacon. Rind-on bacon generates greater returns to the manufacturer due to lower production costs, but rindless bacon introduces greater flexibility as in the event of changing demand it may be converted into other products. Rindless bacon results in lower yields and higher labour costs, but still must compete with rind-on bacon in the market place. The weight of the middles used in bacon manufacture is dictated by the width of the bacon cutting machine and consumer preferences for a particular muscle conformation, colour and texture. Generally middles of about 10 kg originating from pigs about 65 kg carcass weight (90 kg live weight) have the characteristics most acceptable in the market place. Backfat of about 12 mm is appropriate for rind-on bacon whereas backfat thickness up to 30 mm is acceptable for the production of rindless bacon where fat can be trimmed to give the desired fat thickness.

Fermented sausage

Fermented sausage is generally manufactured from meat and fat derived from carcasses weighing greater than 120 kg. In Europe the pigs used for salami manufacture are culled sows at least 10 weeks after weaning and specially fattened boars or castrates. In Australia sows past their reproductive peak, sows mated to take advantage of their improved feed conversion ratio, specially fattened castrates and boars not required for breeding are used.

Boar taint is not a great problem as meat from boars can be diluted with meat from animals culled from the breeding stock or castrates. The fermentation process masks any taint. With regard to the use of sow meat there is the problem of how long after weaning of her litter the sow should be kept before slaughter. Sows slaughtered within one week of weaning tend to produce carcasses which are limp, frequently referred to as sloppy. However, the effect of this on the resulting salami and the relationship between time after weaning and carcass limpness have not been quantified.

Another property of the carcass that is important in the manufacture of fermented sausage is the hardness of fat. Soft fat reduces the permeability of the fermented sausage and interferes with the curing process. In addition, soft fat gives salami a greasy feel which is objectionable to some consumers. Barton-Gade (1983) suggested that soft fat is a quality defect which leads to a greater tendency to rancidity. The iodine number is used as an indication of the defect. She found that the iodine number is related to the melting point of the fat ($r = -0.78$) and to the linoleic and linolenic acid content ($r = -0.86$). Iodine values above 70 are associated with a significant degree of softness and carcasses with fat giving these higher values are considered unacceptable.

The fatty acid composition and therefore hardness of pig fat is affected by genetic variation in fatty acid deposition. This in turn affects the ratio in body fat between the various preformed fatty acids and those associated with *de novo* synthesis (Metz, 1985).

Breeding leaner pigs reduces the ratio between fat deposition and lean deposition which in turn affects fat quality in two ways. Firstly, the proportion of fat in adipose tissue is decreased, which is associated with a lower cohesiveness of these tissues (resulting, e.g. in separation of eye muscle from fat in bacon). Secondly, and more importantly, the fatty acids in adipose tissue are less saturated as a consequence of a relatively smaller contribution of *de novo* fatty acid synthesis to total deposition. The latter can be overcome by paying adequate attention to the amount and composition

of dietary fat at different stages of growth. For mature pigs, bred for lean meat, and fed on grain and vegetable protein supplements, the fat will be derived mainly from dietary fat and as a consequence will be soft.

Hertzman *et al.* (1986, 1988) investigated the relationship between fatty acid composition of body fat as related to feed composition. They showed that long term stability of fat in pigs was related to the nature of the dietary fats. The fat in fermented sausage is stored for long periods at ambient temperatures and increased rancidity due to the presence of high levels of unsaturated fatty acids could be potentially deleterious.

Cooked sausage

Cooked sausage is manufactured from meat and fat derived from pig, beef and mutton carcasses. The proportion of each will depend on the particular formulation. As the product is made from comminuted meat, the conformation of the muscle, muscle colour, and so forth is of marginal interest. The important factors are the ability of the meat to bind water and fat, the stability of the resulting emulsion, the pH of the muscle, the amount of protein that can be solubilized with salt during tumbling, and the moisture:protein ratio. Meat with lower moisture:protein ratios generally performs better in sausage formulations (e.g. pork head meat with a ratio of 3.6:1 is better than pork jowls with a ratio of 3.72:1 (Judge *et al.*, 1989).

The characteristics of pigs best suited for manufacturing purposes

The quality of the manufactured meat product is the end result of a cascade of events. These events are summarised in the accompanying Figure 6. For the purpose of this exercise it is considered that all events in pig processing and manufacturing have been optimised and standardised. Attention is focussed on pre-processing factors such as genetics and nutrition. Perhaps the factor most affecting manufacturing property is the PSE condition (Honkavaara, 1988).

The PSE condition is manifested in meat derived from stress susceptible pigs. The meat is pale, soft and exudes moisture. The technological influences of PSE pork on meat products are well known (Honkavaara, 1988). There are greater cooking losses, and hams prepared from PSE meat have poorer organoleptic quality.

The effect of gender on the manufacturing properties of pigmeat has been considered by several authors. Barton-Gade (1987) compared meat and fat quality of boars, castrates and gilts. It was found there was no difference in meat quality between the sexes with respect to pigmeat content and characteristics expressing PSE status. In contrast it was found that although boars have a higher meat content in the carcass than castrates and gilts, the processing quality of the meat is slightly poorer.

Middles from boars, gilts and castrates have been compared with regard to their use in bacon manufacture. (Smith *et al.*, 1983a,b; Ellis *et al.*, 1983; Wood and Riley, 1982; Wood and Enser, 1982; Mottram *et al.*, 1982). It has been found that middles from boars (at 89 kg carcass weight) had a higher proportion of carcass weight in the shoulder and ham at the expense of the middle region (Ellis *et al.*, 1983). However, boars tended to yield bacon in which there was a higher incidence of separation of the eye muscle from the inner fat layer. On balance, due to the improved feed conversion ratio and grading profile (see Smith *et al.*, 1983a) there was a financial advantage in the production of entire male pigs relative to castrates for bacon manufacture.

Effect of genotype

The genotype of the pig does have a significant effect on the yield of lean from a carcass and also the quality of meat. To date the industry has relied on measures of subcutaneous fat thickness as a guide to yield. A major component of pig breeding programmes in Australia has been selection against subcutaneous fat at the P₂ site.

This within breed selection has been supplemented with the introduction of new genetic material from overseas. A recent trial comparing Durocs, Hampshires and Australian Large White boars as terminal sires found that the Duroc and Hampshire breeds conferred a significant advantage in carcass lean content at heavier weights under *ad libitum* feeding compared to the whites (Luxford, unpublished data).

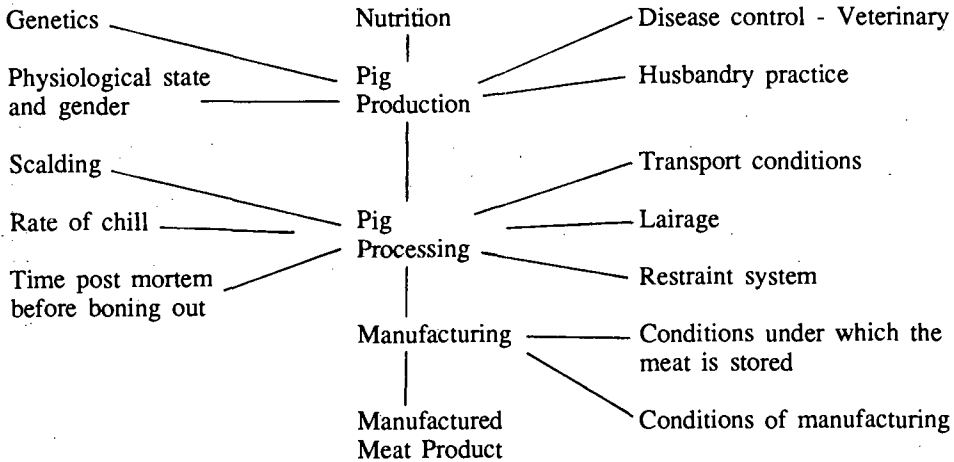


Figure 6. Factors affecting the manufacturing quality of meat derived from a pig carcass.

The effects of increasing the lean content of carcasses on the subsequent quality of the meat and fat has been investigated in a number of studies. In a comparison of carcasses of 8 mm and 16 mm P_2 respectively, Wood *et al.* (1988) found that the leaner carcasses had a higher drip loss, with the fat also being less cohesive and soft. Other studies have concentrated on the meat quality of the lean, heavily muscled, breeds such as the Pietrain and some of the European Landrace breeds. The effect of the Halothane, Hal^+ gene in these studies is of interest. The gene has been associated with increases in the lean content of the carcass and also PSE problems. The action of the gene is thought to be additive for lean content and recessive for the stress or PSE condition. This would allow producers to take advantage of the benefit of the gene while minimizing the losses by utilizing homozygous positive boars and homozygous negative dams (Kalm, 1986). The system would require testing for the presence of the Hal^+ gene in the dam lines. An alternative way of avoiding the problems associated with PSE would be to remove the gene from our current breeds and screen any imported genetic material for the gene.

The possible existence of another major gene influencing meat quality has been canvassed in the US and France. The reputed gene is thought to be dominant and has been associated with the Hampshire breed. The problem is related to a significantly larger decrease in pH post slaughter and results in a poorer yield in processing.

Summary and conclusions

Given the numerous factors affecting quality and consumer acceptability of manufactured meat products (see Figure 6) it is difficult to distinguish any one factor as being more critical than another. However, in respect to ham and bacon

manufacture the pigmeat used must have acceptable conformation, colour and a high moisture retention capacity when cooked. For meat destined for sausage manufacture the important qualities are again moisture retention and fat quality.

At present manufacturing serves both as a means for smoothing out the market fluctuations in the fresh meat market and for the disposal of pigs such as boars and sows which are generally not acceptable for the fresh meat trade. The nature of the pigs produced in Australia at present is governed by the needs of the fresh market.

Nevertheless, both markets share a common requirement for pigmeat with a high water holding capacity. The market differs mainly in respect to the amount of fat contained in the carcass. The fresh meat trade continues to demand leaner pigs whilst the manufacturing trade is less concerned about fat thickness but is particularly concerned about fat quality. The extent to which fat quality is influenced by the various factors outlined in this paper requires further definition.

Symposium continued on next page

SYMPOSIUM CONCLUSION

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Although there is a large growing body of research and technology in the area of pig meat quality available to Australia from Europe and other northern hemisphere countries, we cannot afford to be complacent. Despite this northern hemisphere knowledge bank, problems of great importance to meat quality, for example PSE, remain unsolved. Furthermore it is apparent that our system(s) of pig production are sufficiently different from those in other countries to warrant our own research into pig meat quality. This expertise should also guard against the "importation" of known overseas problems. Such considerations become of even greater significance if our export of pig meat to South-East Asia, China and Japan is to prosper.

Future research efforts are needed to further identify our specific pig meat quality problems and find solutions that are both practical and commercially achievable.

References

- ALLEN, P., TARRANT, P.V., HANRAHAN, J.P. and FITZSIMONS, J.M. (1985). The effect of different levels of cimaterol on the growth and carcass quality of crossbred lambs. (An Foras Taluntais Research Report, Food Science and Technology), p. 6.
- AUSTRALIAN BUREAU OF STATISTICS. Catalogue number 72150 Livestock Products, Australia, May 1988.
- BALTZER, J. and WILSON, D.C. (1965). The occurrence of Clostridia on bacon slaughter lines. *Journal of Applied Bacteriology*. **28**:119-124.
- BARTON-GADE, P.A. (1983). The quality of pork fat. *Slagteriernes Forskningsinstitut, Svin - Spaekkkvalitet*, Manuscript number 643E.
- BARTON-GADE, P.A. (1987). Meat and fat quality in boars, castrates and gilts. *Livestock Production Science*. **16**:187-196.
- BARTON-GADE, P.A. (1988). The effect of breed on meat quality characteristics in pigs. (Proceedings of 34th International Congress of Meat Science and Technology: Brisbane), pp. 568-570, eds. C.S. Chandler and R.F. Thornton.
- BAUMAN, D.E., EPPARD, P.J., DeGETTER, M.J. and LANZA, G.M. (1985). Responses of high-producing dairy cows to long-term treatment with pituitary somatotropin and recombinant somatotropin. *Journal of Dairy Science*. **68**:1352-1362.
- BECHTEL, P.J., EASTER, R.A., McKEITH, F.K., NOVAKOFSKI, J., McLAREN, D.G. and GREBNER, G.L. (1988). Growth, carcass and sensory characteristics for pigs injected daily with natural porcine somatotrophin. (Proceedings of 34th International Congress of Meat Science and Technology: Brisbane), pp. 603-604, eds. C.S. Chandler and R.F. Thornton.
- BEERMANN, D.H., BOYD, R.D., ARMBRUSTER, G., DeNEERGARD, A.F., RONEKER, K., BARTLEY, T.D. and FAGIN, K.D. (1988). Dose response effects of native and recombinant porcine somatotrophin (pST) on growth performance, composition of gain and pork quality. (Proceedings of 34th International Congress of Meat Science and Technology: Brisbane), pp. 601-602, eds. C.S. Chandler and R. F. Thornton.
- BEERMANN, D.H., BUTLER, W.R., HOGUE, D.E., FISHELL, V.K., DALRYMPLE, R.H., RICKS, C.A. and SCANES, C.G. (1987). Cimaterol induced muscular hypertrophy and altered endocrine status in lambs. *Journal of Animal Science*. **65**:1514-1524.
- BEKAERT, H., CASTEELS, N. and BUYSE, F.X. (1987). The effects of a beta-agonist cimaterol on performance, carcass and meat quality of growing-finishing pigs of the Belgian Landrace. In "Beta-Agonists and Their Effects on Animal Growth and Carcass Quality", pp.127-136, ed. J.P. Hanrahan, (Elsevier Applied Science Publishers Ltd.: London and New York).
- BEM, Z., HECHELMANN, H. and LEISTNER, L. (1976). Mikrobiologie des DFD-Fleisches. *Die Fleischwirtschaft*. **56**:985-987.
- BROCKWAY, J.M., MacRAE, J.C. and WILLIAMS, P.E.V. (1987). Side effects of clenbuterol as a repartitioning agent. *The Veterinary Record*. **120**:381.

- COLE, D.J.A., WOOD, J.D. and KILPATRICK, M.J. (1987). Effects of the beta-agonist GAH/034 on growth, carcass quality and meat quality in pigs. In "Beta-Agonists and their Effects on Animal Growth and Carcass Quality", p.137-142, ed. J.P. Hanrahan, (Elsevier Applied Science Publishers Ltd.: London and New York).
- DAINTY, R.H., SHAW, B.G., HARDING, C.D. and MICHANIE, S. (1979). In "Cold Tolerant Microbes in Spoilage and the Environment", pp. 83-100, eds. A.D. Russel, and R. Fuller, (Academic Press: London).
- DEMPSTER, J.F. (1985). Radiation preservation of meat and meat products: A review. *Meat Science*. 12:61-89.
- EGAN, A.F. (1983). Lactic acid bacteria of meat and meat products. *Antonie von Leeuwenhoek*. 49:327-336.
- EGAN, A.F. and SHAY, B.J. (1982). Significance of lactobacilli and film permeability in the spoilage of vacuum-packaged beef. *Journal of Food Science*. 47:1119-1122 and 1126.
- EGAN, A.F. and SHAY, B.J. (1984). The microbiology of vacuum-packaged pork. (Proceedings of 30th European Meeting of Meat Research Workers: Bristol), pp. 215-216.
- ELLIS, M., SMITH, W.C., CLARK, J.B.K. and ININGS, N. (1983). A comparison of boars, gilts and castrates for bacon manufacture. *Animal Production*. 37:1-9.
- ENFORS, S.O., MOLIN, G. and TERNSTROM, A. (1979). Effect of packaging under carbon dioxide, nitrogen or air on the microbial flora of pork stored at 4°C. *Journal of Applied Bacteriology*. 47:197-208.
- ERICHSEN, I., MOLIN, G. and MOLLER, B.M. (1981). Carbon dioxide packaging as a means of controlling the spoilage flora of DFD meat. (Proceedings of 27th European Meeting of Meat Research Workers: Wien), pp.683-685.
- EUSTACE, I.J. (1984). Prolongation of the storage life of vacuum-packaged lamb. *CSIRO Food Research Quarterly*. 44:60-67.
- GILL, C.O. and NEWTON, K.G. (1979). Spoilage of vacuum-packaged dark, firm, dry meat at chill temperatures. *Applied and Environmental Microbiology*. 37:362-364.
- GERATS, G.E., SNIDJERS, J.M.A. and Van LOGTESTIJN, J.C. (1981). Slaughter techniques and bacterial contamination of pig carcasses. (Proceedings of the 27th European Meeting of Meat Research Workers: Vienna), volume I, pp. 198-200.
- HAINES, R.B. (1933). The influence of carbon dioxide on the rate of multiplication of certain bacteria, as judged by viable counts. *Journal of the Society of Chemical Industry*. 52:13T-17T.
- HANCOCK, J.D., PEO, E.R. Jr., LEWIS, A.J. and PARROTT, J.C. (1987). Effects of dietary levels of ractopamine (aphenethanolamine) on performance and carcass merit of finishing pigs. *Journal of Animal Science*. 65 (Supplement 1):309.
- HANRAHAN, J.P. (Editor) (1987). "Beta-Agonists and their Effects on Animal Growth and Carcass Quality" (Elsevier Applied Science Publishers Ltd.: London and New York).
- HERBERT, F., HOVELL, F.D. Deb., REEDS, P.J. (1985). Some preliminary observations on the immediate effects of clenbuterol on heart rate, body temperature and nitrogen retention on lambs wholly nourished by intragastric infusion. *Proceedings of Nutrition Society*. 44:150A.
- HERMANSEN, P. (1980). The spoilage of vacuum-packaged pork with high ultimate pH. (Proceedings of 26th European Meeting of Meat Research Workers: Colorado Springs), pp. 300-303.
- HERTZMAN, C., FERGUSON, D.M., SHORTHORSE, W.R., HARRIS, P.V., WILLIAMS, K.C., SNOSWELL, M., BIGGS, J.S. and THORNTON, R.F. (1989). Meat quality, carcass composition and growth of pigs treated with recombinant porcine growth hormone. (Proceedings of 35th International Congress of Meat Science and Technology: Copenhagen), in press.
- HERTZMAN, C., GORANSSON, L. and RUDERUS, H. (1986). Influence of feed composition in composition and stability of porcine body fat in animal fat, resources - properties - refining - application. (Lipidforum Symposium, AS: Oslo), Oct. 27-28.
- HERTZMAN, C., GORANSSON, L. and RUDERUS, H. (1988). Influence of fishmeal, rope-seed, and rope-seed meal in feed on the fatty acid composition of storage, stability of porcine body fat. *Meat Science*. 23:37-53.
- HONKAVAARA, M. (1988). Influence of PSE pork on the quality and economics of cooked, cured ham and fermented dry sausage manufacture. *Meat Science*. 24: 201-207.
- INGRAM, M. (1972). Meat preservation - past, present and future. *Royal Society of Health Journal*. 92:121-130.
- JOHANSON, L., UNDERDAHL, B., GROSLAND, K., WHELEHAN, O.P. and ROBERTS, T.A. (1983). A survey of the hygienic quality of beef and pork carcasses in Norway. *Acta Veterinaria Scandinavica*. 24:1-13.
- JONES, B., NILSSON, T., EKMAN, L. and OSTLUND, K. (1979). The contamination of pig carcasses with scalding water studied with a radiolabelled colloid. *Die Fleischwirtschaft*. 59:1511-1514.
- JONES, B., NILSSON, T. and SORQVIST, S. (1984). Contamination of pig carcasses with scalding water. Continued studies with radiolabelled solutes and particles. *Die Fleischwirtschaft*. 64:1226-1228.

- JONES, R.W., EASTER, R.A., McKEITH, F.K., DALRYMPLE, R.H., MADDOCK, H.M. and BECHTEL, P.J. (1985). Effect of the β -adrenergic agonist cimaterol (CL263,780) on the growth and carcass characteristics on finishing pigs. *Journal of Animal Science*. 61:905.
- JUDGE, M.D., ABERLE, G.D., FORREST, J.C., HEDRICK, H.B. and MERKEL, R.A. (1989). "Principles of Meat Science" (Kendall/Hunt Publishing Company: New York).
- KALM, E. (1986). Evaluation and utilisation of breed resources as sire lines in crossbreeding. (3rd World Congress Genetics Applied Livestock Production: Lincoln, Nebraska), volume X, pp. 35-44.
- KELLY, C.A., DEMPSTER, J.F. and McLAUGHLIN, A.J. (1981). The effect of temperature, pressure and chlorine concentration of spray washing water on numbers of bacteria on lamb carcasses. *Journal of Applied Bacteriology*. 51:415-424.
- KEMPSTER, A.J. (1987). Consumer attitudes to meat quality. In "Recent Advances in Animal Nutrition - 1987" pp.127-134, eds. W. Haresign and D.J.A. Cole (Butterworths: London).
- KITCHELL, A.G., INGRAM, G.C. and HUDSON, W.R. (1973). Microbial sampling in abattoirs. In "Sampling: Microbial Monitoring of Environments" pp. 43 (Society for Applied Bacteriology, Technical Series Number 7 (Academic Press: London).
- MATTISON, M.L., KRAFT, A.A., OLSON, D.G., WALKER, H.W., RUST, R.E. and JAMES, D.B. (1986). Effect of low dose irradiation of pork loins on the microflora, sensory characteristics and fat stability. *Journal of Food Science*. 51:284-287.
- McKEITH, F.K., SINGH, S.D., STITES, C.R., BECHTEL, P.J. and JONES, D.J. (1988). Palatability and visual characteristics of hams and loin chops from swine fed ractopamine hydrochloride. *Journal of Animal Science*. 66 (Supplement 1): 306.
- MERKEL, R.A. (1988). Is meat quality affected by the use of repartitioning agents. *Proceedings of Reciprocal Meat Conference*. 41:101-111.
- MERKEL, R.A., BURKETT, R.L., BURNETT, R.J., BABIKER, A.S., SCHROEDER, A.L., BERGEN, W.G. (1988). Qualitative properties and carcass composition of pigs fed ractopamine. (Proceedings of 34th International Congress of Meat Science and Technology Part B: Brisbane), p.605-606, eds. C.S. Chandler and R.F. Thornton.
- METZ, S.H.M. (1985). Genetic effects on fat deposition and fat quality in the growing pig. *Pig News and Information*. 6:291-294.
- MILLER, D.E. (1987). Studies of Techniques for Extending the Storage Life of Vacuum-packaged Pork. (Master of Philosophy Thesis, Griffith University: Brisbane).
- MORGAN, I.R., KRAUTIL, F.L. and CRAVEN, J.A. (1987). Bacterial populations on dressed pig carcasses. *Epidemiology and Infection*. 98:15-24.
- MOSER, R.L., DALRYMPLE, R.H., CORNELIUS, S.G., PETTIGREW, J.P., ALLEN, C.E. (1986). Effect of cimaterol (CL263,780) as a repartitioning agent in the diet for finishing pigs. *Journal of Animal Science*. 62:21.
- MOTTRAM, D.J., WOOD, J.D. and PATTERSON, R.L.S. (1982). Comparison of boars and castrates for bacon production. *Animal Production*. 35:75-80.
- NEWTON, K.G. and GILL, C.O. (1980-81). The microbiology of DFD fresh meat: a review. *Meat Science*. 5:223-232.
- NEWTON, K.G., HARRISON, J.C.L. and SMITH, K.M. (1977). The effect of storage in various gaseous atmospheres on the microflora of lamb chops held at -1°C . *Journal of Applied Bacteriology*. 43:53-59.
- NEWTON, K.G. and RIGG, W.J. (1979). The effect of film permeability on the storage life and microbiology of vacuum packed meat. *Journal of Applied Bacteriology*. 47:433-441.
- NICOL, D.J., SHAW, M.K. and LEDWARD, D.A. (1970). Hydrogen sulphide production by bacteria and sulfmyoglobin hydrogen formation in repacked chilled beef. *Applied Microbiology*. 19:937-939.
- NORTJE, C.L., VISSER, D., HOLZAPFEL, W.H. and NAUDE, R.T. (1980). The influence of the dressing procedure on the (mesophilic) bacterial population of baconer carcass surfaces. *South African Journal of Animal Science*. 9:53-57.
- PATTERSON, J.T., and GIBBS, P.A. (1977). Incidence and spoilage potential of isolates from vacuum-packaged meat of high pH value. *Journal of Applied Bacteriology*. 43:25-38.
- RASCH, B., LIE, O. and YNDESTAD, M. (1978). Bakteriefloeraen i svinesvor og forskjellige svimetoders innflytelse pa floeraen. (The bacterial flora in pork skin and the influence of various singeing methods on this flora). *Nordisk veterinærmedecin*. 30:274-281.
- RICKS, C.A., BAKER, P.K. and DALRYMPLE, R.H. (1984). Use of repartitioning agents to improve performance and body composition of meat animals. *Proceedings of Reciprocal Meat Conference*. 37:5-11.
- ROBERTS, T.A. (1980). Contamination of Meat. The effect of slaughter practices on the bacteriology of red meat carcass. *Royal Society of Health Journal*. 100:3-9.
- ROBERTS, T.A., MacFIE, H.J.H. and HUDSON, W.R. (1980). The effect of incubation temperature and site of sampling on assessment of the numbers of bacteria on red meat carcasses at commercial abattoirs. *Journal of Hygiene (Cambridge)*. 85:371-380.

- SEIDEMAN, S.C., VANDERZANT, C., HANNA, M.O., CARPENTER, Z.L. and SMITH, G.C. (1976). Effect of various types of vacuum packages and length of storage on the microbial flora of wholesale and retail cuts of beef. *Journal of Food and Milk Technology*. 39:745-753.
- SCOTT, W.J. and VICKERY, J.R. (1939). Investigations on chilled beef. Part II "Cooling and storage in meatworks" Bulletin No.129 (Council of Scientific and Industrial Research: Australia).
- SHAY, B.J. and EGAN, A.F. (1986). Studies of possible techniques for extending the storage life of chilled pork. *Food Technology in Australia*. 38:144-146.
- SHAY, B.J., EGAN, A.F., MILLER, D. and TIAN, A.J. (1988). Treatment of pork with organic acids prior to vacuum-packaging - a comparison of the effectiveness of lactic acid and acetic acids. (Proceedings of 34th International Congress of Meat Science and Technology: Brisbane), pp. 489-491, eds. C.S. Chandler and R.F. Thornton.
- SHAY, B.J., EGAN, A.F. and WILLS, P.A. (1988). The use of irradiation for extending the storage life of fresh and processed meats. *Food Technology in Australia*. 40:310-313.
- SHERIDAN, J.J. (1982). The effect of freezing on the microbiology and keeping quality of pork and bacon. *Pig News and Information*. 3:275-281.
- SMITH, W.C., ELLIS, M., CLARK, J.B.K. and INNES, N. (1983a). A comparison of boars, gilts and castrates for bacon manufacture. *Animal Production*. 37:17-23.
- SMITH, W.C., ELLIS, M., CLARK, J.B.K. and INNES, N. (1983b). A comparison of boars, gilts and castrates for bacon manufacture. *Animal Production*. 37:11-15.
- SMULDERS, F.J.M., BARENDSEN, P., VAN LOGTESTIJN, J.G., MOSSEL, D.A.A. and VAN DER MAREL, G.M. (1986). Lactic Acid: Considerations in favour of its acceptance as a meat decontaminant. *Journal of Food Technology*. 21:419-436.
- SNIJDERS, J.M.A. (1975). Hygiene bei der schlachtung von schweinen. 1. Das brühen der schlachttschweine. (Pig slaughtering hygiene. 1. Scalding). *Die Fleischwirtschaft*. 55: 836-840.
- SNIJDERS, J.M.A. and GERATS, G.E. (1976). Hygiene bei der schlachtung von schweinen. IV. Bakteriologische beschaffenheit der schlachtierkörper während verschiedener schlachtphasen. (Pig slaughtering hygiene. IV. Bacteriological status of carcasses in various sections of the killing line). *Die Fleischwirtschaft*. 56:717-721.
- SNIJDERS, J.M.A., GERATS, G.E. and CORSTIAENSEN, G.P. (1977). Hygiene bei der schlachtung von schweinen. V. Verwendung chlorierten wassers bei der reinigung der tierkörperoberflächen. (Pig slaughtering hygiene. V. Chlorinated water to clean carcasses). *Die Fleischwirtschaft*. 57:2212-2215.
- TARRANT, V. (1989). The effects of handling, transport, slaughter and chilling on meat quality and yield in pigs. In "Manipulating Pig Production II", pp. 1-25, eds. J.L. Barnett and D.P. Hennessy (Australasian Pig Science Association: Werribee, Victoria, Australia).
- TAYLOR, A.A. and SHAW, B.G. (1977). The effect of meat pH and package permeability on putrefaction and greening in vacuum-packaged beef. *Journal of Food Technology*. 12:515-521.
- THORNTON, R.F. (1987). The partitioning of nutrients by herbivores. In "The Nutrition of Herbivores" pp. 307-331, eds. J.B. Hacker and J.H. Ternouth (Academic Press: London).
- THORNTON, R.F., ADAMSON, D., HARRIS, P.V., SHORTHORSE, W.R. and WILLIAMS, K.C. (1989). Meat quality of pigs fed cimaterol. In "Manipulating Pig Production II" p. 71, eds. J.L. Barnett and D.P. Hennessy (Australasian Pig Science Association: Werribee, Victoria, Australia).
- THORNTON, R.F. and TUME, R.K. (1988). Manipulation of growth in domestic animals. (Proceedings of 34th International Congress of Meat Science and Technology: Brisbane) pp. 6-14, eds. C.S. Chandler and R. F. Thornton.
- THORNTON, R.F., TUME, R.K., WYNN, P.C., LARSEN, T.W. and JOHNSON, G.W. (1987). Modes of action of repartitioning agents in sheep. (Proceedings of 33rd International Congress of Meat Science and Technology: Helsinki), pp. 28-30.
- TROEGER, K. and WOLTERS DORF, W. (1986). Einfluß des brühens und entborstens bei der schweinefleischschlachtung auf die fleischbeschaffenheit. (Influence of scalding and dehairing during pig slaughter on meat quality. *Die Fleischwirtschaft*. 66:893-897.
- Van WEERDEN, E.J. (1987). Effects of clenbuterol on N deposition and carcass composition on castrated male pigs. In "Beta-Agonists and their Effects on Animal Growth and Carcass Quality" pp.152-162, ed. J.P. Hanrahan (Elsevier Applied Science Publishers Ltd.: London and New York).
- WALLACE, H.D., HEDRICK, H.B., SEWARD, R.L., DAURIO, C.P. and CONVEY, E.M. (1987). Growth and efficiency of feed utilization of swine fed a beta-adrenergic agonist (L-644,969). In "Beta-agonists and their Effects on Animal Growth and Carcass Quality" pp. 143-151, ed. J.P. Hanrahan (Elsevier Applied Science Publishers Ltd.: London and New York).
- WOOD, J.D. and BICHARD, M. (1988). Influence of the Duroc breed on pigmeat quality. (Proceedings of 34th International Congress of Meat Science and Technology: Brisbane), pp. 571-572, eds. C.S. Chandler and R.F. Thornton.
- WOOD, J.D. and BROWN, A.J. (1987). Effects of beta agonist GAH/034 on carcass composition and meat quality in pigs. *Animal Production*. 44:477.
- WOOD, J.D., BROWN, A.J., KILPATRICK, M.J. and BUSHHELL, J.E. (1987). Effects of beta-agonist GAH/034 on carcass composition and meat quality in pigs. *Animal Production*. 44: 477.

- WOOD, J.D. and ENSER, M. (1982). Comparison of boars and castrates for bacon production. *Animal Production*. 35:65-74.
- WOOD, J.D., ENSER, M. and MONCRIEFF, C.B. (1988). Effect of carcass fatness and sex on the composition and quality of pigmeat. (Proceedings of 34th International Congress of Meat Science and Technology Part B: Brisbane), pp. 562-564, eds. C.S. Chandler and R. F. Thornton.
- WOOD, J.D. and RILEY, J.E. (1982). Comparison of boars and castrates for bacon production. *Animal Production*. 35:55-63.

AN ASSESSMENT OF THE HENNESSY GRADING PROBE FOR USE IN PIG CARCASS CLASSIFICATION

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Two studies were carried out, the first to compare the precision of the intrascope and Hennessy grading probe in measuring backfat thickness in pig carcasses on the slaughter line and the second to determine the relative accuracy of the two instruments in predicting carcass lean content from their best combination of measurements. In the first study, one backfat measurement, sited 4 cm off the mid-line of the carcass at the last rib (C_4), was taken on 346 carcasses (45-72 kg) with the intrascope and grading probe operated by one person experienced in the use of both instruments. After approximately 4 h in the chillroom, a random sample of the carcasses (101) was cut and C_4 measured with callipers. There was a close relationship between C_4 measurements taken with the two instruments ($r=0.91$) but the accuracy of the prediction of measured C_4 was higher for the Hennessy grading probe ($C_4=1.29 + 0.94\text{HGP}$; $\text{RSD} \pm 1.03$) than the intrascope ($C_4=2.28 + 0.87\text{I}$; $\text{RSD} \pm 1.42$).

In the second study, fat and lean measurements were taken with the intrascope and grading probe on 96 carcasses (62-72 kg) in which two genotypes (Landrace x {Landrace x Large White} and Hampshire x {Landrace x Large White}) and three rearing environments (farms) were represented. The carcasses were subsequently dissected into their component tissues. Use of the grading probe in place of the intrascope to measure $C_{6.5}$ at the last rib reduced the residual standard deviation (RSD) of prediction of carcass lean content by 8%. The addition of a second fat measurement ($C_{6.0}$ at the 3/4th last rib) gave a further modest improvement (4%) in the RSD of predicted carcass lean content but the inclusion of an "eye muscle" depth measurement in the equation was not significant. One prediction equation was adequate for determining carcass lean content in crossbred pigs of Landrace, Large White breeding from the different farms ($Y=69.96 - 0.84\text{HGP}_{6.5} \pm 1.85$) but at the same backfat depth as the white-cross pigs, Hampshire crosses had a higher lean content (1.7% units). Consequently, use of an independent prediction equation was necessary if the extra lean in Hampshire crosses was to receive recognition in a classification scheme. These results agree with overseas findings (Kempster *et al.*, 1985; Cook *et al.*, 1989), except that in the latter the inclusion of eye muscle depth with fat measurements in the regression equations has significantly improved the precision of lean prediction.

Overall, the Hennessy grading probe appears to offer advantages over the intrascope in the accuracy of measurement of backfat depths and in the prediction of carcass lean content. These considerations, in conjunction with the probe being an integral part of an automated data capture system and capable of the objective measurement of meat colour, give encouragement for use of the instrument in commercial pig carcass classification schemes.

References

- COOK, G.L., CHADWICK, J.P. and KEMPSTER, A.J. (1989). *Animal Production*. 48:427-434.
KEMPSTER, A.J., CHADWICK, J.P. and JONES, D.W. (1985). *Animal Production*. 45:323-330.

COMPARATIVE GROWTH PERFORMANCE, CARCASS COMPOSITION AND MEAT QUALITY OF PIGS Sired BY DUROC, HAMPSHIRE, LANDRACE AND LARGE WHITE BOARS

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A production trial was undertaken on one farm to evaluate the Duroc and Hampshire as terminal sires in a three-way cross compared to a two-way backcross with the Landrace and Large White. Three boars of each sire breed were mated with approximately 50 "white cross" sows and the progeny grown to 85 kg live weight on *ad libitum* feeding. Carcass measurements were taken on random samples (100) of the crossbred types and 12 carcasses of each cross, balanced for sex, were physically dissected into lean, fat, bone and rind. Quality assessments which included ultimate pH, colour and drip loss, were made on samples of *longissimus dorsi* muscle and cooked meat was evaluated by a taste panel.

Litters sired by Duroc and Hampshire boars had relatively fewer ($P < 0.001$) stillbirths, but breed of sire did not influence the number of live births in the litter nor litter size at weaning. Duroc and Hampshire crosses reached slaughter weight at 151 days, which was four days earlier ($P < 0.05$), on average, than Landrace and Large White crosses, had higher ($P < 0.05$) carcass yields (0.8 kg) and lower ($P < 0.05$) backfat measurements (3-5 mm). Duroc and Hampshire crossbreds did not differ significantly ($P > 0.05$) in carcass lean content 64.0 vs. 62.1% but this was higher ($P < 0.05$) compared with Landrace and Large White crosses (59.4 and 60.1%, respectively).

Meat from Hampshire crosses was paler ($P < 0.05$) in colour, of lower ($P < 0.05$) ultimate pH and had a higher ($P < 0.05$) drip loss compared with Landrace and Large White crosses, with Duroc crosses having intermediate values. Mean intramuscular fat level was highest in Duroc crosses but not significantly different ($P > 0.05$) from the other crosses. The taste panel did not detect any significant ($P > 0.05$) breed difference in the eating quality of cooked *longissimus dorsi* muscle samples, excepting that meat from Hampshire crosses had a relatively stronger ($P < 0.05$) flavour.

The findings of the production trial receive the support of earlier studies (Smith and Pearson, 1988; Smith *et al.*, 1988). Overall, they suggest that benefits in economy of production and carcass composition are likely to arise from using the Duroc and Hampshire breeds as terminal sires on "white cross" dams compared with Landrace and Large White. The outcome regarding meat quality of the crosses is not so clear, in particular the condition of the muscle tissue in Hampshire crosses and intramuscular fat level and its association with eating quality of the meat in Duroc crosses.

References

- SMITH, W.C. and PEARSON, G. (1988). *New Zealand Journal of Agricultural Research*. 31:307-310.
SMITH, W.C., PEARSON, G. and GARRICK, D.J. (1988). *New Zealand Journal of Agricultural Research*. 31:421-430.

SALBUTAMOL (β -AGONIST) AND MUSCLE FIBRE TYPE IN PIGS

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Chronic administration of β -agonists causes skeletal muscle hypertrophy. In ruminants, this is due to a conversion of small, aerobic fibres (Type I and Type IIa) to large anaerobic fibres (Type IIb). A trial was conducted to test the effects of the β -agonist salbutamol on muscle fibre type in pigs. Six pairs of littermate Landrace x Yorkshire pigs were fed twice daily a diet with or without 3 ppm salbutamol from 50-90 kg body weight. Following slaughter and carcass evaluation, samples of *longissimus dorsi* muscle at the P₂ position were taken and frozen in isopentane cooled in liquid nitrogen. Frozen serial sections (10 μ) were stained for myosin adenosine triphosphatase (Brooke and Kaiser, 1970), after acid and alkaline pre-incubation, and by reduced nicotinamide adenine dinucleotide tetrazolium reductase (Novikoff *et al.*, 1961) for oxidative fibres. Capillaries and cell boundaries were stained by the α -amylase periodic acid Schiff method (Andersen, 1975). Sections were evaluated for capillary number, and fibre type distribution (Types I, IIa, IIb and oxidative, nonoxidative) using an image analyzer (Comfas, Hadsund, Denmark). Volumes of the fibre types were calculated as the product of the relative area of each type and the weight of the dissected *longissimus dorsi* muscle.

Table 1. Effect of salbutamol on growth and muscle fibre type in the pig (mean values \pm SE)

	Control	Salbutamol	t
Protein (g/day)	107 ^x \pm 8	153 ^y \pm 7	3.92
Daily gain (g/day)	735 ^a \pm 19	814 ^b \pm 26	2.46
Feed:gain	3.0 \pm 0.1	2.8 \pm 0.1	0.71
<i>M. longissimus dorsi</i> (kg)	5.1 ^x \pm 0.1	6.0 ^y \pm 0.1	5.46
Fibre volume (ml)			
I	284 \pm 55	303 \pm 40	0.28
IIa	186 ^a \pm 20	112 ^b \pm 26	2.25
IIb	4585 ^x \pm 106	5600 ^y \pm 155	5.40
Oxidative	737 \pm 45	822 \pm 44	1.36
Nonoxidative	4317 ^x \pm 98	5199 ^y \pm 115	5.85
Capillaries/sq mm	142 ^a \pm 8	115 ^b \pm 7	2.62

^{a,b}differ at P<0.05; ^{x,y}differ at P<0.01

Salbutamol stimulated weight gain and protein accretion in growing pigs. Increased muscle mass was probably due to an increase in the volume of IIb fibres with a reduction in IIa fibre volume. Relative capillary supply was reduced to the *longissimus dorsi* muscle and this may compromise muscle function during stress.

References

- ANDERSON, P. (1975). *Acta Physiologica Scandinavica*. **95**:203-205.
 BROOKE, H. and KAISER, K. (1970). *Archives of Neurology*. **23**:369-379.
 NOVIKOFF, A.B., SHIN, W. and DRUKNER, J. (1961). *Journal of Biophysical and Biochemical Cytology*. **9**:47-61.

THE EFFECT OF EXOGENOUS GROWTH HORMONE ADMINISTRATION ON PIG MEAT QUALITY

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Administration of exogenous porcine growth hormone (PGH) has been shown to increase protein deposition and result in improved growth performance and reduced carcass fatness (Beerman, 1987). However there is limited information on the effects of PGH on pig meat quality. This experiment investigated the effects of daily PGH administration and energy intake on the meat quality of finisher pigs.

Eighty pigs (40 boars and 40 gilts) were allocated at 60 kg live weight among 8 treatments in a 4x2 factorial randomised block experiment. The factors were; PGH administration (0 and 90 $\mu\text{g}/\text{kg}$ live weight/day) and four levels of digestible energy (DE) intake (28.5, 33.0, 37.5 MJ DE/day and *ad libitum*). PGH was administered daily by injection and control pigs received an equivalent volume of buffer solution. Pigs were slaughtered at 90 kg live weight. Immediately after slaughter, carcasses were transported from the abattoir to the meat laboratory and chilled at 4°C. At 24 h post-slaughter, fat depth at the P₂ site (P₂), pH and colour attributes (L= lightness/darkness, a= redness/greenness, b= yellowness/greenness) of the cut surface of the *M. longissimus dorsi* (LD) were recorded. Samples of the LD (11-13th rib) were used to determine drip loss over 24 h (DL), cooking loss (CL) and Warner-Bratzler peak shear force (W-B).

Table 1. Effect of exogenous growth hormone (PGH) administration on meat and carcass quality measurements

	pH	L	a	b	W-B (kg)	CL (%)	DL (%)	P ₂ (mm)
Control	5.40	50.5 ^c	5.5	3.8	4.6	32.6 ^c	2.4	18.0 ^x
PGH	5.41	48.9 ^d	5.4	3.3	4.8	33.1 ^d	2.1	13.2 ^y
SED	0.023	0.69	0.21	0.30	0.21	0.27	0.28	0.61

^{c,d}differ at P<0.08; ^{x,y}differ at P<0.001

Energy intake did not significantly affect any meat quality measurements. The meat from pigs treated with PGH had a greater (P<0.08) cooking loss and was darker (L-value) (P<0.08) in colour than meat from control pigs (Table 1). However, these effects were minor and PGH administration did not significantly affect drip loss or Warner-Bratzler peak shear force. These results are in agreement with Beerman (1987) who also reported a small change in colour (slightly darker and less red) with PGH treatment but he found no difference in cooking loss. Although Solomon *et al.* (1988) reported that PGH treated pigs produced tougher meat, they postulated that muscle cold-shortening in the leaner PGH-treated pigs may have contributed to the difference. In conclusion, the results of this experiment confirm work by Beerman (1987), that PGH treatment of pigs does not adversely affect pork quality.

References

- BEERMAN, D.H. (1987). *National Swine Improvement Federation Proceedings*. 12:1-22.
 SOLOMON, M.B., CAMPBELL, R.G., STEELE, N.C., CAPERNA, T.J. and McMURTRY, J.P. (1988). *Journal of Animal Science*. 66:3279-3284.

EFFECT OF PORCINE SOMATOTROPIN ON NON-ESTERIFIED FATTY ACID AND GLYCEROL KINETICS IN GROWING BARROWS

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Treatment of growing pigs with porcine somatotropin (pST) decreases lipid accretion by either increasing lipolysis and/or decreasing lipid synthesis. The end products of lipolysis are non-esterified fatty acids (NEFA) and glycerol, the whole body kinetics of which reflect adipose tissue lipid mobilization and breakdown, respectively. The aim of this study was to determine the contribution of lipolysis and NEFA mobilization to the decreased lipid accretion in pST treated growing pigs.

Eight barrows (initial BW, 70 kg) fed six times/day were injected at 1800 h with 120 $\mu\text{g/kg/day}$ pST ($n=4$) or excipient ($n=4$) for 11 days. Frequent blood samples for measurement of diurnal changes in metabolite concentrations were taken on days -1, 1, 2 and 7. At 0900 h on day 8 a simultaneous infusion of [9,10(n)- ^3H]-oleic acid (0.5 $\mu\text{Ci/min}$) and [2- ^3H]-glycerol (1.2 $\mu\text{Ci/min}$) was given for 7 h. An insulin infusion (6 mU/kg/min) and a variable dextrose infusion to maintain glycaemia were given over the final 3.5 h. Arterial blood samples were collected between 1.5 and 3.5 h (basal) and 5 and 7 h (insulin).

Table 1. Plasma non-esterified fatty acid (NEFA) and glycerol concentrations and irreversible loss rates (ILR) in growing swine

	Basal		Insulin		SE
	Control	pST	Control	pST	
Plasma NEFA ($\mu\text{mol/l}$)	44.6	64.3	41.6	55.8	4.8
NEFA ILR ($\mu\text{mol/kg/min}$)	1.3	1.6	1.1	1.3	0.10
Plasma glycerol ($\mu\text{mol/l}$)	8.6	9.5	8.0	8.7	0.53
Glycerol ILR ($\mu\text{mol/kg/min}$)	0.67	0.71	0.64	0.69	0.05

Plasma NEFA and glycerol concentrations and their respective ILR were very low and were not different between treatment groups. Not surprisingly, there was no discernible antilipolytic action of insulin. Plasma NEFA and glycerol concentrations were positively correlated with their respective ILR ($r=0.91$ and 0.90 , respectively). Daily profiles demonstrated that plasma NEFA concentrations were in steady state during the period when kinetic measurements were made (15-22 h post-injection). However, in the pST treated pigs plasma NEFA concentrations began to moderately rise about 4-5 h post-injection, remaining elevated for approximately 8 h. This increase in plasma NEFA concentrations was augmented by time on treatment suggesting some chronic homeorhetic adjustment. As plasma NEFA concentrations were highly correlated with NEFA ILR, daily fat mobilization can be estimated from the area under the plasma NEFA curve. Plasma NEFA area was greater ($P<0.01$) with pST treatment and this difference predicted an increase in NEFA ILR of about 40 g/day. We have observed that lipid accretion rates are reduced by 210 g/day in similar pigs treated with the same dose of pST between 45-100 kg (Dunshea, unpublished data). Therefore, the major change in lipid metabolism during pST treatment must be decreased lipid synthesis.

MEAT QUALITY OF PIGS FED CIMATEROL

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The β -adrenergic agonist, cimaterol, significantly increases muscle and reduces fat deposition in sheep, cattle and pigs (Ricks *et al.*, 1984). However, there are few reports of its effects on meat quality in pigs (reviewed by Merkel, 1988).

The effects of cimaterol on meat quality characteristics of three muscles of boars and gilts were studied. Four pigs were allocated to each of the five dietary treatments (C1 - control diet fed *ad libitum*, T0.5 - cimaterol diet {0.5 ppm; fed *ad libitum*}, C2 - control diet {paired intake to T0.5}, T1 - cimaterol diet {1 ppm; fed *ad libitum*}, C3 - control diet {paired intake to T1}) at 53 kg live weight. They were fed a 13 MJ digestible energy diet and slaughtered at 92 kg live weight; cimaterol was withdrawn three days prior to slaughter.

Data on the growth, feed conversion and dietary digestibility have been presented by Williams *et al.* (1989). The results of carcass and meat quality attributes are shown in Table 1.

Table 1. Carcass and meat quality attributes of pigs fed cimaterol

Variable		C1	T0.5	C2	T1	C3	SED ¹	
Carcass weight (kg)		63.5	63.6	63.0	63.0	61.6	1.40	
P ₂ fat thickness (mm)		15.6 ^{ab}	15.7 ^{ab}	17.4 ^a	13.9 ^b	15.9 ^{ab}	1.08	
Ultimate pH	LD	5.9	6.0	5.9	6.0	5.9	0.12	
	SM	6.0	6.1	6.0	6.1	6.0	0.13	
WBPF (kg)	ST	6.4	6.5	6.4	6.5	6.4	0.14	
	LD	5.5	6.1	5.2	6.1	4.5	0.80	
	SM	5.8	5.9	5.4	4.7	6.0	0.80	
	ST	2.4	2.5	2.4	2.4	2.5	0.30	
Instron SM Compression (kg)		1.3	1.4	1.3	1.3	1.4	0.13	
Lightness of lean (L)								
	(LD)	L	40.8	41.7	44.5	41.0	41.7	2.1
	(ST)	L	42.6	40.7	42.8	41.7	43.3	2.6

¹ = P < 0.05; ^{a,b} differ at P < 0.05; LD = *longissimus dorsi*; SM = *semimembranosus*; ST = *semitendinosus*; WBPF = Warner Bratzler Peak Force; C1 = *ad libitum*; C2 = pair fed to T0.5; C3 = pair fed to T1

In conclusion subcutaneous fat thickness was decreased but meat quality was unaffected by cimaterol feeding.

References

- RICKS, C.A., BAKER, P.K., DALRYMPLE, R.H. (1984). *Proceedings of Reciprocal Meat Conference*. 37:5-11.
- MERKEL, R.A. (1988). *Proceedings of Reciprocal Meat Conference*. 41:101-111.
- WILLIAMS, K.C., NEILL, A.R., PETERS, R.T. and THORNTON, R.F. (1989). In "Manipulating Pig Production II" p. 189, eds. J.L. Barnett and D.P. Hennessy (Australasian Pig Science Association: Werribee, Victoria, Australia).

SOW LACTATION

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Introduction

Successful reproduction in animals requires an adequate supply of food for the young to grow and develop into adults. In many species of vertebrates, the adults can thrive in environments which lack suitable foods for their young. Reproduction is characterized by these animals either being confined to ecologically diverse environments or migrating to special breeding grounds. In contrast, mammals nourish their young with milk which is synthesized from the products of maternal digestion. Furthermore, because mammals have the ability to deplete their body reserves to supply the precursors for milk synthesis, a temporary interruption to the food intake of the mother does not interrupt the supply of food to her young. Thus, the evolution of lactation has conferred on mammals the ability to successfully reproduce wherever the adults can thrive (Pond, 1984).

The intensive management of domesticated mammals was introduced during the early part of this century as a means of improving meat production to cope with the demands of the ever increasing human population. Since then, intensive animal production has grown into an industry that is highly specialized, very competitive and dependent upon advances in technology and scientific research. Intensive husbandry involves keeping most aspects of the life of domesticated mammals under strict control, including their hygiene, nutrition, breeding, reproduction and general environment.

Lactation is the final phase of the reproductive cycle in mammals and in most species maternal milk is essential for the survival of the young during early postnatal life. There are large differences in the maturity of mammals at birth, and the milk of one species generally is not suited to the physiological requirements for the optimal development of the young of another species. A great deal of research has been carried out on lactation in dairy species such as the cow and the goat, as well as in laboratory animals such as the rat, whereas much less is known about lactation in the pig. Nevertheless, recent research has demonstrated that milk is a very complex secretion with the potential to have both unique nutritional and other subtle influences on the development of the young.

It has been demonstrated in one or more of these species that milk:

- (1) provides the young with a source of nourishment uniquely adapted to its digestive and metabolic requirements;
- (2) provides the young with protection against pathogenic micro-organisms;
- (3) suppresses inflammatory reactions in the gastrointestinal tract of the young;
- (4) supplements the digestive enzymes of the young;
- (5) has the potential to stimulate cell division and differentiation in the young;
- (6) may exercise a degree of control over the metabolism of the young;
- (7) may modulate the endocrine system of the young;
- (8) may contain compounds which have the potential to influence the behaviour of the young.

These nutritional, protective and developmental aspects of lactation must be considered in the effective management of intensive pig production. In total, the biochemistry and physiology of lactation is concerned with the development of the mammary gland from the foetal stages to sexual maturity, together with mammary

growth during pregnancy (mammogenesis), the initiation of milk secretion (lactogenesis), the maintenance of milk secretion (lactation) and weaning (involution).

Anatomy and histology

The mammary glands of the sow extend from the anterior (pectoral) to the posterior (inguinal) regions of the abdominal wall in two parallel rows, with each row divided by the ventral median line (Turner, 1952). Sows have up to 10 pairs of mammary glands and teats, with additional teats being classified either as supernumerary, rectal or inverted (Turner, 1952; Pond and Houpt, 1978). There are two duct openings (streak canals) to each teat and each duct opening is connected to a separate glandular system by lactiferous ducts. In contrast to ruminants, the teats of the fully developed mammary glands of the sow contain only very small cisternae (Turner, 1952).

As with other mammals the mammary glands of the sow are composed of a large number of sphere-shaped alveoli which are drained by an interconnected ductal system and encapsulated with connective tissue to form lobules (Patton and Jensen, 1976). These alveoli contain a mono-layer of epithelial cells positioned around a hollow lumen. During lactation the epithelial cells synthesize the major components of milk and discharge their contents into the lumina of the alveoli. Myoepithelial cells are situated longitudinally above the stromal surface of the epithelial cells (Cross *et al.*, 1958) and function as contractile units for the ejection of milk from the lumina of the alveoli into the lactiferous ducts. A dense arterio-venous capillary system is located on the exterior surface of each alveolus and this system supplies blood to the epithelial and myoepithelial cells (Patton and Jensen, 1976).

Blood is supplied to the anterior mammary glands from branches of the right and left brachial arteries, while a branch of the abdominal aorta supplies the posterior mammary glands (Turner, 1952). Anterior and posterior mammary veins provide two separate routes of venous return from the mammary glands to the heart. The complex lymphatic system which drains the mammary glands of the sow has been extensively reviewed (Turner, 1952; Pond and Houpt, 1978).

Mammogenesis

Development of the mammary glands during the growth of the pig foetus has been well reviewed by Turner (1952), Marrable (1971) and Hartmann *et al.* (1984a). Mammary tissue originates embryologically from the ectoderm with the appearance of two parallel mammary lines first noticeable by day 23 of gestation. Fragmentation of the mammary lines leads to the formation of small separate nodules of ectodermal cells known as the mammary buds and can be observed in the embryo at about day 28 of gestation. A proliferation of mesenchymal tissue surrounding these mammary buds results in the development of teats by about day 40 of gestation. Further cell proliferation leads to the formation of the primary mammary sprouts at about day 55 of gestation and these primary sprouts eventually differentiate into the duct openings and cisternae of the glands. Secondary mammary sprouts, which become the main milk ducts of the glands, appear at around day 85 of gestation.

The mammary glands of the newborn piglet have a duct system that is poorly developed and are largely composed of stromal (connective) tissue (Hughes and Varley, 1980). The glands remain quiescent until puberty at 5-6 months of age (Turner, 1952). With the onset of ovarian activity there is a more rapid development of the duct system, particularly around the gland cisternae. By the time the gilt is mated (8 months of age) the mammary glands consist of an extensive duct system with various "bud-like outgrowths" (Turner, 1952). Developmental changes within the mammary glands of

gilts throughout the oestrous cycle have not been well established.

Throughout pregnancy (approximately 115 days), the mammary glands undergo major histological changes as the adipose and stromal tissues are replaced by lobulo-alveolar epithelial tissue to become the milk secretory apparatus. Changes in the content of total DNA and RNA in the mammary glands of gilts indicated that there was little tissue growth during the first half of pregnancy (Hacker and Hill, 1972; Kensinger *et al.*, 1982). Major development of the lobulo-alveolar system occurred between 75-90 days of gestation and corresponded to a period of rapid tissue growth. During this period there was an increase in the number of alveoli and lobules, a large increase in the total DNA and RNA associated with the epithelial cells and a major reduction in adipose and stromal tissue (Hacker and Hill, 1972; Kensinger *et al.*, 1982). The total DNA associated with the epithelial cells was almost maximal by day 90 of gestation, indicating the completion of mammogenesis (Kensinger *et al.*, 1982).

Information regarding the precise hormonal regulation of mammary growth in sows during pregnancy is scanty. However, studies of mammary gland growth in ovariectomized-hypophysectomized-adrenalectomized rats and mice (Lyons, 1958) have shown that mammary duct growth was brought about by a combination of oestrogen, growth hormone and corticosteroids, while the proliferation of alveoli required the further presence of progesterone and prolactin (Cowie *et al.*, 1980). It has been observed that the development of the lobulo-alveolar epithelial tissue in the mammary glands of gilts was delayed by 3 weeks if an ovariectomy was performed during pregnancy (Ellicott and Dzuik, 1973; Buttle, 1988). Moreover, if only the corpora lutea were removed then the development of the lobulo-alveolar tissue continued as normal (Buttle, 1988). These studies suggested that either the ovaries of the sow, or an "ovarian factor", was important for the stimulation of mammary growth during the second half of pregnancy.

Lactogenesis

The initiation of lactation in a number of species (cows, goats and rats) has been divided into two stages (Hartmann, 1973; Fleet *et al.*, 1975; Nicholas and Hartmann, 1981). The first stage (lactogenesis I) is characterized by a gradual accumulation of pre-colostrum in the mammary glands towards the end of pregnancy. The second stage (lactogenesis II) is associated with the onset of copious milk secretion at about the time of parturition.

There also is evidence of a two stage initiation of lactation in the sow. A gradual accumulation of colostrum in the lumina of alveoli has been observed in the mammary glands of gilts between day 90 and day 105 of gestation (Kensinger *et al.*, 1982). This coincided with an increase in the DNA:RNA ratio (a measure of synthetic activity in the epithelial cells) after day 90 of gestation and indicated the histological transition from non-lactating to lactating mammary tissue. Thus, it has been proposed by Kensinger *et al.* (1986) that lactogenesis I in the gilt (or sow) occurred between day 90 and 105 of gestation and was associated with the "initiation of structural and metabolic differentiation" within the mammary glands.

Towards the end of pregnancy the mammary glands of the sow become visibly engorged with colostrum. At this stage the alveoli become distended with secretory products and fat globules accumulate at the apical end of the epithelial cells (Cross *et al.*, 1958). Small amounts of a clear, serous-coloured pre-colostrum could be expressed from the teats of some sows up to 2-3 days before parturition (Willcox *et al.*, 1983). However, it was not until the day of farrowing that copious amounts of a yellow, viscous colostrum were available.

Some workers have assessed lactogenesis II in the sow as the earliest time that milk can be manually expressed from the teats during the perinatal period (Nara and First, 1981; Nara *et al.*, 1982). Others have used the abrupt increase in the concent-

ration of lactose in post-partum mammary secretion as a measure of lactogenesis II in the sow (Martin *et al.*, 1978; Gooneratne *et al.*, 1979). A study by Willcox *et al.* (1983) has shown that in some sows mammary secretion can be expressed from the glands from 2-3 days before parturition, whereas the concentration of lactose in the same animals did not begin to rise until after the piglets were delivered. It was pointed out in this study that the expression of mammary secretion may not provide an accurate measure of lactogenesis II in the pig because the onset of lactation was rapid and varied considerably between sows.

Hartmann *et al.* (1984b) have proposed that the changes in the concentration of lactose in the blood may be used as a measure of the time of lactogenesis II in sows. They observed that the concentration of lactose in the blood of the sow was low (3-4 μM) until day 107 of gestation, whereupon there was a gradual increase up to about 35 μM by day 1 pre-partum. Thereafter, the concentration of blood lactose increased rapidly to reach maximal values of about 260 μM by 6 h post-partum and this second rise in blood lactose coincided with the increase in the concentration of lactose in the mammary secretion. Thus it seems that the changes in blood lactose during late pregnancy may reflect the increased synthesis of lactose by the mammary epithelial cells and therefore the start of lactogenesis II.

The removal of secretory products from the alveoli with the onset of sucking by the piglets resulted in the epithelial cells assuming a columnar shape (Cross *et al.*, 1958). An absence of fat globules at the apical end of the epithelial cells suggested that the secretion of milk by the alveoli was linked to the high synthetic activity of the epithelial cells (Kensinger *et al.*, 1982; Kensinger *et al.*, 1986). The consistency and composition of the mammary secretion changed considerably during the first days of lactation when there was a rapid transition from colostrum to mature milk (Willcox *et al.*, 1983). By day 4 post-partum most of the adipose and stromal tissue in the sow mammary gland had been replaced by secretory tissue that was characteristic of the normal lactating gland (Cross *et al.*, 1958; Kensinger *et al.*, 1982).

Hormonal control

Prolactin plays a major role in the onset of lactation in the sow (Cowie *et al.*, 1980). The concentration of prolactin in the peripheral blood of the pregnant sow was reported to be below 25 $\mu\text{g/l}$ until approximately 2-3 days before parturition (Dusza and Krymowska, 1981; Vale and Wagner, 1981), and then rose steadily prior to farrowing. This pre-partum rise in prolactin paralleled the rise in the concentration of relaxin and occurred at a time when the concentration of oestradiol was high (Taverne *et al.*, 1982; Kendall *et al.*, 1982). The peak concentration of prolactin occurred at about the time of the late gestational decline in the concentration of progesterone (Taverne *et al.*, 1979; Kendall *et al.*, 1982). On the day of farrowing the concentration of prolactin varied between 100 and 150 $\mu\text{g/l}$, and then declined to around 40-50 $\mu\text{g/l}$ by days 5-7 of lactation (Dusza and Krymowska, 1981; Vale and Wagner, 1981).

The ingestion of ergot by sows prevented mammary gland development and lactation (Nordskog and Clark, 1945). It has become clear that ergot alkaloids such as bromocryptine specifically inhibit the synthesis of prolactin in the sow. When bromocryptine was fed to sows during late pregnancy the concentration of prolactin declined to less than 2 $\mu\text{g/l}$ and the onset of lactation was delayed (Taverne *et al.*, 1982). The suppression of prolactin during late pregnancy failed to delay the birth of the piglets. Therefore, it was concluded that the pre-partum rise in the concentration of prolactin in the blood was not essential for the initiation of parturition in the sow (Taverne *et al.*, 1982).

The corpus luteum is the main source of relaxin during late pregnancy in the sow (Hisaw and Zarrow, 1948). The concentration of relaxin in blood increased from low levels (< 20 $\mu\text{g/l}$) at 4-5 days to reach peak levels (80-100 $\mu\text{g/l}$) at 12 h before farrowing

(Sherwood *et al.*, 1975; Taverne *et al.*, 1982). Furthermore the administration of relaxin for a prolonged period throughout late pregnancy resulted in a reduction of lactational performance in the sow and an increased mortality rate of piglets between birth and weaning (Kertiles and Anderson, 1979).

The concentration of progesterone was high during late gestation and then declined to less than 4 $\mu\text{g/l}$ at parturition (Cowie *et al.*, 1980). At this time there was a significant negative correlation between the concentration of progesterone in blood and the concentration of lactose in the colostrum (Martin *et al.*, 1978). Treating sows with progesterone during late pregnancy not only delayed the time of parturition but also delayed lactogenesis II so that milk still appeared on the day of farrowing (Gooneratne *et al.*, 1979). However, the administration of progesterone to late-pregnant sows did not alter the time of the late gestational rise in the concentrations of prolactin and relaxin (Taverne *et al.*, 1982). Although prolactin and relaxin are involved in the initiation of lactation in the sow, it seems that as in other mammals (Cowie *et al.*, 1980) the withdrawal of progesterone during late pregnancy is the trigger for the initiation of lactogenesis II.

Lactation

Composition of colostrum and milk

The gross composition of sow colostrum and milk has been previously reviewed (Braude *et al.*, 1947; Perrin, 1955; Bowland, 1966; Aumaitre and Seve, 1978; Pond and Houpt, 1978; Klobasa *et al.*, 1987). Early colostrum has a high total solids and protein content but a comparatively low concentration of carbohydrate (lactose) and fat. The transition from colostrum to milk during the first 2-3 days of lactation is characterized by a sharp decline in total solids and protein and a simultaneous increase in the concentration of lactose and fat. Protein, lactose and fat contribute approximately 18%, 22%, and 60%, respectively, of the total energy content (5.2 kJ/g) of sows' milk (Hartmann *et al.*, 1984a; Oftedal, 1984). In contrast to many other species, the ash content (salts and minerals) is lower in sow colostrum than in the milk secreted during established lactation (Perrin, 1955).

Variations in the gross composition of sows' milk also have been attributed to differences between breeds, differences among individuals of a breed, nutrition, environmental conditions and infection of the mammary glands (Fahmy, 1972; Pond and Houpt, 1978; Gooneratne *et al.*, 1982). These variations in gross composition are most likely a consequence of differing rates of synthesis and secretion of the milk components, as only small variations have been found in the gross composition of milk expressed from different mammary glands of the same sow, provided that the glands were being actively suckled (Pond *et al.*, 1962; Martin *et al.*, 1978).

Protein

Although the mammary glands of the sow synthesize and secrete a large quantity of the milk proteins, there are some proteins such as serum albumin which are transported from the blood without modification (Larson and Jorgensen, 1974). It has been shown by Linzell *et al.* (1969) that all of the essential amino acids, and some of the non-essential amino acids, present in the milk proteins which were synthesized *de novo* by the mammary gland, originated from the corresponding amino acids in the blood supply. Overall, the proteins of sow colostrum had a higher content of threonine, valine, phenylalanine and leucine and a lower content of methionine and lysine than the proteins in mature milk (Beacom and Bowland, 1951).

Milk proteins have been broadly classified into two classes - the "caseins", which are defined as the proteins precipitated from milk at pH 4.0-5.0, and the more soluble "whey proteins" which remain after acid precipitation of the caseins (Jenness, 1985).

The caseins are a group of phosphoproteins which in association with calcium, inorganic phosphate, magnesium and citrate form stable micellar structures in milk (Lyster, 1972; Jenness, 1974). In the sow the percentage of total protein represented by the caseins is much lower in colostrum as compared to milk (Figure 1). The primary function of the caseins in sows' milk is to provide the piglets with a source of amino acids. Furthermore, it is known that the neonates of other species receive a greater amount of calcium and phosphate from the casein micelles than from the soluble fraction of these salts in milk (Jenness, 1974).

Only the α_2 -, β - and k - casein sub-units have been isolated from sows' milk (Woychik and Wondolowski, 1969). Porcine α_2 -casein seems to be a homogeneous protein (M_r 24,500) with 15-16 phosphates/molecule and a high lysine and low proline content (Cerning-Beroard, 1984). Porcine β -casein (M_r 24,900) has a higher phosphorous content (eight phosphates/molecule) than the β -caseins examined from other species (Mulvihill and Fox, 1979; Jenness, 1985). Porcine k -casein (M_r 30,000) is highly glycosylated and has an amino acid content similar to bovine k -casein (Cerning-Beroard and Zevaco, 1984).

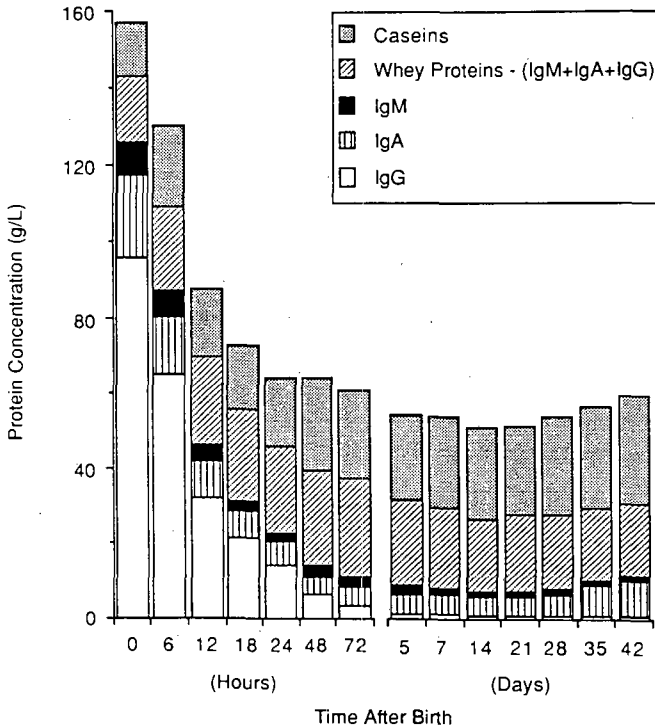


Figure 1. Changes in the concentration of proteins of sows' milk during a 42 day lactation (modified from Klobasa *et al.*, 1987).

In bovine milk the k -casein has the important property of stabilising the casein micelles (Jenness, 1974). Upon hydrolysis of k -casein by the enzyme chymosin (rennin) in the digestive tract of the calf, the stability of the casein micelle is destroyed and a firm curd is formed. The products arising from bovine k -casein hydrolysis were a para- k -casein and caseinomacropeptide (Jenness, 1985). The complete amino acid sequence has been determined for the caseinomacropeptide derived from k -casein of sows' milk (Chobert *et al.*, 1976). Since piglets suckle at frequent intervals the formation of a firm casein curd in their stomachs would retard the digestion and

absorption of milk nutrients. However, only a fine precipitate, which is difficult to sediment, is formed after the chemical precipitation of the proteins from sows' milk (Hartmann *et al.*, 1984a). From this consideration it seems that the casein curd that forms in the stomach of piglets may be softer than the curd that forms from cows' milk.

Recent studies have shown that the partial enzymic digestion of milk caseins resulted in the release of short peptides with various biological activities (Migliore-Samour and Jolles, 1988). These peptides are located in protected regions of the casein molecule and only become active after being cleaved from casein by the proteolytic enzymes in the digestive tract of the newborn. Some of the peptides, such as β -casomorphin from bovine β -casein (Brantl *et al.*, 1979) and human β -casein (Brantl, 1984) and exorphins from bovine α -casein (Zioudrou *et al.*, 1979), exhibited activities similar to that of the endogenous opioid peptides and have now been implicated as immunomodulators in the newborn (Migliore-Samour and Jolles, 1988). Furthermore, Umbach *et al.* (1985) demonstrated the presence of β -casomorphin immunoreactive material in the blood of newborn calves after their first milk ingestion and suggested that the casein-derived opioid peptides may pass into the circulation of the newborn and either elicit effects at various target organs (e.g. central nervous system) or assist the newborn in adapting to environmental stressors. It is quite conceivable that biologically active peptides derived from caseins also may be present in sows' milk.

Proteins associated with the fat globule membranes have been classified with the casein fraction as the fat globules precipitate together with the caseins at pH 4.0-5.0 (Jenness, 1985). The membrane of the fat globule in bovine milk has been studied intensively and shown to consist of 25-60% (w/w) protein, which included various lipoproteins, glycoproteins and enzymes (Patton and Keenan, 1975). There is some evidence that the fat globule membrane in sows' milk may be a target for the adhesion of bacteria such as *E. coli* (Atroshi *et al.*, 1983). However, the involvement of this adhesion in the development of piglet diarrhoea remains unresolved.

The principal whey proteins in sows' milk are the immunoglobulins, albumin, β -lactoglobulin and α -lactalbumin. The concentration of the total whey protein declined by almost 70% over the first day and reached minimal concentrations by the second week of lactation (Figure 1). Whey protein contributed about 90% of the high concentration of total protein in colostrum at parturition, but only accounted for about 60% of the total protein in milk secreted from 5-42 days of lactation.

About 90% of the whey protein in sow colostrum at parturition was accounted for by the immunoglobulins (Figure 1). The immunoglobulins in early colostrum consisted of 76% IgG (IgG₁ and IgG₂ subclasses), 17% IgA and 7% IgM (Klobasa *et al.*, 1987). Most of the immunoglobulins in colostrum are derived from the maternal serum, including all IgG, more than 80% IgM and 40% IgA (Bourne and Curtis, 1973). This has been supported by the finding that there are greater numbers of IgA secreting cells in the mammary glands of the sow during late pregnancy than IgG and IgM secreting cells (Brown *et al.*, 1975). The IgG, IgM and IgA isotypes have been found to be important for the transfer of passive immunity to the newborn piglets (Hartmann *et al.*, 1989).

The concentrations of all the colostrum immunoglobulins declined rapidly during the first 24 h after parturition (Figure 1). However, towards the end of lactation the predominant immunoglobulin secreted in mature milk was IgA (78%), whereas IgG, the major immunoglobulin in colostrum, and IgM were only 7% and 15% of the total immunoglobulin concentration, respectively (Klobasa *et al.*, 1987). In contrast to colostrum, it was found that more than 90% of IgA and IgM and almost 70% of IgG in milk were synthesized locally in the mammary glands (Bourne and Curtis, 1973). Indeed, the number of cells secreting these three isotypes of immunoglobulins in the mammary glands increased with lactation but at all times there was a much greater proportion of IgA than either IgG or IgM secreting cells (Brown *et al.*, 1975).

Albumin accounted for most of the non-immunoglobulin protein in the whey of early colostrum but contributed a much smaller proportion in the whey of milk after days 2-3 of lactation (Klobasa *et al.*, 1987). It was established by Carlsson *et al.* (1977) that the albumin in sow colostrum (M_r 65,000) originated from the maternal blood and was absorbed from the small intestine into the circulation of the newborn piglets. Therefore, during the early postnatal period the intestinal transport of fatty acids and amino acids may be facilitated by the ingestion of maternal albumin via the colostrum (Aumaitre and Seve, 1978).

Although β -lactoglobulin is the most abundant whey protein in sows' milk, the biological role of this protein is unknown (Kessler and Brew, 1970; Bell *et al.*, 1981). In contrast to the dimeric ruminant β -lactoglobulin, porcine β -lactoglobulin (M_r 18,500) is a monomeric protein without free sulphhydryl groups (Kessler and Brew, 1970). Unlike β -lactoglobulin, the biological role of α -lactalbumin has been determined. This protein was found to be a sub-unit (B protein) of lactose synthase, the enzyme catalysing the biosynthesis of lactose in the mammary glands (Ebner and Brodbeck, 1968; Brew, 1969). It acts as a "modifier" protein for the enzyme galactosyltransferase, the A protein sub-unit of lactose synthase, and promotes a change in substrate specificity of the transferase required for the synthesis of lactose. The α -lactalbumin isolated from sows' milk (M_r 14,500) has similar chemical and physical properties to that of other species (Schmidt and Ebner, 1971). A porcine pre- α -lactalbumin with an amino acid terminal extension of 19 amino acids also acts as a modifier protein for the galactosyltransferase (Raymond *et al.*, 1982).

There are a variety of minor proteins in the whey of sows' milk. Lactoferrin, an iron-binding protein which non-specifically inhibits bacterial growth, is secreted by the mammary glands of the sow. It was found in high concentrations (1.1-1.3 g/l) in early colostrum and declined rapidly during the first week of lactation (Elliot *et al.*, 1984). A large variation in the concentration of lactoferrin was noted in the milk taken from different sows, although the intermammary variation from the same sow remained low. Transferrin, the iron-binding protein in blood, and binding proteins for vitamins such as folate and vitamin B₁₂ and hormones such as the corticosteroids, have been found in the milk of several species, including the sow (Jenness, 1985).

It has been proposed that many of the enzymes in milk originally participated in the metabolic and biosynthetic reactions of the mammary epithelial cells and were most likely transferred into the milk by a process of cell necrosis (Shahani, 1966; Shahani *et al.*, 1980). Whereas these enzymes have no biological role in milk, it is possible that they may reflect the metabolic changes of the mammary glands during lactation. Indeed, the activities of lactate dehydrogenase and malate dehydrogenase, along with their isoenzymes, were found to be characteristic for the milk secreted at different stages of lactation in the sow (Kjellberg and Karlsson, 1967). Furthermore, Grigor and Hartmann (1985) observed that there was considerable variation in the absolute activities of certain NADP-linked dehydrogenases in sows' milk. However, when these authors expressed the activities relative to that of either lactate dehydrogenase or cytoplasmic malate dehydrogenase there was a close relationship between the activities in milk and those reported for the homogenates of sow mammary tissue.

Some of the milk enzymes are beneficial to neonatal development, particularly during the early postnatal period, and it was suggested that these enzymes may be secreted directly into the milk (Shahani *et al.*, 1980). Together with lactoferrin, the enzymes lactoperoxidase, lysozyme (N-acetylmuramylhydrolase) and xanthine oxidase provide the newborn with non-specific protection against enteropathogens (Reiter, 1985). Digestive enzymes such as α -amylase, lipase, esterase, protease and alkaline phosphatase may assist the newborn with their digestion and assimilation of milk components. Protease inhibitors, such as sow colostrum inhibitor and the serum type inhibitors, were at high concentrations in early colostrum but declined to very low

concentrations in the milk secreted during the first days of lactation (Laskowski *et al.*, 1957; Jensen and Pedersen, 1979; Westrom *et al.*, 1982). The protease inhibitors in sow colostrum have been shown to improve the transfer of intact proteins, such as the immunoglobulins, into the blood of the newborn piglet by inhibiting proteolysis in the digestive tract (Carlsson *et al.*, 1980; Westrom *et al.*, 1985). They also may provide the mammary gland with protection against leucocytic and lysosomal proteases that arise either from tissue development during lactogenesis, or infective mastitis (Hamosh *et al.*, 1985).

The presence of the lysosomal enzyme N-acetyl- β -D-glucosaminidase (NAGase) in milk has been used as an indicator of mastitis in the cow and ewe (Mattila and Sandholm, 1985; Maisi *et al.*, 1987). Damage to the mammary epithelial cells during mastitis resulted in an increase of NAGase in the milk of these species. Unfortunately, the high basal levels of NAGase that were found in sow colostrum and milk has meant that the determination of this enzyme can not be used successfully for the monitoring of mastitis andagalactia in the sow (Raekallio, 1987).

Carbohydrate

The di-saccharide lactose (4-O- β -D-galactopyranosyl- α -D-glucopyranose) is the predominant carbohydrate in sows' milk and is synthesized within the Golgi apparatus of the mammary epithelial cells (Ebner and Schanbacher, 1974). Lactose is then secreted into the lumen of the alveolus, along with other milk constituents such as the caseins, α -lactalbumin, calcium, phosphate and citrate, by the exocytosis of the Golgi vesicles at the apical membrane of the epithelial cell. Blood glucose is the major precursor (70%) for the synthesis of lactose in sows' milk (Spincer and Rook, 1971). The initiation of lactation in many species, including the sow, is associated with a major surge in lactose synthesis (Kuhn *et al.*, 1980). Consequently, there is a rapid increase in the concentration of lactose in the mammary secretion during the early post-partum period. Lactose is the major osmotic constituent in milk and, with the milk salts and proteins, maintains the milk iso-osmotic with the blood plasma (Jenness, 1985). It has been proposed by Linzell and Peaker (1971a) that lactose, which is unable to permeate any cell membranes, draws water osmotically into the golgi vesicles and is, thereby, the main mechanism by which water moves into milk. Since the aqueous phase constitutes a large proportion of milk, it appears that the formation of lactose may be an important determinant of milk yield.

Lactose is readily hydrolysed to glucose and galactose by the enzyme lactase in the small intestine of newborn piglets. These monosaccharides are absorbed into the blood of the piglets and used to replenish their stores of liver and muscle glycogen (Hartmann *et al.*, 1989). Galactose is also used by the neonate in the formation of galactocerebrosides for myelination, and in glycoprotein and glycolipid synthesis. Recent evidence suggests that the presence of lactose in milk may improve the absorption of calcium in the newborn (Gaulle *et al.*, 1982).

There are various carbohydrates in milk besides lactose such as the monosaccharides, sugar phosphates, nucleotide sugars, oligosaccharides and the carbohydrates complexed with peptides and proteins (Jenness, 1985). Although some of these carbohydrates are only present in small amounts in milk, they may still have an important biological role in the neonate.

Fat

Milk fats of many species, including the sow, are composed mainly of triacylglycerols, together with smaller proportions of di- and mono-acylglycerols, phospholipids, glycolipids, cholesterol and cholesterol ester, fat soluble vitamins, and free fatty acids (Jenness, 1985). Most of the fats (>95%) in sows' milk exist in membrane-bound fat globules that were shown to be approximately 0.5-7.0 μ m in size (Whittlestone, 1952).

The remainder of the fats are present in membrane fragments and cellular debris that is extruded from the mammary tissue into the milk.

Unlike other mammals such as cows, ewes, goats and women which show an increase in concentration of fat between fore and hind milk when either suckled or milked, no such trend has been observed in the sow (Whittlestone, 1952; Perrin, 1954). However, this finding must be accepted with caution as the administration of relatively large doses of oxytocin before milking a sow may cause mixing of stored milk and obscure any real changes in the concentration of fat. Furthermore, due to the extremely short duration of milk ejection (10-20 sec) it is extremely difficult to obtain fore and hind milk samples in the sow for fat analysis.

Supplementing the diet of the sow with fat during late pregnancy and lactation increased the concentration of fat in the colostrum and milk (Pettigrew, 1981; Britt, 1986). In some experiments there was also an increase in the milk production of the sow (Pettigrew, 1981). When the sow's diet was supplemented with fat, an improvement in the survival rate of the neonatal piglets was most obvious in studies where the survival rate in the piggery was below 80% (Pettigrew, 1981).

There is a change in the composition of fatty acids in sows' milk fat during the course of lactation. Generally, there was a higher content of oleic acid ($C_{18:1}$) and linoleic acid ($C_{18:2}$), and a lower content of palmitic acid ($C_{16:0}$) and palmitoleic acid ($C_{16:1}$), in colostrum compared to mature milk (Duncan and Garton, 1966). DeMan and Bowland (1963) reported that capric acid ($C_{10:0}$) and lauric acid ($C_{12:0}$) were absent in colostrum but present in mature milk. Furthermore, they have demonstrated that the body depot fat of the sow resembled the colostrum fat more closely than the milk fat.

Fatty acids in the milk are derived from two sources; firstly, from the blood lipids that are carried in the chylomicrons and very low density lipoproteins and include both endogenous and dietary fatty acids; and secondly, from *de novo* synthesis in the mammary glands (Patton and Keenan, 1975). A comprehensive study by Linzell *et al.*, (1969) has shown that triacylglycerols were the only lipid class to be consistently removed in significant amounts from the blood supply by the mammary glands of the lactating sow. They also established that the fatty acids in milk ($C_{14:0}$ and greater) were derived mainly from the fatty acids of the blood triacylglycerols. Indeed, Spincer and Rook (1971) later reported that greater than 60% of palmitic acid and 70% of stearic acid in the fat of sows' milk could be accounted for by the mammary uptake of the corresponding fatty acids from blood triacylglycerols. Most of the fatty acids in colostrum and milk fat were found to reflect closely any changes that occurred in the fatty acid composition of the blood triacylglycerols (Witter *et al.*, 1970), which in turn were influenced by the type of fat in the feed ingested by the sow (Witter and Rook, 1970). This has been supported by a number of dietary studies. For example, the percentage of oleic acid in milk fat was increased with beef tallow (DeMan and Bowland, 1963), animal fat (Seerley *et al.*, 1978) and horse beans (Nielson and Kruse, 1974) in the feed, whereas the percentage of linoleic acid was increased with corn oil (Tollerz and Lindberg, 1965; Miller *et al.*, 1971; Kruse *et al.*, 1977) but decreased with horse beans (Nielson and Kruse, 1974) in the feed. It seems that the well-fed, lactating sow utilizes fatty acids of dietary origin for the synthesis of milk fats to a greater extent than the fatty acids originating from the body depot. However, when sows were fasted for a short-time during their lactation there was an increase of the milk C_{18} fatty acids, and a decrease of C_{14} and C_{16} fatty acids, so that the milk fat began to resemble the body depot fat (Tollerz and Lindberg, 1965).

The mammary glands of the sow were found to be capable of synthesizing milk fatty acids of chain lengths C_{18} or less, mainly from glucose, but to a lesser extent from acetate (Linzell *et al.*, 1969). They were also capable of desaturating palmitic acid and stearic acid to the corresponding 9,10-mono-unsaturated derivatives, palmitoleic acid and

oleic acid (Bickerstaffe and Annison, 1968; Spincer and Rook, 1971). This may be significant in providing the neonatal piglets with milk fat that is readily digestible. In contrast to the ruminants, very low concentrations of acetate and β -hydroxybutyrate were found in the blood of the lactating sow (Linzell *et al.*, 1969; Spincer *et al.*, 1969). This resulted in a minimal uptake of these substrates by the mammary glands and helped to explain why there was a low content of short-chain fatty acids in sows' milk.

Carnitine plays an essential role in most tissues for the transport of fatty acids across the inner membrane of the mitochondria during β -oxidation. The concentration of total carnitine declined from a high 370 μ M in colostrum to 270 μ M in milk at day 2 of lactation (Kerner *et al.*, 1984). More than 95% of the carnitine in sow colostrum and milk was acylated, with the major acylcarnitines being acetyl- and isovalerylcarnitine. However, newborn piglets contained much lower amounts of total carnitine in their blood and liver than 2-day-old piglets (Kerner *et al.*, 1984). From this observation it was proposed that sows' milk was a primary source of carnitine for the tissues of newborn piglets.

The monoacylglycerols, diacylglycerols and free fatty acids are present in the secreted milk but also may arise from lipolysis during the storage of milk either in the mammary glands or awaiting analysis (Jenness, 1985). Phospholipids constitute only a minor proportion (<1%) of the total fat content in milk. Nevertheless, they are the major structural lipids in biological membranes and are most likely required by the piglets for the development of tissues such as the brain and nervous system. Milk phospholipids originate primarily from synthesis in the mammary tissue, and up to two thirds of the phospholipids are associated with the fat globule membrane, with the remainder located in the skim milk phase (Patton and Jensen, 1976; Jenness, 1985). In sows' milk, the proportions of the principal phospholipids were phosphatidylethanolamine (36.8 moles %), phosphatidylcholine (21.6 moles %), phosphatidylserine (3.4 moles %), phosphatidylinositol (3.3 moles %) and sphingomyelin (34.9 moles %) (Morrison, 1970).

Hormones and growth factors

Many peptide and steroid hormones have been detected in milk (Koldovsky, 1980; Pope and Swinburne, 1980; Strbak, 1985). The presence of hormones in milk suggests that maternal hormones may be able to effect some endocrine control in the newborn. However, milk hormones also have proved useful for both research into maternal endocrinology during lactation and as a tool for veterinary diagnosis, particularly in domestic animals (Pope and Swinburne, 1980). In the sow, as in the sheep and dairy cow, the concentration of prolactin in the blood was similar to those in the milk and changes in blood prolactin were usually reflected in the milk (Mulloy and Malven, 1979). These results suggested that milk prolactin could be a useful indicator of the concentration of blood prolactin at different stages of lactation in the sow. Although the exact role of milk prolactin is unknown, it has been postulated that this hormone may improve the transport of fluid and electrolytes from the intestinal lumen of the neonate during the early postnatal period (Malven, 1977). The concentration of oestrone in sows' milk declined from 14 μ g/l at parturition to below 0.5 μ g/l by day 2 of lactation (Farmer *et al.*, 1987). If steroid hormones such as the oestrogens do influence the viability of piglets, then those consuming the most colostrum may be conferred a greater survival advantage (Farmer *et al.*, 1987). Other hormones reported in sow colostrum and milk include prostaglandin-like substances (Maffeo *et al.*, 1987), insulin (Slebodzinski *et al.*, 1986; Jaeger *et al.*, 1987; Westrom *et al.*, 1987) neurotensin, bombesin (Westrom *et al.*, 1987) and thyroid hormones (Slebodzinski *et al.*, 1986).

A number of polypeptide growth factors have been identified in the milk of various species. *In vitro* growth promoting activity was first characterized in sows' milk by Cera *et al.* (1987) and found to be greatest in early colostrum. Since then,

epidermal growth factor (Jaeger *et al.*, 1987) and insulin-like growth factor (Simmen *et al.*, 1988) have been detected in sows' milk. The concentration of these growth factors also was high in colostrum and rapidly decreased to low levels within the first week of lactation. The exact role of polypeptide growth factors in colostrum and milk is still uncertain, but it has been proposed by Brown and Blakely (1983) that they may mediate the growth and development of the mammary glands during lactogenesis, as well as control the growth and maturation of the intestinal epithelium of the newborn.

Salts and trace elements

The salts in milk are present in the form of ions and ion complexes, with the major cations being sodium, potassium, calcium, and magnesium, and major anions being chloride, inorganic phosphate, citrate, bicarbonate, sulphate, and proteins (Jenness, 1985). The complexities of the secretion and partitioning of the milk salts have been dealt with in great detail (Linzell and Peaker, 1971a; Holt, 1981, 1985). Sodium, potassium and chloride are normally present as free ions and contribute to the osmotic pressure of milk. Since milk is iso-osmotic with the blood plasma, it has generally been found that the concentrations of these ions vary inversely with the concentration of lactose (Linzell and Peaker, 1971a). In the sow there was a fall in the concentration of sodium in milk during the first four days of lactation which coincided with the post-partum rise in the concentration of lactose (Willcox *et al.*, 1983). As with other milks, and intracellular fluids, the concentration of potassium in sows' milk was constantly higher than the concentration of sodium. In contrast to sodium, there were no marked changes in the concentration of potassium during the first four days of lactation. The sodium, potassium and chloride ions in sows' milk are utilized by the neonatal piglet for the maintenance of electrolyte balance.

Citrate, calcium and phosphate are secreted into milk along with lactose and casein by a transcellular pathway involving the exocytosis of golgi vesicles at the apical membrane of the mammary epithelial cell (Holt, 1981). Citrate originates from the mitochondria of the mammary epithelial cell and exists in milk as either di-citrate and tri-citrate ions or complexed with calcium (calcio-citrate), magnesium (magnesiocitrate) and the casein micelles (Faulkner and Peaker, 1982). A rapid rise in the concentration of total citrate in milk has been observed during the perinatal period of the cows, goats and a woman, and this was followed by a gradual decline in the concentration over the first week of lactation (Peaker and Linzell, 1975). Our preliminary observations have shown that the concentration of citrate in sows' colostrum increased from 3-5 mM on the day before farrowing to peak concentrations of 6-9 mM by day two of lactation (Hartmann *et al.*, unpublished data). This is followed by a gradual decline during the first week of lactation, with no clear trend thereafter (Konar *et al.*, 1971). Approximately 42% of the carbon for the citrate in sows' milk originates from blood glucose (Spincer and Rook, 1971). Although citrate has no known nutritional role in milk, it does appear to be a buffer in milk for H^+ and Ca^{2+} ions, and may play a part in determining the overall structure of the casein micelles (Faulkner and Peaker, 1982). Calcium required by the neonate for bone and tooth development is adequately supplied by the milk. There are three forms of calcium found in milk; ionised calcium; calcium complexed with phosphate, citrate and other milk anions; and calcium associated with the casein micelles (Neville and Watters, 1983). The concentration of total calcium in sows' milk more than doubled during the first few days after birth, and then gradually increased with lactation (Perrin, 1955; Gooneratne *et al.*, 1983). The total phosphorous content of sows' milk also increased steadily during lactation but less markedly than calcium (Perrin, 1955). As with citrate, the phosphate ions in milk serve as a buffer for H^+ and Ca^{2+} ions.

Trace elements in milk include a large number of metals, the metalloids such as arsenic, boron, and silicon, and the halogens such as fluorine, bromine and iodine

(Jenness, 1974). Sows' milk is normally deficient in iron and copper, and the concentration of these metals cannot be improved by either dietary supplementation or injection (Pond and Houpt, 1978). In contrast, the concentration of zinc (Pond and Jones, 1964; Earle and Stevenson, 1965) and manganese (Plumlee *et al.*, 1956) in milk usually reflect their levels in the maternal diet. The iodine and selenium content of colostrum is much higher than milk, and therefore newborn piglets rarely show deficiencies in these elements if they have received adequate amounts of colostrum (Aumaitre and Seve, 1978).

Vitamins

The vitamins in sows' milk are derived both from the diet and from maternal stores in the liver and other tissues (Pond and Houpt, 1978). Thus the concentration of vitamins in sows' milk is influenced by factors such as seasonal changes (Braude *et al.*, 1947), the quantities stored in the liver and other tissues at the beginning of lactation, and the lactational diet. Generally, the concentrations of vitamins A, C, E, and total and free thiamine were higher in colostrum than mature milk, whereas the concentrations of pantothenic acid and niacin were lower, and riboflavin similar to, that of mature milk (Braude *et al.*, 1947; Pond and Houpt, 1978). In some piggeries, subclinical rickets may be a problem as the deprivation of ultraviolet light may lower the endogenous synthesis of cholecalciferol (vitamin D) in both sows and piglets. Goff *et al.* (1984) observed that the intramuscular injection of cholecalciferol into sows before parturition increased its concentration in milk and suggested that this route of administration may provide an effective method of supplementing the piglets with cholecalciferol.

Cells

Different cell types have been identified in sow colostrum and milk by Lee *et al.* (1983). Neutrophils were the major cell type in colostrum but decreased in number as lactation proceeded so that by the end of the first week epithelial cells became the predominant cell type. As the epithelial cells were viable and intact, it was proposed that they originated from abrasion to the epithelium of the mammary glands during the vigorous and frequent sucking of the piglets. Macrophages, lymphocytes and eosinophils were found in lower concentrations in both colostrum and milk. Overall, these phagocytic and lymphoid cells in colostrum and milk may help to provide protection against infection for the mammary glands of the sow and the digestive tract of the neonatal piglet (Reiter, 1978; Lee *et al.*, 1983).

Sucking behaviour

The piglets begin to develop a preference for either a "teat pair" (the right and left teats at the same anterior-posterior location) or a particular teat within the first few hours after birth (Wyeth and McBride, 1964; De Passille *et al.*, 1988). The teat order of the litter is established during the first week after birth and then rarely varies for the rest of the lactation (Fraser, 1975; Hartstock *et al.*, 1977; Jeppesen, 1982). Piglets on the anterior and posterior teats seem to settle into a teat order sooner than those piglets on the middle teats (Fraser and Thompson, 1986). Some workers have suggested that the formation of a teat order confers an advantage on the piglets by minimising fighting for teats during each sucking period (Hartstock and Graves, 1976; De Passille *et al.*, 1988).

In commercial piggeries, the piglets suckle the sow 20 times or more a day (Fraser, 1980) at mean intervals of 44.3 min, with a range of 21-92 min (Ellendorf *et al.*, 1982). A characteristic behavioral pattern is associated with the sucking of sows and involves vocalisation from the sow, together with a sequence of jostling, nuzzling, slow sucking, rapid sucking and, finally, slow sucking and nuzzling from the piglets

(Whittemore and Fraser, 1974; Fraser, 1980; Ellendorf *et al.*, 1982). The vocalisations of the sow, particularly the early slow grunting, seem to be an important means by which the sow attracts the piglets to suckle (Lewis and Hurnik, 1986). Then, as the sucking proceeds the change in the rhythm of the sow's vocalisations from slow to rapid may be used by the piglets as a cue for milk letdown (Fraser, 1980; Algers and Jensen, 1985). Observations made at the termination of sucking indicated that the piglets usually moved away from the sow unless the mammary glands remained accessible (Petherick, 1983).

Milk letdown

The mammary glands of the sow contain no cisternae to store the milk secreted by the epithelial cells of the alveoli and therefore milk cannot be passively withdrawn by the sucking piglets. Thus the removal of milk from the alveoli and ductal system of the mammary glands requires the operation of the neuro-endocrine milk ejection reflex which consists of an afferent neural pathway and an efferent pathway involving the release of oxytocin and the ejection of milk (Lincoln and Paisley, 1982). Oxytocin originates from neurones which are situated in the supra-optic and paraventricular nuclei of the hypothalamus and is released from the posterior pituitary gland in response to the activation of neural receptors within the teats of the mammary gland by the nuzzling and sucking of piglets (Cowie *et al.*, 1980; Ellendorf *et al.*, 1982). An increase in the concentration of oxytocin has been observed in samples of jugular venous blood collected from sows during the initial nuzzling phase, reaching peak values up to 30 sec before the ejection of milk from the mammary gland (Folley and Knaggs, 1966; Ellendorf *et al.*, 1982). Oxytocin stimulates the contraction of the myoepithelial cells which surround the alveolar lumen, thus forcing the milk from the alveoli, through the ductal system to the teats. Milk ejection in the sow, as indicated by a rise in intramammary pressure, occurred on average 2.4 min (range 1-6 min) after the piglets started to suckle and was associated with the period of rapid sucking (Ellendorf *et al.*, 1982). The duration of milk flow in the sow was found to be very short and lasted for only 10-20 sec (Fraser, 1980).

Acute, episodic releases of relaxin have been observed in the blood of sows, both when piglets suckled and after the administration of exogenous oxytocin (Afele *et al.*, 1979; Whitely *et al.*, 1985). It has been suggested (Whitely *et al.*, 1985) that the role of relaxin at this time may be to oppose the action of oxytocin by stimulating the relaxation of the myoepithelial cells and/or providing a negative feedback on the hypothalamus for the suppression of oxytocin secretion. The source of relaxin secretion during lactation was unclear in these studies as the corpora lutea, which are the principal source of relaxin during late pregnancy, would have regressed at term.

Whittemore and Fraser (1974) noted that a significant percentage of piglet suckings was unsuccessful and that these "incomplete sucklings" may affect both the sow's capacity to produce milk and the growth rate of the piglets. Failure of milk ejection during an incomplete sucking was indicated by an absence of rapid grunting from the sow during the sucking phase, no change in intramammary pressure and no pulsatile release of oxytocin during the nuzzling phase (Ellendorf *et al.*, 1982). Studies using purely behavioral observations found that up to 27% of suckings were unsuccessful (Fraser, 1977; Watson and Bertram, 1980), whereas data obtained using changes in intramammary pressure indicated that 21.4% of suckings were unsuccessful (Ellendorf *et al.*, 1982). It was suggested by Ellendorf and Poulain (1984) that behavioral observations alone do not provide an accurate means of investigating incomplete suckings and that either changes in intramammary pressure or other non-invasive methods must be used to detect the failure of milk ejection in the sow. Reasons provided for incomplete suckings include the resumption of sucking by the piglets during the refractory period of the milk ejection reflex and disturbances to the sow by

abnormal behaviour of the piglets, feeding times, the presence of workers and conditions of housing in the piggery sheds (Fraser, 1977).

Hormonal control

Prolactin is maintained at high concentrations in the peripheral blood of sows throughout lactation by the frequent sucking of the piglets (van Landeghem and van de Wiel, 1978; Mulloy and Malven, 1979; Bevers *et al.*, 1978). Although the concentrations of prolactin did not correlate with the number of piglets in the litter (Bevers *et al.*, 1978), as in other species there was a slight elevation in the concentration of prolactin in the sow either during or immediately after an episode of sucking (van Landeghem and van de Wiel, 1978; Kendall *et al.*, 1983; Mattioli *et al.*, 1986). A rapid decline in the concentration of prolactin in the blood of lactating sows occurred within a few hours of the removal of piglets (Bevers *et al.*, 1978; Holmes *et al.*, 1988). However, if the piglets were replaced after a separation period of 2-4 h the concentration of prolactin returned to normal (Bevers *et al.*, 1978). Fluctuations in the concentration of prolactin in blood over a 24 h period did not conform to any definite circadian rhythm in the sow and it was proposed that any underlying rhythm may be completely masked by the release of prolactin during the periods of sucking (Bevers *et al.*, 1978). The progressive decline in the concentration of prolactin in late lactation has been attributed to the tendency of piglets to suckle less frequently as they grow older (van Landeghem and van de Wiel, 1978; Mulloy and Malven, 1979; Kirkwood *et al.*, 1984).

An inhibition of ovulatory oestrus occurs throughout the first 4-6 weeks of lactation in sows and is associated with considerable suppression of follicular growth (Crighton and Lamming, 1969; Britt *et al.*, 1985). The concentrations of both luteinizing hormone (LH) and follicle-stimulating hormone (FSH) in the blood of sows are low during the first 3 weeks of lactation (Stevenson *et al.*, 1981). This inhibition of LH and FSH secretion is slowly released during long lactations and only then do sows undergo a fertile ovulation (Hughes and Varley, 1980). It seems that the concentrations of LH and FSH are kept low during early lactation so as to suppress follicular growth and prevent ovulation from occurring within a short time of farrowing (Stevenson *et al.*, 1981).

The role of hypothalamic gonadotropin-releasing hormone (GnRH) in regulating the secretion of LH and FSH by the anterior pituitary gland of lactating sows has been well reviewed by Britt *et al.* (1985). It was shown that both the synthesis and secretion of LH were inhibited throughout the lactation of sows (Crighton and Lamming, 1969) and that this was attributed to a suppression of hypothalamic GnRH by the sucking stimulus (Cox and Britt, 1982a,b). However, high levels of FSH in the anterior pituitary gland of lactating sows suggested that there was an inhibition of FSH secretion but not synthesis (Crighton and Lamming, 1969). Two possible mechanisms have been proposed for the control of FSH secretion from the pituitary gland of lactating sows: firstly a sucking induced inhibition of GnRH release and secondly the existence of an inhibitory "non-steroidal factor" that may originate from the ovaries (Stevenson *et al.*, 1981; Britt *et al.*, 1985).

The levels of total thyroxine decreased during the final month of gestation, remained at low values on the day of farrowing and were low throughout the subsequent lactation period (Benjaminsen, 1981). It was suggested that lactation may suppress thyroid function in the sow, possibly through a mechanism regulating the release of thyrotropin-releasing hormone and thyrotropin-stimulating hormone (Benjaminsen, 1981). This mechanism appears to be similar to that regulating LH and FSH release during lactation and may be related to the intensity of sucking.

Autocrine control

Studies in the lactating goat have revealed that the rate of milk secretion may be under autocrine control from the mammary glands through the action of a chemical

inhibitor in the milk (Peaker and Wilde, 1987). Goats that were milked either hourly or thrice daily from one mammary gland, but only twice daily from the other mammary gland, had a marked increase in milk yield only from the mammary gland receiving the more frequent milkings (Linzell and Peaker, 1971b; Blatchford and Peaker, 1982; Henderson *et al.*, 1983). Moreover, this stimulation of milk yield was rapidly reversed with a return to twice daily milkings (Henderson *et al.*, 1983). These findings indicated that there was a local intramammary control on milk secretion in the goat. It has become clear that the increase in milk yield with more frequent milkings is not associated with either systemic factors or a reduction in the physical distension of the mammary glands but may be accounted for by the continual removal of a local chemical inhibitor (Henderson and Peaker, 1984). Indeed, a fraction of goat's milk containing whey proteins (M_r 10,000-30,000) was shown to reversibly inhibit lactose and casein synthesis in rabbit mammary explants that were maintained in organ culture (Wilde *et al.*, 1987). The same fraction also was capable of reducing the milk secretion of lactating rabbits and goats when injected into the ducts of the mammary gland through the teat (Wilde *et al.*, 1987, 1988). It appears that the site of action for the chemical inhibitor is the secretory alveoli which means that milk yield will increase only when the residual milk in the alveoli is removed during sucking or milking (Henderson and Peaker, 1987). Conversely, a reduction in milk yield would arise from an accumulation of the chemical inhibitor with prolonged storage of milk in the mammary glands.

Although there is no direct evidence for autocrine control on milk secretion in the mammary glands of the sow, various aspects of sow lactation suggest that a feedback regulation also may be present. For instance, it has been observed that when individual mammary glands on a sow failed to receive an adequate suckling stimulus they would involute rapidly, while others that were being actively suckled would continue to produce milk (Martin *et al.*, 1978). Also, extending the suckling interval of piglets to longer than 2 h resulted in a decrease in the milk yield of the sow (Barber *et al.*, 1955). It is of interest that the mammary glands of the sow lack teat cisternae for milk storage and that most of the secreted milk is held within the alveoli. If a local chemical inhibitor for milk secretion is present in sows' milk, then it may have been of evolutionary importance that the mammary glands were suckled at frequent intervals.

Milk yield

Milk yield in the sow has been assessed with the weigh-suckle-weigh (WSW) method (Barber *et al.*, 1955; Lewis *et al.*, 1978; Speer and Cox, 1984), by machine-milking the sow after the administration of oxytocin (Hartman and Pond, 1960), by weekly weight gains of piglets (Lewis *et al.*, 1978) and, more recently, with the technique of isotope dilution (Pettigrew *et al.*, 1987). Most workers use the WSW method for estimating milk yield. However, it is a laborious method which involves the weighing of piglets immediately before and after a sucking with the difference in weight being taken as the amount of milk ingested by the piglets. This procedure is repeated several times throughout a day and selected weighings are used to estimate the daily milk yield (Speer and Cox, 1984). Normally the litters are separated from their sows between weighings and are only returned at predetermined intervals for sucking. Pettigrew *et al.* (1985) have pointed out that such a procedure disrupts the normal sucking patterns of the piglets and may either adversely affect the milk production of the sow or lead to an unsuccessful milk letdown. Certainly the sucking interval imposed on the litter must be kept similar to their natural sucking frequency, particularly as Barber *et al.* (1955) have shown that piglets suckling only every 2-3 h received less milk and had a much lower growth rate than those piglets allowed to suckle every hour. There is also the problem of an under-estimation of milk yield because of piglet weight loss from urination, defecation, metabolic processes, salivation and sweat loss during sucking (Pettigrew *et al.*, 1985). Correction factors for some of these losses have been proposed

(van Spaendonck and Vanschoubroek, 1964; Klaver *et al.*, 1981; Speer and Cox, 1984) but may not be satisfactory for all experimental conditions. An alternative to using the piglet WSW method would be to weigh the sow immediately before and after the suckling (Braude *et al.*, 1954). However, this method requires a highly sensitive balance with an integrator to compensate for sow movement during the weighings and the estimated milk yield can only be related to the litter and not to the individual piglets. Also, correction for moisture loss would be important during each weighing period (Arthur *et al.*, 1987).

Isotope dilution was originally used by MacFarlane *et al.* (1969) for the estimation of milk intake of lambs and calves but has since been applied to other mammalian neonates with reasonable success. Recent efforts have been directed at developing this technique as an accurate means of estimating the milk intake of piglets (Yang *et al.*, 1980; Rudolph *et al.*, 1984; Pettigrew *et al.*, 1985; Pettigrew *et al.*, 1987). Basically isotope dilution works on the assumption that colostrum and milk are the only sources of water ingested by the piglets during the early stages of lactation. Water intake is determined by injecting into the piglets water labelled with an isotope of hydrogen (deuterium or tritium) and measuring its dilution in the total body water. This can be converted into milk intake if the percentage of water in milk is known. Normally the measurement intervals are 12-24 h or more which means that isotope dilution has the advantage of minimal disturbance to the sow and her litter. Indeed, some studies have shown that in comparison to isotope dilution, the WSW method underestimates the milk intake of piglets (Rudolph *et al.*, 1984; Pettigrew *et al.*, 1985). However, an important limitation on isotope dilution is that piglets begin to consume supplied water, and perhaps solid feed, when they are about 4 weeks of age (Yang *et al.*, 1980). Since most intensive piggeries wean piglets around 3-4 weeks of age this may not be considered a problem but various experimental restrictions would be required if isotope dilution is to be used for the estimation of milk production in sows with extended lactations.

A sow milking machine has been used to compare the yield of milk from different teats during the very early lactation period (Fraser *et al.*, 1985), but there has been no attempt to use one during a natural suckling. Perhaps this is not surprising considering that the sow has a very tight control over her milk letdown. In studies where milk yield has been determined during established lactation with a milking machine, the sow was transferred into a restraining crate, injected with a large dose of oxytocin and milked over an extended letdown period (Hartman and Pond, 1960; Hartman *et al.*, 1962). Docile sows were usually selected for machine milking, otherwise it was necessary to administer a tranquillizer. Even so, when the milk yield was estimated both with a milking machine and with the WSW method it was found that the correlation between the two methods improved as lactation progressed (Hartman *et al.*, 1962). Thus, one of the suggestions put forward was that the sows may need to be accustomed to the milking machine before an accurate estimation of milk yield can be achieved (Hartman *et al.*, 1962).

Estimating milk yield from the weight gains/week of the piglets was reported by Lewis *et al.* (1978) to be subject to large error. Their study indicated that the weight gain of piglets was greatly influenced by genetic and environmental factors as well as by the milk yield of the sows.

Generally the average milk yield for sows has been estimated to range from 4-7 kg/week/piglet over an 8 week lactation with peak productions occurring during the third and fifth week of lactation (Figure 2). Milk yield then declines to very low levels by the ninth and tenth week of lactation (Pond and Houpt, 1978). The decline in milk yield may be attributed to a combination of a natural decrease in sucking frequency and the management procedure of introducing solid feed to piglets during the later stages of lactation. Considerable variation has been observed with the milk yield of sows of

different breeds and crosses (Allen and Lasley, 1960). Also, Smith (1952) has reported that the milk yield of sows tended to increase with subsequent lactations (Figure 2).

Involution (weaning)

The process of involution, which occurs in the sow after the piglets are weaned, leads to a rapid regression of the milk secretory tissue in the mammary glands. As a result of continued milk synthesis the glands become maximally engorged with milk within the first 24-30 h of weaning (Turner, 1952). Histological examination at this time indicated that the alveoli had become greatly distended with milk products and that the arterio-venous capillary system around the alveoli was still present (Cross *et al.*, 1958). After this period, synthesis ceased and the stored milk was slowly reabsorbed leading to a reduction in the size of the mammary glands. By 48 h after weaning degeneration of the alveoli was evident and few blood capillaries remained (Cross *et al.*, 1958). A period of slow reconstruction occurred after day 4 of involution as the lobulo-alveolar tissue was replaced by proliferating intralobular stromal tissue along with adipose tissue (Cross *et al.*, 1958).

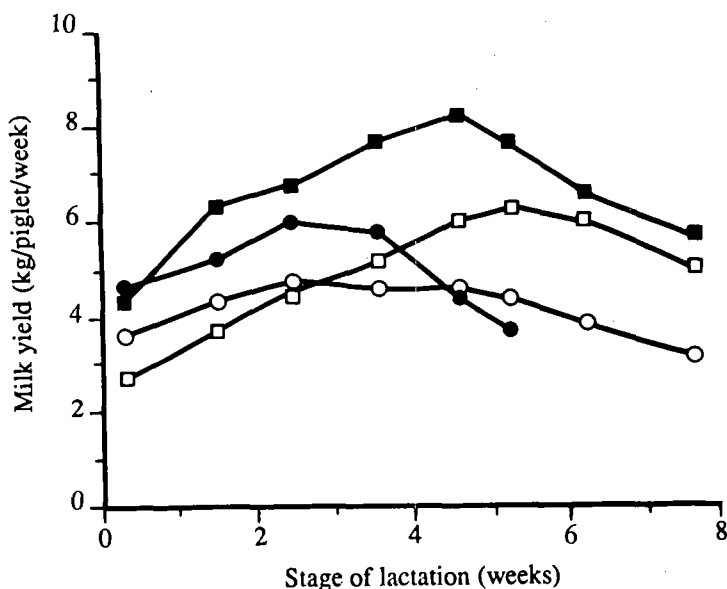


Figure 2. Milk yield (kg/week/piglet) in sows from 0-8 weeks of lactation. Berkshire first lactation (open squares), Berkshire third lactation (solid squares) (from Smith, 1952); Large White gilts (open circles) (from Barber *et al.*, 1985); Hampshire sows (solid circles) (from Hartman *et al.*, 1962).

The concentration of prolactin in plasma declined rapidly to low values (1-2 $\mu\text{g/l}$) within a few hours of weaning (Bever *et al.*, 1978) and remained low until the next oestrus. In contrast, the concentration of thyroxine increased rapidly in the post-weaning period (Benjaminsen, 1981).

Many intensive piggeries aim at increasing their annual productivity by reducing the length of lactation in their sows. However, when the length of lactation was less than 3 weeks, not only was the weaning to return of oestrus interval prolonged but there was also a reduction in the number of piglets born at the end of the next pregnancy (Hughes and Varley, 1980). On the other hand, extended lactations do not

result in large increases in litter size at subsequent pregnancies, and therefore most intensive piggeries tend to wean piglets between 21-28 days after parturition.

Conclusion

Increased profits have been achieved in the intensive pig industry by exploiting the full reproductive potential of the sow. This increase has been achieved by the use of selection pressure and improved environmental control. Despite the advances which have been made in increasing the reproductive potential of the sow, the important role of sows' milk in promoting piglet growth, development and protection against pathogenic micro-organisms has been largely ignored. Therefore, we need to know considerably more about the factors affecting the relationship between the piglets' demand for milk and the sow's capacity to produce milk before real progress can be made in developing management strategies to maximize the benefits of an important resource, sows' milk.

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References

- AFELE, S., BRYANT-GREENWOOD, G.D., CHAMLEY, W.A. and DAX, E.M. (1979). Plasma relaxin immunoactivity in the pig at parturition and during nuzzling and suckling. *Journal of Reproduction and Fertility*. **56**:451-457.
- ALGERS, B. and JENSEN, P. (1985). Communication during suckling in the domestic pig. Effects of continuous noise. *Applied Animal Behaviour Science*. **14**:49-61.
- ALLEN, A.D. and LASLEY, J.F. (1960). Milk production of sows. *Journal of Animal Science*. **19**:150-155.
- ARTHUR, P.G., HARTMANN, P.E. and SMITH, M. (1987). Measurement of the milk intake of breast-fed infants. *Journal of Pediatric Gastroenterology and Nutrition*. **6**:758-763.
- ATROSHI, F., ALAVIUHKOLA, T., SCHILDT, R. and SANDHOLM, M. (1983). Fat globule membrane of sow milk as a target for adhesion of K88-positive *Escherichia coli*. *Comparative Immunology, Microbiology and Infectious Diseases*. **6**:235-245.
- AUMAITRE, A. and SEVE, B. (1978). Nutritional importance of colostrum in the piglet. *Annales de Recherches Veterinaires*. **9**:181-192.
- BARBER, R.S., BRAUDE, R. and MITCHELL, K.G. (1955). Studies on milk production of large white pigs. *Journal of Agricultural Science*. **46**:97-118.
- BEACOM, S.E. and BOWLAND, J.P. (1951). The essential amino acid (except tryptophan) content of colostrum and milk of the sow. *Journal of Nutrition*. **45**:419-429.
- BELL, K., MCKENZIE, H.A. and SHAW, D.C. (1981). Porcine β -lactoglobulin A and C. Occurrence, isolation and chemical properties. *Molecular and Cellular Biochemistry*. **35**:103-111.
- BENJAMINSEN, E. (1981). Plasma thyroxine in the sow during pregnancy and lactation and during resumption of ovarian activity after weaning. *Acta Veterinaria Scandinavica*. **22**:369-381.
- BEVERS, M.M., WILLEMSE, A.H. and KRUIP, TH.A.M. (1978). Plasma prolactin levels in the sow during lactation and the post-weaning period as measured by radioimmunoassay. *Biology of Reproduction*. **19**:628-634.
- BICKERSTAFFE, R. and ANNISON, E.F. (1968). The desaturation of stearic acid by mammary-gland tissue of the lactating goat and sow. *Biochemistry Journal*. **108**:47P-48P.
- BLATCHFORD, D.R. and PEAKER, M. (1982). Effects of frequent milking on milk secretion during lactation in the goat: Relation to factors which limit the rate of secretion. *Quarterly Journal of Experimental Physiology*. **67**:303-310.
- BOURNE, F.J. and CURTIS, J. (1973). The transfer of immunoglobulins IgG, IgA and IgM from serum to colostrum and milk in the sow. *Immunology*. **24**:157-162.
- BOWLAND, J.P. (1966). In "Swine in Biomedical Research", pp. 97-107, eds. L.K. Bustad, R.O. McClellan and M.P. Burns. (Pacific Northwest Laboratory: Richland, Washington).
- BRANTL, V. (1984). Novel opioid peptides derived from human β -Casein: Human β -casomorphins. *European Journal of Pharmacology*. **106**:213-214.
- BRANTL, V., TESCHEMACHER, H., HENSCHEN, A. and LOTTSPREICH, F. (1979). Novel opioid peptides derived from casein (β -Casomorphins). *Hoppe-Seyler's Zeitschrift Fur Physiologische Chemie*. **360**:1211-1216.

- BRAUDE, R. (1954). In "Progress in the Physiology of Farm Animals", volume 1, pp. 40-105, ed. J. Hammond (Butterworths Scientific Publications: London).
- BRAUDE, R., COATES, M.E., HENRY, K.M., KON, S.K., ROWLAND, S.J., THOMPSON, S.Y. and WALKER, D.M. (1947). A study of the composition of sow's milk. *British Journal of Nutrition*. 1:64-77.
- BREW, K. (1969). Secretion of α -lactalbumin into milk and its relevance to the organization and control of lactose synthetase. *Nature*. 222:671-672.
- BRITT, J.H. (1986). Improving sow productivity through management during gestation, lactation and after weaning. *Journal of Animal Science*. 63:1288-1296.
- BRITT, J.H., ARMSTRONG, J.D., COX, N.M. and ESBENSHADE, K.L. (1985). Control of follicular development during and after lactation in sows. *Journal of Reproduction and Fertility*. Supplement 33:37-54.
- BROWN, K.D. and BLAKELY, D.M. (1983). Inhibition of the binding of 125 I-labelled epidermal growth factor to mouse cells by a mitogen in goat mammary secretions. *Biochemistry Journal*. 212:465-472.
- BROWN, P.J., BOURNE, F.J. and DENNY, H.R. (1975). Immunoglobulin-containing cells in pig mammary gland. *Journal of Anatomy*. 120:329-335.
- BUTTLE, H.L. (1988). Role of the ovaries in inducing mammogenesis in pregnant pigs. *Journal of Endocrinology*. 118:41-45.
- CARLSSON, L.C.T., WESTROM, B.R. and KARLSSON, B.W. (1980). Intestinal absorption of proteins by the neonatal piglet fed on sow's colostrum with either natural or experimentally eliminated trypsin-inhibiting activity. *Biology of the Neonate*. 38:309-320.
- CARLSSON, R.N.K., INGVARSSON, B.I. and KARLSSON, B.W. (1977). Isolation and characterization of albumin from porcine serum, colostrum and urine. *International Journal of Biochemistry*. 8:285-294.
- CERA, K., MAHAN, D.C. and SIMMEN, F.A. (1987). *In vitro* growth-promoting activity of porcine mammary secretions: Initial characterization and relationship to known peptide growth factors. *Journal of Animal Science*. 65:1149-1159.
- CERNING-BEROARD, J. (1984). Isolation, amino acid composition and phosphorus content of porcine α_2 -casein. *Milchwissenschaft*. 39:526-527.
- CERNING-BEROARD, J. and ZEVACO, C. (1984). Purification and characterization of porcine k-casein. *Journal of Dairy Research*. 51:259-266.
- CHOBERT, J.-M., MERCIER, J.-C., BAHY, C. and HAZE, G. (1976). Structure primaire du caseino-macropéptide des caseines porcine et humaine. *Federation of European Biochemical Societies Letters*. 72:173-178.
- COWIE, A.T., FORSYTH, I.A. and HART, I.C. (1980). In "Hormonal Control of Lactation" (Monographs of Endocrinology, Number 15), pp. 144-229, eds. F. Gross, A. Labhart, T. Mann and J. Zander (Springer-Verlag: New York).
- COX, N.M. and BRITT, J.H. (1982a). Relationships between endogenous gonadotropin-releasing hormone, gonadotropins, and follicular development after weaning in sows. *Biology of Reproduction*. 27:70-78.
- COX, N.M. and BRITT, J.H. (1982b). Pulsatile administration of gonadotropin releasing hormone to lactating sows: Endocrine changes associated with induction of fertile estrus. *Biology of Reproduction*. 27:1126-1137.
- CRIGHTON, D.B. and LAMMING, G.E. (1969). The lactational anoestrus of the sow: The status of the anterior pituitary-ovarian system during lactation and after weaning. *Journal of Endocrinology*. 43:507-519.
- 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.
- De PASSILLE, A.M., RUSHEN, J. and HARTSTOCK, T.G. (1988). Ontogeny of teat fidelity in pigs and its relation to competition at suckling. *Canadian Journal of Animal Science*. 68:325-338.
- DeMAN, J.M. and BOWLAND, J.P. (1963). Fatty acid composition of sow's colostrum, milk and body fat as determined by gas-liquid chromatography. *Journal of Dairy Research*. 30:339-343.
- DUNCAN, W.R.H. and GARTON, G.A. (1966). The component fatty acids of the colostrum fat and milk fat of the sow. *Journal of Dairy Research*. 33:255-259.
- DUSZA, L. and KRYMOWSKA, H. (1981). Plasma prolactin levels in sows during pregnancy, parturition and early lactation. *Journal of Reproduction and Fertility*. 61:131-134.
- EARLE, I.P. and STEVENSON, J.W. (1965). Relation of dietary zinc to composition of sow colostrum and milk. *Journal of Animal Science*. 24:325-328.
- EBNER, K.E. and BRODBECK, URS. (1968). Biological role of α -lactalbumin: A review. *Journal of Dairy Science*. 51:317-322.
- EBNER, K.E. and SCHANBACHER, F.L. (1974). In "Lactation: A Comprehensive Treatise", volume II, pp. 77-113, eds. B.L. Larson and V.R. Smith (Academic Press: New York and London).

- ELLENDORF, F., FORSLING, M.L. and POULAIN, D.A. (1982). The milk ejection reflex in the pig. *Journal of Physiology*. 333:577-594.
- ELLENDORF, 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.
- ELLICOTT, A.R. and DZIUK, P.J. (1973). Minimum daily dose of progesterone and plasma concentration for maintenance of pregnancy of ovariectomized gilts. *Biology of Reproduction*. 9:300-304.
- ELLIOT, J.I., SENFT, B., ERHARDT, G. and FRASER, D. (1984). Isolation of lactoferrin and its concentration in sows' colostrum and milk during a 21-day lactation. *Journal of Animal Science*. 59:1080-1084.
- FAHMY, M.H. (1972). Comparative study of colostrum and milk composition of seven breeds of swine. *Canadian Journal of Animal Science*. 52:621-627.
- FARMER, C., HOUTZ, S.K. and HAGEN, D.R. (1987). Estrone concentration in sow milk during and after parturition. *Journal of Animal Science*. 64:1086-1089.
- FAULKNER, A. and PEAKER, M. (1982). Secretion of citrate into milk. *Journal of Dairy Research*. 49:159-169.
- FLEET, I.R., GOODE, J.A., HAMON, M.H., LAURIE, M.S., LINZELL, J.L. and PEAKER, M. (1975). Secretory activity of goat mammary glands during pregnancy and the onset of lactation. *Journal of Physiology*. 251:763-773.
- FOLLEY, S.J. and KNAGGS, G.S. (1966). Milk-ejection activity (oxytocin) in the external jugular vein blood of the cow, goat and sow, in relation to the stimulus of milking or suckling. *Journal of Endocrinology*. 34:197-214.
- FRASER, D. (1975). The "teat order" of suckling pigs. II. Fighting during suckling and the effects of clipping the eye teeth. *Journal of Agricultural Science (Cambridge)*. 84:393-399.
- FRASER, D. (1977). Some behavioral aspects of milk ejection failure by sows. *British Veterinary Journal*. 133:126-133.
- FRASER, D. (1980). A review of the behavioral mechanism of milk ejection of the domestic pig. *Applied Animal Ethology*. 6:247-255.
- FRASER, D., NICHOLLS, C. and FAGAN, W. (1985). A sow milking machine designed to compare the yield of different teats. *Journal of Agricultural Engineering Research*. 31:371-376.
- FRASER, D. and THOMPSON, B.K. (1986). Variation in piglet weights: Relationship to suckling behaviour, parity number, and farrowing crate design. *Canadian Journal of Animal Science*. 66:31-46.
- GAULL, G.E., JENSEN, R.G., RASSIN, D.K. and MALLOY, M.H. (1982). In "Advances in Perinatal Medicine", volume 2, pp. 47-120, eds. A. Milunsky, E.A. Friedman and L. Gluck (Plenum Publishing Corporation: New York).
- GOFF, J.P., HORST, R.L. and LITLEDIKE, E.T. (1984). Effect of sow vitamin D status at parturition on the vitamin D status of neonatal piglets. *Journal of Nutrition*. 114:163-169.
- GOONERATNE, A.D., BRYANT-GREENWOOD, G., MAULE WALKER, F., NOTTAGE, H.M. and HARTMANN, P.E. (1983). Pre-partum changes in the plasma concentrations of progesterone, relaxin, prostaglandin F-2 α and 13,14-dihydro-15-keto prostaglandin F-2 α in meclofenamic acid-treated sows. *Journal of Reproduction and Fertility*. 68:33-40.
- GOONERATNE, A.D., HARTMANN, P.E., MCCAULEY, I. and MARTIN, C.E. (1979). Control of parturition in the sow using progesterone and prostaglandin. *Australian Journal of Biological Sciences*. 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.
- GRIGOR, M.R. and HARTMANN, P.E. (1985). NADP-linked dehydrogenases in secreted milk. *Journal of Dairy Research*. 52:501-506.
- HACKER, R.R. and HILL, D.L. (1972). Nucleic acid content of mammary glands of virgin and pregnant gilts. *Journal of Dairy Science*. 55:1295-1299.
- HAMOSH, M., FREED, L.M., JONES, J.B., BERKOW, S.E., BITMAN, J., MEHTA, N.R., HAPP, B. and HAMOSH, P. (1985). In "Human Lactation: Milk Components and Methodologies", pp. 251-266, eds R.G. Jensen and M.C. Neville (Plenum Press: New York).
- HARTMAN, D.A., LUDWICK, T.M. and WILSON, R.F. (1962). Certain aspects of lactation performance in sows. *Journal of Animal Science*. 21:888-886.
- HARTMAN, D.A. and POND, W.G. (1960). Design and use of a milking machine for sows. *Journal of Animal Science*. 19:780-785.
- HARTMANN, P.E. (1973). Changes in the composition and yield of the mammary secretion of cows during the initiation of lactation. *Journal of Endocrinology*. 59:231-247.
- HARTMANN, P.E., BIRD, P.H. and HOLMES, M.A. (1989). The influence of lactation on piglet survival. In "Manipulating Pig Production II", pp. 101-134, eds J.L. Barnett and D.P. Hennessy (Australasian Pig Science Association: Werribee, Victoria, Australia).
- HARTMANN, P.E., MCCAULEY, I., GOONERATNE, A.D. and WHITELY, J.L. (1984a). In "Physiological Strategies in Lactation" (Symposia of the Zoological Society of London, volume 51), pp. 301-326, eds. M. Peaker, R.G. Vernon and C.H. Knight (Academic Press: London).

- HARTMANN, P.E., WHITELEY, J.L. and WILLCOX, D.L. (1984b). Lactose in plasma during lactogenesis, established lactation and weaning in sows. *Journal of Physiology*. **347**:453-463.
- HARTSTOCK, T.G. and GRAVES, H.B. (1976). Neonatal behaviour and nutrition-related mortality in domestic swine. *Journal of Animal Science*. **42**:235-241.
- HARTSTOCK, T.G., GRAVES, H.B. and BAUMGARDT, B.R. (1977). Agonistic behaviour and the nursing order in suckling piglets: Relationships with survival, growth and body composition. *Journal of Animal Science*. **44**:320-330.
- HENDERSON, A.J., BLATCHFORD, D.R. and PEAKER, M. (1983). The effects of milking thrice instead of twice daily on milk secretion in the goat. *Quarterly Journal of Experimental Physiology*. **68**:645-652.
- HENDERSON, A.J. and PEAKER, M. (1984). Feed-back control of milk secretion in the goat by a chemical in milk. *Journal of Physiology*. **351**:39-45.
- HENDERSON, A.J. and PEAKER, M. (1987). Effects of removing milk from the mammary ducts and alveoli, of diluting stored milk, on the rate of milk secretion in the goat. *Quarterly Journal of Experimental Physiology*. **72**:13-19.
- HISAW, F.L. and ZARROW, M.X. (1948). Relaxin in the ovary of the domestic sow (*Sus scrofa*, L.). *Proceedings of the Society for Experimental Biology and Medicine*. **69**:395-398.
- HOLMES, M.A., MAUGHAN, C., PATERSON, A., BRYANT-GREENWOOD, G., RICE, G. and HARTMANN, P.E. (1988). The uptake of glucose by the mammary glands of lactating sows. *Proceedings of the Nutrition Society of Australia*. **13**:113.
- HOLT, C. (1981). Some principles determining salt composition and partitioning of ions in milk. *Journal of Dairy Science*. **64**:1958-1964.
- HOLT, C. (1985). In "Developments in Dairy Chemistry", pp. 143-181, ed. P.F. Fox. (Elsevier Applied Sciences: London and New York).
- HUGHES, P.E. and VARLEY, M.A. (1980). In "Reproduction in the Pig", pp. 136-168, eds P.E. Hughes and M.A. Varley (Butterworth and Co. Ltd.: London and Boston).
- JAEGAR, L.A., LAMAR, C.H., BOTTOMS, G.D. and CLINE, T.R. (1987). Growth-stimulating substances in porcine milk. *American Journal of Veterinary Research*. **48**:1531-1533.
- JENNESS, R. (1974). In "Lactation: A Comprehensive Treatise", volume III, pp. 3-107, eds B.L. Larson and V.R. Smith (Academic Press: New York and London).
- JENNESS, R. (1985). In "Lactation", pp. 164-197, eds B.L. Larson and R.R. Anderson (Iowa State University Press: Iowa).
- JENSEN, T.P. and PEDERSEN, K.B. (1979). Studies on immunoglobulins and trypsin inhibitor in colostrum and milk from sows and in serum of their piglets. *Acta Veterinaria Scandinavica*. **20**:60-72.
- JEPPESSEN, L.E. (1982). Teat-order in groups of piglets reared on an artificial sow. I. Formation of teat-order and influence of milk yield on teat preference. *Applied Animal Ethology*. **8**:335-345.
- KENDALL, J.Z., RICHARDS, G.E. and SHIH, L. N. (1983). Effect of haloperidol, suckling, oxytocin and hand milking on plasma relaxin and prolactin concentrations in cyclic and lactating pigs. *Journal of Reproduction and Fertility*. **69**:271-277.
- KENDALL, J.Z., RICHARDS, G.E., SHIH, L. N. and FARRIS, T.S. (1982). Plasma relaxin concentrations in the pig during the periparturient period: Association with prolactin, estrogen and progesterone. *Theriogenology*. **17**:677-687.
- 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.
- KERNER, J., FROSETH, J.A., MILLER, E.R. and BIEBER, L.L. (1984). A study of the acylcarnitine content of sows' colostrum, milk and newborn piglet tissues: Demonstration of high amounts of isovaleryl-carnitine in colostrum and milk. *Journal of Nutrition*. **114**:854-861.
- KERTILES, L.P. and ANDERSON, L.L. (1979). Effect of relaxin on cervical dilation, parturition and lactation in the pig. *Biology of Reproduction*. **21**:57-68.
- KESSLER, E. and BREW, K. (1970). The whey proteins of pig's milk isolation and characterization of a β -lactoglobulin. *Biochimica et Biophysica Acta*. **200**:449-458.
- KIRKWOOD, R.N., LAPWOOD, K.R., SMITH, W.C. and ANDERSON, I.L. (1984). Plasma concentrations of LH, prolactin, oestradiol-17 β and progesterone in sows weaned after lactation for 10 or 35 days. *Journal of Reproduction and Fertility*. **70**:95-102.
- KJELLBERG, B. and KARLSSON, B.W. (1967). Comparative analyses of lactic and malic dehydrogenases and their multiple molecular forms in milk from various animal species and man. *Comparative Biochemistry and Physiology*. **22**:397-413.
- KLAVER, J., van KEMPEN, G.J.M., De LANGE, P.G.B., VERSTEGEN, M.W.A. and BOER, H. (1981). Milk composition and daily yield of different milk components as affected by sow condition and lactation/feeding regimen. *Journal of Animal Science*. **52**:1091-1097.

- KLOBASA, F., WERHAHN, E. and BUTLER, J.E. (1987). Composition of sow milk during lactation. *Journal of Animal Science*. 64:1458-1466.
- KOLDOVSKY, O. (1980). Hormones in milk. *Life Sciences*. 26:1833-1836.
- KONAR, A., THOMAS, P.C. and ROOK, J.A.F. (1971). The concentrations of water-soluble constituents in the milks of cows, ewes and goats. *Journal of Dairy Research*. 38:333-341.
- KRUSE, P.E., DANIELSEN, V., NIELSEN, H.E. and CHRISTENSEN, K. (1977). The influence of different dietary levels of linoleic acid on reproductive performance and fatty acid composition of milk fat and plasma lipids in pigs. *Acta Agricultura Scandinavica*. 27:289-297.
- KUHN, N.J., CARRICK, D.T. and WILDE, C.J. (1980). Lactose synthesis: The possibilities of regulation. *Journal of Dairy Science*. 63:328-336.
- LARSON, B.L. and JORGENSEN, G.N. (1974). In "Lactation: A Comprehensive Treatise", volume II, pp. 115-145, eds. B.L. Larson and V.R. Smith (Academic Press: New York and London).
- LASKOWSKI, M., KASSELL, B. and HAGERTY, G. (1957). A crystalline trypsin inhibitor from swine colostrum. *Biochimica et Biophysica Acta*. 24:300-305.
- LEE, C.S., McCAULEY, I. and HARTMANN, P.E. (1983). Light and electron microscopy of cells in pig colostrum, milk and involution secretion. *Acta Anatomica*. 116:126-135.
- LEWIS, A.J., SPEER, V.C. and HAUGHT, D.G. (1978). Relationship between yield and composition of sows' milk and weight gains of nursing pigs. *Journal of Animal Science*. 47:634-638.
- LEWIS, N.J. and HURNIK, J.F. (1986). An approach response of piglets to the sow's nursing localisations. *Canadian Journal of Animal Science*. 66:537-539.
- LINCOLN, D.W. and PAISLEY, A.C. (1982). Neuroendocrine control of milk ejection. *Journal of Reproductive Fertility*. 65:571-586.
- LINZELL, J.L., MEPHAM, T.B., ANNISON, E.F. and WEST, C.E. (1969). Mammary metabolism in lactating sows: Arteriovenous differences of milk precursors and the mammary metabolism of [¹⁴C] glucose and [¹⁴C] acetate. *British Journal of Nutrition*. 23:319-332.
- LINZELL, J.L. and PEAKER, M. (1971a). Mechanism of milk secretion. *Physiological Reviews*. 51:564-597.
- LINZELL, J.L. and PEAKER, M. (1971b). The effects of oxytocin and milk removal on milk secretion in the goat. *Journal of Physiology*. 216:717-734.
- LYONS, W.R. (1958). Hormonal synergism in mammary growth. *Proceedings of the Royal Society of London, (B)*. 149:303-324.
- LYSTER, R.L.J. (1972). Reviews of the progress of dairy science. Section C. Chemistry of milk proteins. *Journal of Dairy Research*. 39:279-318.
- MacFARLANE, W.V., HOWARD, B. and SIEBERT, B.D. (1969). Tritiated water in the measurement of milk intake and tissue growth of ruminants in the field. *Nature*. 221:578-579.
- MAFFEO, G., DAMASIO, M., BALABIO, R. and JOCHLE, W. (1987). Detection of prostaglandin-like substances in sows' milk. *Zuchthygiene*. 22:209-214.
- MAISI, P., JUNTILLA, J. and SEPPANEN, J. (1987). Detection of subclinical mastitis in ewes. *British Veterinary Journal*. 143:402-409.
- MALVEN, P.V. (1977). Prolactin and other protein hormones in milk. *Journal of Animal Science*. 46:609-616.
- MARRABLE, A.W. (1971). "The Embryonic Pig: A Chronological Account" (Beckman: New York).
- 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 Sciences*. 31:517-525.
- MATTILA, T. and SANDHOLM, M. (1985). Antitrypsin and N-acetyl- β -D-glucosaminidase as markers of mastitis in a herd of Ayrshire cows. *American Journal of Veterinary Research*. 46:2453-2456.
- MATTIOLI, M., CONTE, F., GALEATI, G. and SEREN, E. (1986). Effect of naloxone on plasma concentrations of prolactin and LH in lactating sows. *Journal of Reproduction and Fertility*. 76:167-173.
- MIGLIORE-SAMOUR, D. and JOLLES, P. (1988). Casein, a prohormone with an immunomodulating role for the newborn? *Experientia*. 44:188-193.
- MILLER, G.M., CONRAD, J.H. and HARRINGTON, R.B. (1971). Effect of dietary unsaturated fatty acids and stage of lactation on milk composition and adipose tissue in swine. *Journal of Animal Science*. 32:79-83.
- MORRISON, W.R. (1970). In "Topics in Lipid Chemistry", volume 1, pp. 51-106, ed. F.D. Gunstone (Logos Press Limited Scientific Publications: London).
- MULLOY, A.L. and MALVEN, P.V. (1979). Relationships between concentrations of porcine prolactin in blood serum and milk of lactating sows. *Journal of Animal Science*. 48:876-881.
- MULVIHILL, D.M. and FOX, P.F. (1979). Isolation and characterization of porcine β -casein. *Biochimica et Biophysica Acta*. 578:317-324.
- NARA, B.S. and FIRST, N.L. (1981). Effect of indomethacin and prostaglandin F-2 α 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.
- NEVILLE, M.C. and WATTERS, C.D. (1983). Secretion of calcium into milk: A review. *Journal of Dairy Science*. **66**:371-380.
- NICHOLAS, K.R. and HARTMANN, P.E. (1981). Progesterone control of the initiation of lactose synthesis on the rat. *Australian Journal of Biological Sciences*. **34**:435-443.
- NIELSON, H.E. and KRUSE, P.E. (1974). Effects of dietary horse beans (*Vicia faba*) on colostrum and milk composition and milk yield in sows. *Livestock Production Science*. **1**:179-185.
- NORDSKOG, A.W. and CLARK, R.T. (1945). Ergotism in pregnant sows, female rats and guinea-pigs. *American Journal of Veterinary Research*. **6**:107-116.
- OFTEDAL, O.T. (1984). In "Physiological Strategies in Lactation" (Symposia of the Zoological Society of London, volume 51), pp. 33-85, eds. M. Peaker, R.G. Vernon and C. H. Knight (Academic Press: London).
- PATTON, S. and JENSEN, R.G. (1976). "Biomedical Aspects of Lactation" (Pergamon Studies in the Life Sciences) (Pergamon Press: Oxford).
- PATTON, S. and KEENAN, T.W. (1975). The milk fat globule membrane. *Biochimica et Biophysica Acta*. **415**:273-309.
- PEAKER, M. and LINZELL, J.L. (1975). Citrate in milk: A harbinger of lactogenesis. *Nature*. **253**:464.
- PEAKER, M. and WILDE, C.J. (1987). Milk secretion: Autocrine control. *News in Physiological Sciences*. **2**:124-126.
- PERRIN, D.R. (1954). The composition of sow's milk during the course of lactation. *Journal of Dairy Research*. **21**:55-62.
- PERRIN, D.R. (1955). The chemical composition of the colostrum and milk of the sow. *Journal of Dairy Research*. **22**:103-107.
- PETHERICK, J.C. (1983). A note on nursing termination and resting behaviour of suckling piglets. *Applied Animal Ethology*. **9**:359-365.
- PETTIGREW, J.E. (1981). Supplemental dietary fat for periparturient sows: A review. *Journal of Animal Science*. **53**:107-117.
- PETTIGREW, J.E., CORNELIUS, S.G., MOSER, R.L. and SOWER, A.F. (1987). A refinement and evaluation of the isotope dilution method for estimating milk intake by piglets. *Livestock Production Science*. **16**:163-174.
- PETTIGREW, J.E., SOWER, A.F., CORNELIUS, S.G. and MOSER, R.L. (1985). A comparison of isotope dilution and weigh-suckle-weigh methods for estimating milk intake by pigs. *Canadian Journal of Animal Science*. **65**:989-992.
- PLUMLEE, M.P., THRASHER, D.M., BEESON, W.M., ANDREWS, F.N. and PARKER, H.E. (1956). The effects of a manganese deficiency upon the growth, development and reproduction of swine. *Journal of Animal Science*. **15**:352-367.
- POND, C.M. (1984). In "Physiological Strategies in Lactation" (Symposia of the Zoological Society of London, volume 51), pp. 1-32, eds. M. Peaker, R.G. Vernon and C.H. Knight (Academic Press: London).
- POND, W.G. and HOUP, K.A. (1978). "Biology of the Pig" (Cornell University Press: Ithaca and London).
- POND, W.G. and JONES, J.R. (1964). Effect of level of zinc in high calcium diets on pigs from weaning through one reproductive cycle and on subsequent growth in their offspring. *Journal of Animal Science*. **23**:1057-1060.
- POND, W.G., van VLECK, I.D. and HARTMAN, D.A. (1962). Parameter for milk yield and for percents of ash, dry matter, fat and protein in sows. *Journal of Animal Science*. **21**:293-297.
- POPE, G.S. and SWINBURNE, J.K. (1980). Hormones in milk: Their physiological significance and value as diagnostic aids. *Journal of Dairy Research*. **47**:427-449.
- RAEKALLIO, M. (1987). N-acetyl- β -D-glucosaminidase (NAGase) in porcine milk. *Acta Veterinaria Scandinavica*. **28**:173-176.
- RAYMOND, M., GAYE, P., HUE, D., HAZE, G. and MERCIER, J. (1982). Amino terminal sequence, processing, and biological activity of porcine pre- α -lactalbumin. *Biochimie*. **64**:271-278.
- REITER, B. (1978). Review of the progress of dairy science: Antimicrobial systems in milk. *Journal of Dairy research*. **45**:131-147.
- REITER, B. (1985). Protective proteins in milk - Biological significance and exploitation: Lysozyme, lactoferrin, lactoperoxidase, xanthineoxidase. *International Dairy Federation Bulletin*. **191**:1-35.
- RUDOLPH, B.C., STAHLY, T.S. and CROMWELL, G.L. (1984). Accuracy of milk intake estimates in pigs by water turnover (via D₂O dilution) and weigh-suckle methods. *Journal of Animal Science*. **59** (Supplement 1):101-102.
- SCHIMDT, D.V. and EBNER, K.E. (1971). Isolation and properties of α -lactalbumin from various sources. *Biochimica et Biophysica Acta*. **243**:273-283.

- SEERLEY, R.W., GRIFFIN, F.M. and McCAMPBELL, H.C. (1978). Effect of sow's dietary energy source on sow's milk and piglet carcass composition. *Journal of Animal Science*. **46**:1009-1017.
- SHAHANI, K.M. (1966). Milk enzymes: Their role and significance. *Journal of Dairy Science*. **49**:907-920.
- SHAHANI, K.M., KWAN, A.J. and FRIEND, B.A. (1980). Role and significance of enzymes in human milk. *The American Journal of Clinical Nutrition*. **33**:1861-1868.
- SHERWOOD, O.D., CHANG, C.C., BEVIER, G.W. and DZIUK, P.J. (1975). Radioimmunoassay of plasma relaxin levels throughout pregnancy and at parturition in the pig. *Endocrinology*. **97**:834-837.
- SIMMEN, F.A., SIMMEN, R.C.M. and REINHARDT, G. (1988). Maternal and neonatal somatomedin C/insulin-like growth factor-1 (IGF-1) and IGF binding proteins during early lactation in the pig. *Developmental Biology*. **130**:16-27.
- SLEBODZINSKI, A.B., NOWAK, J., GAWEKA, H. and SECHMAN, A. (1986). Thyroid hormones and insulin in milk: A comparative study. *Endocrinologia Experimentalis*. **20**:247-255.
- SMITH, D.M. (1952). Yield and composition of milk of New Zealand Berkshire sows. *New Zealand Journal of Science and Technology*. **34**:65-75.
- SPEER, V.C. and COX, D.F. (1984). Estimating milk yield of sows. *Journal of Animal Science*. **59**:1281-1285.
- SPINCER, J. and ROOK, J.A.F. (1971). The metabolism of [U - ^{14}C] glucose, [1 - ^{14}C] palmitic acid and [1 - ^{14}C] stearic acid by the lactating mammary gland of the sow. *Journal of Dairy Research*. **38**:315-322.
- SPINCER, J., ROOK, J.A.F. and TOWERS, K.G. (1969). The uptake of plasma constituents by the mammary gland of the sow. *Biochemistry Journal*. **111**:727-732.
- STEVENSON, J.S., COX, N.M. and BRITT, J.H. (1981). Role of the ovary in controlling luteinizing hormone, follicle stimulating hormone, and prolactin secretion during and after lactation in pigs. *Biology of Reproduction*. **24**:341-353.
- STRBAK, V. (1985). "The Role of Maternal Milk in Endocrine Regulation of Sucklings" (Veda Publishing House of the Slovak Academy of Sciences: Bratislava).
- TAVERNE, M., BEVERS, M., BRADSHAW, J.M.C., DIELEMAN, S.J., WILLEMSE, A.H. and PORTER, D.G. (1982). Plasma concentrations of prolactin, progesterone, relaxin and oestradiol- 17β in sows treated with progesterone, bromocriptine or indomethacin during late pregnancy. *Journal of Reproduction and Fertility*. **65**:85-96.
- TAVERNE, M., WILLEMSE, A.H., DIELEMAN, S.J. and BEVERS, M. (1979). Plasma prolactin, progesterone and oestradiol- 17β concentrations around parturition in the pig. *Animal Reproduction Science*. **1**:257-263.
- TOLLERZ, G. and LINDBERG, P. (1965). Influence of dietary fat and short-time starvation on the composition of sow-milk fat. *Acta Veterinaria Scandinavica*. **6**:118-134.
- TURNER, C.W. (1952). "The Mammary Gland. I. The Anatomy of the Udder of Cattle and Domestic Animals" (Lucas Brothers: Columbia, Missouri).
- UMBACH, M., TESCHEMACHER, H., PRAETORIUS, K., HIRSCHHAUSER, R. and BOSTEDT, H. (1985). Demonstration of a β -casomorphin immunoreactive material in the plasma of newborn calves after milk intake. *Regulatory Peptides*. **12**:223-230.
- VALE, G.T. and WAGNER, W.C. (1981). Plasma prolactin in the periparturient sow. *Theriogenology*. **15**:537-546.
- Van LANDEGHEM, A.A.J. and VAN DE WIEL, D.F.M. (1978). Radioimmunoassay for porcine prolactin: Plasma levels during lactation, suckling and weaning and after TRH administration. *Acta Endocrinologica*. **88**:653-667.
- Van SPAENDONCK, R.L. and VANSCHOUBROEK, F.X. (1964). Determination of the milk yield of sows and correction for loss of weight due to metabolic processes of piglets during suckling. *Animal Production*. **6**:119-123.
- WESTROM, B.R., EKMAN, R., SVENDSEN, L., SVENDSEN, J. and KARLSSON, B.W. (1987). Levels of immunoreactive insulin, neurotensin, and bombesin in porcine colostrum and milk. *Journal of Pediatric Gastroenterology and Nutrition*. **6**:460-465.
- WESTROM, B.R., OHLSSON, B.G., SVENDSEN, J., TAGESSON, C. and KARLSSON, B.W. (1985). Intestinal transmission of macromolecules (BSA and FITC-dextran) in the neonatal pig: Enhancing effect of colostrum, proteins and proteinase inhibitors. *Biology of the Neonate*. **47**:359-366.
- WESTROM, B.R., SVENDSEN, J. and KARLSSON, B.W. (1982). Protease inhibitor levels in porcine mammary secretions. *Biology of the Neonate*. **42**:185-194.
- WHATSON, T.S. and BERTRAM, J.M. (1980). A comparison of incomplete nursing in the sow in two environments. *Animal Production*. **30**:105-114.
- WHITELY, J., WILLCOX, D.L., HARTMANN, P.E., YAMAMOTO, S.Y. and BRYANT-GREENWOOD, G.D. (1985). Plasma relaxin levels during suckling and oxytocin stimulation in the lactating sow. *Biology of Reproduction*. **33**:705-714.

- WHITTEMORE, C.T. and FRASER, D. (1974). The nursing and suckling behaviour of pigs. II. Vocalisation of the sow in relation to suckling behaviour and milk ejection. *British Veterinary Journal*. **130**:346-355.
- WHITTLESTONE, W.G. (1952). The distribution of fat-globule size in sow's milk. I. The effect of sampling at intervals throughout milking. *Journal of Dairy Research*. **19**:127-132.
- WILDE, C.J., ADDEY, C.V.P., CASEY, M.J., BLATCHFORD, D.R. and PEAKER, M. (1988). Feed-back inhibition of milk secretion: The effect of a fraction of goat milk on milk yield and composition. *Quarterly Journal of Experimental Physiology*. **73**:391-397.
- WILDE, C.J., CALVERT, D.T., DALY, A. and PEAKER, M. (1987). The effect of goat milk fractions on synthesis of milk constituents by rabbit mammary explants and on milk yield *in vivo*. Evidence for autocrine control of milk secretion. *Biochemistry Journal*. **242**:285-288.
- WILLCOX, D.L., ARTHUR, P.G., HARTMANN, P.E. and WHITELY, J.L. (1983). Perinatal changes in plasma oestradiol-17 β , cortisol and progesterone and the initiation of lactation in sows. *Australian Journal of Biological Sciences*. **36**:173-181.
- WITTER, R.C. and ROOK, J.A.F. (1970). The influence of the amount and nature of dietary fat on milk fat composition in the sow. *British Journal of Nutrition*. **24**:749-760.
- WITTER, R.C., SPINCER, J., ROOK, J.A.F. and TOWERS, K.G. (1970). The effects of intravenous infusions of triglycerides on the composition of milk fat in the sow. *British Journal of Nutrition*. **24**:269-278.
- WOYCHIK, J.H. and WONDOLOWSKI, M.V. (1969). Chromatographic isolation and amino acid composition of sow α_s -, β -, and k -caseins. *Journal of Dairy Science*. **52**:901.
- WYETH, G.S.F. and McBRIDE, G. (1964). Social behaviour of domestic animals. V. A note on sucking behaviour in young pigs. *Animal Production*. **6**:245-247.
- YANG, T.S., HOWARD, B. and MACFARLANE, W.V. (1980). A note on milk intake of piglets measured by tritium dilution. *Animal Production*. **31**:201-203.
- ZILOUDROU, C., STREATY, R.A. and KLEE, W.A. (1979). Opioid peptides derived from food proteins. *Journal of Biological Chemistry*. **254**:2446-2449.

PATTERN OF MILK PRODUCTION IN SOWS

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The pattern and amount of milk produced by lactating sows was reviewed by Elsley (1971) and this review formed the basis of establishing nutrient requirements by the factorial method (ARC, 1981). However, in view of the litter growth rates achieved in commercial pig production, it is likely that present-day sows produce more milk than that suggested by ARC (1981). The aim of this study was to determine the pattern and amount of milk produced during the first 22 days of lactation of first-litter sows.

Eighteen first-litter sows received a mean daily intake of 4.66 kg sow diet during a 22-day lactation period. The diet contained (per kg) 13.5 MJ digestible energy, 168 g crude protein and 8 g lysine. Milk yield of six sows was estimated at five-day intervals throughout lactation, while milk yield of the remaining 12 sows was estimated between days 17 and 22 of lactation. Milk yield was calculated from milk intakes of individual piglets as estimated from their water turnover, determined by dilution of injected deuterium oxide (Prawirodigdo, 1989). Growth rates of individual piglets were recorded for each interval during which milk production was estimated.

Mean milk production of six sows suckling an average of 8.3 piglets between days 4-7, 7-12, 12-17 and 17-22 was 6.91, 8.78, 9.79 and 9.46 kg/day, respectively. Milk production between days 17 and 22 tended to rise as the litter size suckling the sow increased from 7 to 10 piglets (Table 1). There was a significant relationship between growth rate of individual piglets and milk consumption; $\bar{Y}=0.1636X + 46.5$, ($R^2=0.68$, $n=399$, $P<0.001$) where \bar{Y} = growth rate (g/d) and X = milk consumption (g/d).

Table 1. Milk yield of first-litter sows between days 17 and 22 of lactation

	Number of piglets in the litter					Overall 8.39
	6	7	8	9	10	
Number of sows	1	3	6	4	4	20
Mean milk yield (kg/day)	6.46	7.93	8.79	8.37	10.22	8.74
± SE		± 1.10	± 0.91	± 0.76	± 0.51	± 0.43

The estimates of milk yield proposed by Elsley (1971) for first-litter sows are approximately 55% of the yields determined in our study. The observed conversion efficiency of milk to gain supports the relatively high milk yields determined in our experiment. Changes in method of measurement, genotype and husbandry could contribute to these large differences in the pattern and yield of milk in sows.

References

- AGRICULTURAL RESEARCH COUNCIL. (1981). "The Nutrient Requirements of Pigs" (Commonwealth Agricultural Bureau: Slough).
ELSLEY, F.W.H. (1971). In "Lactation" pp. 393-411, ed. I.R. Falconer (Butterworths: London).
PRAWIRODIGDO, S. (1989). "Techniques for Estimating Milk Production by Sows" (Master of Science Thesis, University of Melbourne: Melbourne, Victoria).

WITHIN LITTER VARIATION IN MILK INTAKE DURING SUCKLING BY PIGLETS

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A combination of endocrine and behavioural investigations during established lactation have concluded that about 20% of all sucklings of sows by piglets are unsuccessful (Ellendorff *et al.*, 1982). Unsuccessful sucklings not only deprive the piglets of a source of nourishment but also may lead to a decrease in the rate of milk synthesis by the sow. Most studies have been unable to determine the number of piglets within a litter that have obtained milk at particular sucklings which, on a behavioural basis, appeared to have been successful. The current investigation determined the proportion of piglets obtaining milk by following the changes in the concentration of galactose (released from the intestinal hydrolysis of milk lactose) in the plasma of piglets after a particular undisturbed suckling.

Twelve litters (9-11 piglets/litter) were studied, six litters at 5 days and six litters at 10 days of age. Eight of the litters were observed for two consecutive undisturbed sucklings separated by 45-60 min. After the second suckle, the piglets were then separated from the sow and blood samples (60 μ l) were taken from the piglets' ear veins every 3-4 min over the next 45 min and then at 60, 75 and 90 min (suckled litters). The remaining four litters (unsuckled litters) were observed for an apparently successful suckling, separated from their respective sows for 60 min and then blood samples were collected as per the "suckled" litters. The plasma was deproteinized and assayed for galactose using luminometry. The plasma concentration of galactose was plotted against time for each piglet and the area under the curve (AUC) calculated. AUC was corrected for each piglet's basal concentration of galactose.

The results from the unsuckled litters were used to determine when milk intake had not occurred in the "suckled" litters. The mean \pm SD of the AUC for the piglets in the unsuckled litters was found to be 0.67 ± 0.27 mM/min ($n=40$) and a value equal to 3 SD above the mean (i.e. AUC=1.47 mM/min) was taken to be an arbitrary value above which all AUCs indicated milk intake. There were no significant correlations ($P>0.05$) either within litters or within age groups, between AUC and body weight and AUC and teat order.

Table 1. Proportion of piglets obtaining milk at a behaviourally successful suckling attempt and mean galactose AUC for piglets which obtained milk

Age of litter	Litter				Mean (\pm SD) AUC
	1	2	3	4	
5 days old	1.0	1.0	0.9	0.6	3.85 ± 1.68 mM/min ($n=36$)
10 days old	1.0	0.64	0.6	0.11	2.76 ± 1.00 mM/min ($n=24$)

Behavioural observations indicated that all the sucklings were successful, however, based on the absorption of milk galactose, it is apparent that this was not the case. It is concluded that not only are certain sucklings unsuccessful but that within an apparently successful suckling certain piglets may fail to obtain milk.

References

ELLENDORFF, F., FORSLING, M.L. and POVLAIN, D.A. (1982). *Journal of Physiology*. 333:557-594.

DIGESTION OF LACTOSE AND ABSORPTION OF GALACTOSE AND GLUCOSE IN NURSING PIGLETS

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The intestinal activity of lactase in piglets is high at birth, falls rapidly to two weeks of age and then falls more slowly to reach adult levels by eight weeks of age. However, based on the calculation of total intestinal lactase, it has been concluded that the capacity of intestinal lactase to hydrolyze lactose is at a maximum at about 15 days of age (Leibholz, 1986). This current study assessed the functional importance of lactase activity and monosaccharide absorption during the suckling period in piglets.

Groups of piglets were studied at 2, 5, 10, 15 and 20 days of age. In study A, six piglets from the same litter were given a galactose (0.675 g) + glucose (0.675 g) dose, and six piglets from another litter of the same age were given an equivalent lactose dose (i.e. 1.35 g). In study B, six piglets from six different litters of the same age were given a galactose + glucose dose followed by an equivalent lactose dose. Blood samples (60 μ l) were then taken from the ear veins of the piglets at frequent intervals.

The area under the curve (AUC) for plasma galactose, adjusted to the calculated plasma volume of each piglet, was determined (Table 1). There was no significant differences in galactose AUC between studies A and B for 2, 5, 10 and 15 day old piglets after a galactose + glucose dose and for 5, 10 and 15 day old piglets after a lactose dose. In study B there was a significant difference ($P < 0.05$) in galactose AUC firstly between the 20 day old piglets and each of the other ages after either a galactose + glucose or lactose dose, and secondly at each age between the galactose + glucose and the lactose doses. The efficiency of lactose digestion (calculated as ratio of galactose AUC following the lactose dose divided by that for the galactose + glucose dose) was not significantly different. There was a significant correlation ($r = 0.447$, $P < 0.02$, $n = 30$) between the age of the piglets and the change in glucose concentration after a galactose + glucose dose.

Table 1. Plasma galactose AUC (mean \pm SE, $n = 6$) after dose G (galactose + glucose) and dose L (lactose) for piglets from study A and B, and efficiency of lactose digestion (Dig. %) in study B

Study	Dose	Age (days)				
		2	5	10	15	20
A	G	2253 \pm 402	2238 \pm 267	2164 \pm 479	2221 \pm 168	3322 \pm 459
A	L	892 \pm 87	1249 \pm 172	1655 \pm 125	1592 \pm 217	785 \pm 52
B	G	2241 \pm 121	1989 \pm 18	2994 \pm 322	2670 \pm 569	5691 \pm 1136
B	L	1680 \pm 137	1558 \pm 89	1774 \pm 260	1411 \pm 123	2739 \pm 216
Dig.%		75 \pm 3	78 \pm 17	65 \pm 17	60 \pm 9	53 \pm 9

Up to 15 days of age all piglets had a similar capacity to absorb and metabolize galactose. It appeared that the digestion of lactose was the functional limitation to the piglets' capacity to utilize lactose as an energy source and that this limitation did not alter significantly up to 20 days of age.

References

LEIBHOLZ, J. (1986). *Proceedings of the Nutrition Society of Australia*. 11:32-39.

A SYMPOSIUM - NEONATAL MORTALITY IN THE PIG

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Symposium introduction

Piglet mortality to weaning has been reported to range from about 12-30% of piglets born alive (English and Morrison, 1984a). Of the numerous surveys of piglet mortality (e.g. Longwill, 1952; Sharpe, 1966; Fahmy and Bernard, 1971; English *et al.*, 1977; Glastonbury, 1977; Spicer *et al.*, 1986), there are two consistent findings. Firstly, the majority of deaths occur within 72 h of birth with at least 50% occurring in the first 24 h, and secondly, the causes of mortality are often multifactorial.

During the past 30 years the number of piglets weaned/sow/year has increased. While this has been in part due to lower weaning ages and more litters/sow/year, English and Morrison (1985) provide data for the United Kingdom which show that pre-weaning mortality declined from above 22% in 1960 to under 12% in 1982. On the other hand however, Robertson and Clarke (1980) and Hoogerbrugge (1983) are not convinced of a genuine improvement in the level of piglet survival. Whether or not this is so, there is still a concerted effort to reduce piglet mortality levels world-wide. That piglet mortality levels are too high does not appear to be disputed.

The precarious nature of neonatal existence in the pig and the potential effect of piglet mortality level on herd profitability, have caused attention to focus on how to reduce mortality levels. One major approach to promoting neonatal survival has been the provision of a controlled farrowing environment in which the degree of activity of the sow and the range of environmental conditions are limited. The importance of farrowing accommodation to total production efficiency is clearly recognised. For example, Robertson (1977) states that farrowing accommodation is in many ways the most important of all the types of housing in pig production in that it caters for both the sow and piglet at a critical stage in their life cycle, namely parturition and birth, respectively. Furthermore, it is also clear that neonatal survival depends largely on piglets reaching the udder soon after birth, nursing successfully and continuing to do so regularly thereafter (English and Morrison, 1984a), while also avoiding traumatic injury from the sow (Svendsen *et al.*, 1986).

While we accept that piglet mortality levels are too high, even at 10% of piglets born, we also realize that mortality rates in many piggeries are greater than this. A mortality rate of say 5% of piglets born alive is possible and should be the target.

The aims of this symposium are to identify factors that influence neonatal mortality in the pig and to suggest means to reduce mortality. The four papers in the symposium approach the subject from different perspectives. While there is some degree of overlap between the papers in the material presented, this highlights the awareness that neonatal mortality often has a multifactorial component and therefore any comprehensive examination requires a multidisciplinary approach. In this symposium the effects of (1) the structural environment, (2) maternal behaviour, (3) lactation and (4) management are critically examined in relation to neonatal mortality.

Symposium continued on next page

NEONATAL MORTALITY: THE INFLUENCE OF THE STRUCTURAL ENVIRONMENT

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Introduction

The baby pig is born into a hostile environment. It moves from a dependent intra-uterine existence to an independent life in the new world of social competition, thermal, chemical and structural hazard, is challenged by disease and, in some cases, attacked by predators.

In almost all commercial situations, the neonate faces the challenge of its early post-natal environment accompanied by the sow and its littermates, the former providing the major source of continuity from the intra-uterine environment and the latter, the greatest social competition. In this new environment the baby pig adapts, learns and matures. The sow contributes to the process of adaptation by providing the initial source of nutrition, immunity, warmth and security. Man too attempts to reduce the hazards of this new world by modifying the environment through temperature control (heat lamps), sow control (crates), and structural amelioration (straw or slotted floors).

Each baby pig however faces the challenge alone. The first 24-48 h post-partum are clearly the most crucial period in the association of the sow and her offspring. Piglet mortality is greatest during this period and the causal factors, though complex in their interactions (English, 1969), now appear obvious and explainable. For example, sows are large clumsy animals who outweigh their newborn by a factor of 100 or more. Crushing and trampling in an unnatural environment does not seem unusual under the circumstances. Neonates are also frail and prone to infection, chilling and starvation in environments which are climatically and pathogenically hostile.

However, for all the knowledge available, the problem of piglet mortality seems intractable to simple solutions. Although specific cases have been recorded where neonatal mortality in a herd has been reduced to less than 5%, this is unusual and on the whole there appears to have been only limited success in this area in the last 20 years of technical progress (Svendsen and Bille, 1981). Environmental factors clearly continue to contribute to probably more than three-quarters of all neonate deaths (Nielsen *et al.*, 1974).

Many of the features of contemporary farrowing pens are obvious attempts to reduce the impact of environmental hazard. Heat lamps and covered creeps improve the thermal environment for those piglets who reach them and farrowing crates are intended to reduce the incidence of overlying. Some features, however well intended, may be counterproductive. Perforated floors may reduce the presence of excreta but they undoubtedly increase physical hazards (Smith and Mitchell, 1976). Smaller pens will cost less to build but may also inhibit the behavioural repertoire of the sow and/or piglets and so contribute, then or later, to a less manageable animal. The smaller the farrowing pen becomes, the greater the proportion of space occupied by the sow and perhaps the greater the chance of traumatic injury to the piglet by the sow. A closer look at the evolution of the contemporary farrowing pen and the range of designs is of some relevance before attempting to analyse the structural features which influence neonatal mortality.

Evolution and present status of farrowing environments

The history of pig housing is long and fascinating and interspersed with recurrent examples of technical attempts to solve many of the persistent problems which still exist today (Baxter, 1984a). For example, the Romans tried farrowing rails and close confinement (Hooper, 1934; Forster and Heffner, 1954) and slatted floors were in use in several countries in the 19th century (Mechi, 1852; Harris, 1891). The writings on stockpersons' observations make it equally obvious that many of their problems also centred on pig behaviour (Henderson, 1811; Youatt, 1847). Even then however, the new technical solutions were not free from criticism on the grounds of animal welfare (Porcius (pseudonym) 1850; Spooner, 1850). However, since the 1950s, changes in farrowing pen design have been consolidated and internationalised. Inevitably it seems, the pen has become smaller, the environment more barren and the pigs more constrained. Sow confinement in a crate throughout lactation is commonplace (Baxter, 1984b). Nevertheless many isolated and interesting alternatives to close confinement have been tried and discarded as the farrowing pen has moved inexorably towards standardization. Many of these same innovations now appear to provide the source of future designs for pro-welfare, alternative systems.

Three categories of farrowing and lactation pen now appear to exist and each contains a range of pen types. In the first category, the sow is closely confined from the time she enters the pen until she is removed, usually at weaning. The total pen area is about 4-5 m² depending on the duration of occupancy by the sow and litter. In the second category of pens, the sow is confined only for the period immediately prior to parturition until 2-3 days thereafter. The area of the pen now extends to 6-8 m² and the extra space is needed for sow manoeuvres. Finally, in pens of the third group, the sow is never restrained and she is only limited in her movements by the boundaries of the pen. In this case, total pen space may exceed 8 m². The fundamental difference in the three categories of pen clearly lies in the amount of freedom and hence space given to the sow whilst she occupies the pen. However, apart from the gross space in a pen and the resultant freedom for sow and litter movement, all pens have similar, fundamental characteristics and these are now considered in some detail.

The amount and articulation of specific spaces in farrowing pens

In all farrowing pens, the following spatial zones can be identified:

- (1) safe zone(s) for piglets where they can rest and feed if necessary free from the unwanted attentions of the sow;
- (2) interaction zone(s) in which the sow and piglets occupy a common space primarily for the purpose of suckling, although other interactions of importance for the welfare of the sow, or the piglets' development, may also take place here. For example, processes leading to infant recognition, aggressive play between siblings, etc.

Where sows are confined to crates or tether stalls, the interaction zone may be little more than the area encompassed by the crate or stall but it may still occupy 40% of the total area of the pen available to the piglets. In contrast, in voluntary pens, where the sow is not constrained, the interaction zone may account for up to 85% of the total area. Piglets can only be crushed by the sow in the interaction zones.

There must be sufficient space in a farrowing pen for the sow at least to perform all her maintenance behaviours (feeding, drinking, resting and excreting) (Baxter and Schwaller, 1983) and this space may be referred to as sow space. In crate confinement, sow space is accommodated solely by the farrowing crate, an area of about 1.6 m²,

whereas in the voluntary system, it amounts to as much as 6 m². In voluntary systems, the sow space moves with the sow.

The requirements for safe zones

The following brief specification has been suggested for safe zones (Baxter, 1981a):

- (1) it must be attractive to suckling pigs of all ages and must be conducive to the piglets' use of it for resting;
- (2) it must be of sufficient size and of the correct shape to accommodate all piglets in their preferred huddling/grouping arrangements at all times;
- (3) it must provide the optimum climatic environment for health, welfare and performance. Where there is sufficient safe space for piglets to play in addition to resting, then the environments in the different spaces may also be different.

The implementation of these requirements in practice highlights a number of problems. It is difficult to find ways of attracting piglets into the safe zone in the first 48 h of life at times when they are not suckling. There are probably a number of strong motivational processes which serve to keep the piglets at the udder and until now no agricultural practices have been able to overcome these piglet motivations (Welch, 1986a). Heat and light are often used to attract piglets away from the sow. The use of a farrowing box (Robertson and McCartney, 1980) may have some influence on these motivational processes by changing the environmental circumstances immediately following birth of the piglets. Some work on the environmental control of piglet behaviour at parturition and during the suckling period has led to suggestions that suitable "pathways" might be incorporated in farrowing pens to encourage the newborn to find the safe zone as quickly and easily as possible (Welch, 1986b). Using wooden and warm soft pathways, Welch (1986a) found that piglets would use the creep area significantly earlier in life than if no pathway was present and the warm soft pathway was effective for a longer period of time than a wooden pathway.

Allometric data (Petherick, 1982, 1983) can be used to calculate the space occupied by immobile pigs and can therefore be used to estimate the size of safe zones like creep areas (Baxter, 1984a). The shape of the collective group of resting pigs will be influenced by the shape of the space available and the thermal conditions provided by heat lamps, warm floors, etc. Conflicting situations can occur when the shape of the space available for the piglets is incompatible with the shape adopted by the resting group as influenced by their thermal conditioning. Table 1 shows the estimated area occupied by ten piglets when resting in one group in a circular configuration and Table 2 for the group adopting a linear configuration. The two lying patterns described, circular and linear, are obviously oversimplifications of a much wider range of lying patterns found in practice. In some cases, the alternative patterns are the result of free choice of resting position by individual pigs in a space unconstrained by physical surroundings or limited by thermal conditions. In most cases however, the lying pattern will be at least partly influenced by predetermined thermal and spatial factors.

It is also necessary to consider where the safe zone(s) should be located with reference to other spaces and artefacts in and outside the farrowing pen. In pens with crates, every attempt is made to provide some safe space all around the crate but with the creep area(s) either on each side of the sow or at the head of the crate. With covered creeps the latter position is often favoured with its more convenient location for access and observation adjacent to the feeding passageway. There have however been attempts to change the position of the thermally enhanced areas during the period from birth to two or three days thereafter (English and Morrison, 1984b). In this situation the heat lamp is placed behind the sow at parturition and then moved to

either side of the sow soon after. Placing a heat lamp at the rear of the sow during parturition, in addition to a heated front creep, was associated with a reduction in piglet mortality, and during the first day after parturition multiple heat lamps were effective in attracting piglets to safe zones. This action was reflected in fewer deaths from overlying and lower piglet mortality at 7 days of age (Morrison *et al.*, 1983).

Table 1. Estimated area and diameter of a circular huddled group of ten piglets in three temperature conditions at three different average live weights

Thermal conditions	Age and average live weight					
	Birth (1.2 kg)		3 weeks (5 kg)		6 weeks (11 kg)	
	area (m ²)	diameter (m)	area (m ²)	diameter (m)	area (m ²)	diameter (m)
Cool	0.283	0.600	0.735	0.967	1.246	1.259
Thermoneutral	0.384	0.700	1.000	1.128	1.695	1.469
Hot	0.509	0.805	1.320	1.296	2.244	1.690

Table 2. Estimated area and linear dimension of a group of 10 piglets resting in a linear pattern in three temperature conditions at three different mean live weights

Thermal conditions	Age and average live weight					
	Birth (1.2 kg)		3 weeks (5 kg)		6 weeks (11 kg)	
	area (m ²)	width (m)	area (m ²)	width (m)	area (m ²)	width (m)
Cool	0.283	0.885	0.735	1.441	1.246	1.888
Thermoneutral	0.384	1.200	1.000	1.961	1.695	2.568
Hot	0.509	1.591	1.320	2.588	2.244	3.400

The thermal environment in safe zones

The thermal conditions required for neonates are significantly different from that of the lactating sow. The newborn piglet has a lower critical temperature of about 30-34°C (Mount, 1968), whilst that of the lactating sow may be nearer 15°C or lower. Clearly two thermal environments in the farrowing pen provides the best compromise. The heat lamp is the most common method of providing a thermal environment suitable for young pigs but other alternatives also exist. Large amounts of clean, dry deep straw bedding was the traditional method but this has now largely been superseded by heated floors or heat pads, covered creeps or insulated and heated boxes, or dull or bright emitter heat lamps.

Within the safe zones at least one area, large enough to accommodate a whole litter, should be provided with the appropriate thermal conditions. It has been shown that the average area occupied by a newborn piglet in a group under a dull emitter heat lamp was 0.0285 m² and under a bright emitter lamp it was 0.0367 m² (Thomson, 1983). Thermal conditions for at least some of the group under the dull emitter were below thermoneutrality; the bright emitter provided better thermal conditions. These results show remarkable agreement with the estimates given in column 1 of Table 1.

Swedish studies (Gustafsson, 1985) have calculated that the extra infra-red heat required by a 1.5 kg neonate in a poor thermal environment and with an energy intake satisfying maintenance requirements only, should be at least 2.0 Watts/m² in the resting area. It is also suggested that the heated area should accommodate 10 resting piglets and should be approximately 1.5 x 0.3 m in size (0.045 m²/pig).

Although circular areas will be correct for round heat lamps, there are clearly more spaces in contemporary pens of a rectangular format as indicated by the Swedish recommendations. The shape of the heated area should encourage the piglets to adopt a resting pattern compatible with the available space. Table 2 provides estimates of space for rectangular resting areas. Geers *et al.* (1986) have shown that temperature control of the floor surface can, in association with appropriate age and air temperature data, produce a preferred comfort zone for growing pigs. When the air temperature was between 14-25°C and the floor temperature was held constant at a temperature higher than air temperature, the pigs preferred a lying area enhanced by an air velocity of 0.3 m/sec. Conversely, directing jets of air into danger zones can inhibit use of these zones by piglets (Thacker, 1986).

Where the heated safe zone is not clearly differentiated by boundaries, e.g. creep boxes, it is important that the heated area does not extend into the sow space otherwise overlying may occur.

Heat lamps, creep boxes and heat pads

As a result of increasing energy costs, two methods of energy conservation have been adopted for facilities in farrowing pens. In the first case, heat lamps have been fitted with energy controlling devices and in the second, the heat lamp has been replaced by the insulated nest box. Prior to the introduction of energy controllers, the heat to the piglets was adjusted as they grew older by simply raising the heat lamp. Although this reduces the radiant heat load on the piglets, it does not reduce the energy consumed. Energy control devices now do both without adjusting the height of the lamps. Nevertheless with bad or careless management heat lamps remain potential sources of danger; electrocution of sows and fires are not infrequent occurrences.

Insulated nest boxes or covered creeps with a small light source simultaneously reduce the dangers and lower energy costs. Covered creeps contain the heat generated by metabolism and other sources, e.g. light or heat lamp, in a limited zone around the young pigs. Insulation of the box minimizes conductive heat loss and the manipulation of the openings into the box reduces heat losses by ventilation. The rate of air exchange in the box is however influenced by the sensible heat produced by the number of piglets and the size and the position of the openings in the box. Baxter (1984a) has provided data relating sensible heat production, size of opening into nest box and the temperature difference attained between the box and its surrounding environment.

However covered creeps and nest boxes are not approved of by all stockpersons because the piglets are not easily seen at a glance. Boxes with clear plastic tops or made completely from clear plastic material have been tried but they are expensive, easily scratched and soon become coated with dust which reduces their transparency. Nest boxes with open ends or with clear plastic curtains may be a suitable alternative and clear sighting of the interior will be a function of the size of the opening and the location of the stockperson (Baxter, 1984a).

The interaction zone

The interaction zone is the most dangerous area in the farrowing pen for the neonate. Only when the sow is lying resting or when suckling, is the danger from overlying reduced. It is however impossible to prevent the development of dangerous situations as many of these are unpredictable and in some cases unique. It is clear however that certain activities are associated with the greatest risk to baby pigs. In the interaction zone, the high risk sow behaviours are as follows:

- (1) the sow lying down;
- (2) the sow standing up;
- (3) the sow moving about in the pen.

The first two activities occur in all farrowing pens, whilst the third is limited in those systems where the sow is tethered or confined to a crate.

In the transitional movements between standing and lying and vice versa, the danger of crushing piglets is compounded by the proximity of walls and other hardware e.g. crates. This is particularly unfortunate as piglets appear to prefer the convenience, comfort and/or security of lying against or close to walls, sow, etc. Welch (1986a) has shown that piglets have a maximum suckling response to warm and soft surfaces at and above 34°C but prefer warmer surfaces to be against, even up to 60°C. Rapid movements by the young pig which might help escape from dangerous situations may be impeded by smooth or irregular floors. All dangerous situations may be worsened if the piglets are not easily seen or sensed by the sow e.g. piglets buried in deep straw.

Dangerous situations are compounded if the sow acts suddenly and violently. Slow, gentle, deliberate movements of a healthy sow are readily tolerated provided that enough suitable space for the movement is allowed.

Detailed studies have been made of the changes in posture resulting from the standing up and lying down behaviour of the sow (Schwaller, 1981; Clough, 1985). Using these data, Baxter and Schwaller (1983) have demonstrated that many designs of farrowing crate are inadequate to cope with the postural changes of sows. Whilst most sows can adapt their behaviours to suit these confined conditions, such enforced adaptations may only increase the risk of injury to sow and piglet.

In voluntary pens, general locomotor behaviour appears to cause few problems unless the space is severely restricted, the piglets are buried in straw or the sow is awkward or hurried in her movements. Violent or awkward movements may be the result of muscle fatigue, arthritic joints or injury caused by previous confinement. In large pens piglets may be trampled or crushed as a result of the playful actions of an exuberant sow.

The structure of floors in farrowing pens

The floor structure of modern farrowing pens is designed to facilitate the activities of stockpersons rather than to provide an ideal environment for the pigs. Floor design is a compromise: A floor suited to the needs of the pig would be unsuited to those of management.

There are a number of factors which interact and influence decisions on the choice of floor in piggeries and these have been discussed by Baxter (1984a). A good floor should not be a contributory factor to injuries, disease, distress and discomfort or inconvenience (Baxter and Mitchell, 1977).

Floors do not usually contribute directly to neonate mortality. However as a result of causing injury, chilling or impeding rapid escape from dangerous situations, they do contribute indirectly to the death of young pigs.

There is evidence of a wide range of injuries sustained by pigs on both solid and perforated floors. Local necrosis of the skin of young suckling pigs is commonly attributed to trauma from concrete floors with lesions appearing most frequently on the knees, fetlocks, hock, elbows and coronets (Penny *et al.*, 1971; Stanislaw, 1971). Necrotic lesions seem to occur within hours of birth with bruising of the feet on the sole/heel junction having been observed in neonates of less than 24 h of age. The extent of these injuries appears to be largely determined by the roughness of the floor in the farrowing pen. Clark (1985) using a rubber block drag test to measure abrasiveness found that newly installed concrete floors decreased in abrasiveness by 88% in a period of four months. He also found that the number of sole injuries inflicted by the floor diminished as the abrasiveness decreased. Necrosis of teats is also a fairly common occurrence with piglets apparently unaffected at birth showing necrosis one day later (Smith, 1978). Piglets on flattened expanded metal perforated floors have developed teat and knee necrosis and bruising and erosion of the sole, heel and

accessory digits (Smith and Mitchell, 1976). Clark (1985) found that perforated metal slabs and plastic coated expanded metal inflicted more side injuries than a more abrasive solid concrete floor. Injuries to accessory digits, coronets and parts of the upper foot and leg have resulted from the foot passing through the voids in the mesh panels. Using data from Mitchell and Smith (1978), Baxter (1984a) has suggested that the size of the perforation in a farrowing pen floor should not exceed one half of the width of the piglet's foot and might be expressed in relation to body weight (W kg) as $5W^{0.33}$ (mm). Detailed studies by Webb (1984) who considered peak hoof pressure and hog compressive strengths, suggest that highly profiled floors with a large percentage of voids (>8%) are not suitable for pigs. Whilst many of the early injuries subside by the age of 14-20 days (Smith and Mitchell, 1976), it would appear that piglets may be inhibited in their movements at a time when they are also vulnerable to other environmental assault and this is likely to contribute to the cause of mortality in the first 48 h after parturition.

Floors also contribute to disease in farrowing pens by providing an environment for the survival of pathogenic organisms and a continuous source of viable organisms in close proximity to the pigs. Perforated floors help to remove the bulk of faecal and urinary material from the floor surface but they do not eliminate the breeding ground of bacteria and flies etc.

When resting, pigs lose heat to the floor. Depending on the environmental conditions, pigs may have between 8 and 20% of their body surface in contact with the floor (Grommers *et al.*, 1970). Neonates are particularly susceptible to chilling and so they should be housed on floors which are either well insulated or of neutral thermal value. Solid insulated floors should have the insulant as near to the surface as is practical. Clean, dry, straw bedding on the floor surface is an ideal insulant. For neonates weighing 2-5 kg, a floor with an Rf_{45} value (Rf_{45} = thermal resistance of a floor as measured by a thermal simulator of a 45 kg pig) of $0.25^{\circ}\text{C m}^2/\text{W}$ would be regarded as a neutral floor, one where the pig loses no more heat when resting than when standing (Bruce, 1979). From several floors which were tested by Bruce (1977), only wooden slats 58 mm wide, 10 mm gap and 70 mm deep achieved this standard together with some solid floors bedded with sawdust or straw.

Slipperiness and abrasiveness are two other characteristics of floor surfaces which have an important influence on the behaviour and welfare of pigs. Whilst various methods have been devised to measure these characteristics and some useful progress has been made in our understanding of floor characteristics, injury and piglet preference (Webb and Nilsson, 1983), a satisfactory model of the interrelationships is still not available.

Conclusions

Environmental factors make a significant contribution to neonatal mortality. The environment of the neonate includes the structure of the pen and its fittings, the thermal and chemical constituents of the climate of the pen and any sub-climates in creep boxes etc. and the social and physical contributions of the sow and the remaining littermates.

The total space in farrowing/lactation pens has been getting smaller and further reductions may add to the causes of neonatal mortality. The space in a pen can be divided into safe zones and interaction zones, the latter being the only source of sow/piglet interactions and hence the only place for mortality resulting from crushing or overlying. Piglets should be encouraged to use safe zones at all times except when suckling. Safe zones should be large enough to contain all piglets in their preferred huddling/grouping arrangements and an area of up to $0.045 \text{ m}^2/\text{piglet}$ may be required. Safe zones should be thermally enhanced.

Within the interaction zone, the sow's environment should encourage her to stand up and lie down with the minimum of violent and sudden movements and in a way which minimises the risk of injury to neonates. Where sow space is limited to a crate, it must be large enough and of the correct geometry to allow the sow to stand up and lie down in her preferred manner and with the minimum of discomfort. The interaction zone should be surrounded by a safe zone and there should be no obstructions at the boundary of the interaction and safe zones.

The floor of the safe zones should be insulated where solid or, where perforated should have an $R_{f_{45}}$ value of $0.15-0.25^{\circ}\text{C m}^2/\text{W}$. The size of the voids in the floor should be related to the foot size of the piglets. The distinction between slatted, decussated (e.g. woven mesh) and terebrated (e.g. perforated metal) floors should be noted when establishing dimensional criteria (Baxter, 1984a).

NEONATAL MORTALITY: THE INFLUENCE OF MATERNAL BEHAVIOUR

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Introduction

There is little doubt that a major function of maternal behaviour in pigs is to minimize neonatal mortality. From descriptive ethological studies of wild swine (Gundlach, 1968), and feral (Hanson and Karstad, 1959) and domestic pigs (Jensen, 1986) in naturalistic environments, the main elements of maternal behaviour relevant to neonate survival would appear to be:

- (1) the selection of the birth site, and behaviours involved in scraping a hollow at the chosen site, gathering materials and constructing a nest;
- (2) farrowing, including the acceptance of young, suckling and defence of the nest and/or the litter.

While it is not too difficult to envisage how these behaviours have survival value for the neonate in naturalistic environments, we may consider the performance of some behaviours to be irrelevant to neonatal survival in modern farrowing accommodation. Alternatively, by restraining the sow in a crate, key aspects of maternal behaviour may be affected such that it is improperly directed (e.g. savaging occurs) or may not occur at all (e.g. sow prevents neonates from suckling).

Potentially, maternal behaviours can affect neonatal mortality in a number of ways, and although the causes may not be mutually exclusive, they include savaging, crushing, starvation and illness. Sow experience or age, and her nutritional, health and injury status may also affect the occurrence of mortality. Furthermore, while certain maternal behaviours may not have direct effects on neonatal mortality, there are potential consequences for piglet morbidity and growth.

What is maternal behaviour?

We may speculate that "good" maternal behaviour for sows in farrowing crates might be for the sow to lie quietly on her side during the entire parturition (Fraser, 1983/84) and the subsequent 10 h or so, when "neo-nursing" occurs (Lewis and Hurnik, 1985). Thereafter, whenever changing posture, the sow would take extreme care particularly if piglets are within the sow-piglet interaction zone (Baxter, 1984a). While this may well be the ideal, it rarely occurs in practice.

What then is porcine maternal behaviour and how does it influence neonatal survival? To answer this it is first necessary to define maternal behaviour in pigs. A working definition might be based on either the species-specific behaviours reported for domestic sows in naturalistic environments (e.g. Jensen, 1986), or alternatively be restricted to those behaviours reported for sows in farrowing crates in intensive production systems. For the purposes of this paper it is practical to choose the former and to broadly define maternal behaviour as all behaviours of the sow involved in (1) selection of the birth site, (2) construction of the nest, (3) delivery and acceptance of the young, (4) suckling and (5) defence of the birth site and/or litter.

The function of maternal behaviour for neonate survival

Birth-site selection behaviour

According to Jensen (1986) the first behavioural changes indicative of farrowing in domestic sows in naturalistic environments occurred 2-2.5 days before parturition. During this period sows leave the group and become more active. Sows selected a birth site up to 550 m from the common group nest of the herd (Jensen, 1986, 1989; Jensen and Redbo, 1987). Usually, nests were sheltered by a roof of branches to provide protection from weather and were situated in or just below a slope (Jensen, 1986; Jensen *et al.*, 1987), possibly to provide a view of potential approach paths of predators (Jensen, 1986).

Sows in commercial piggeries are unable to perform birth-site selection behaviours, but will if given the opportunity. For example, Hunt and Petchey (1987) reported that sows which farrowed indoors in a large pen showed a strong preference for a hut with roof and sides as compared to more open situations. These authors found that the minority of sows which did not farrow in the more protected situations, selected sites against vertical surfaces in preference to open positions. This finding is supported by the results of Haskell (1989). Thus, while birth-site selection may be functional for neonatal survival in naturalistic environments, and commercial sows may still exhibit the motivation to perform the behaviour, it would seem to be irrelevant in intensive farrowing accommodation where climatic conditions are controlled and predators are absent. However, there is no information on the effects of inhibiting this behaviour and/or denying pre-partum isolation on the performance of other maternal behaviours and neonatal mortality in the intensive situation.

The performance of nest-site selection behaviour together with the increased restlessness before farrowing probably facilitates pre-partum isolation from other pigs (Curtis, 1970; Jensen *et al.*, 1987), and provides for a generally safe, undisturbed farrowing environment. The function of this may be to ensure an undisturbed labour, since disturbance results in suppression of myometrial activity, thus prohibiting the course of labour and expulsion of the foetuses (Naaktgeboren, 1979). Further, Jensen (1989) reported that neonatal mortality was significantly higher in nests that were located less than 100 m compared to more than 100 m from the common group nest, particularly during winter (34.1 vs 15.7% of liveborn piglets, respectively).

It has long been recognized that farrowing sows in commercial production need seclusion with minimal disturbance, which they are more likely to get in a smaller building (Sainsbury, 1963). More recently (e.g. English *et al.*, 1977) the emphasis has changed to providing adequate opportunity for supervision of farrowing sows by increasing access to, and visibility of, the sow by the stockperson, but without causing undue disturbance. Further, the effects on neonatal mortality of the design of the modern farrowing house containing a large number (e.g. greater than 100) of farrowing crates in the one room, which may be brightly lit and which experiences a high level of human activity, are not known.

Nest building behaviours

While questions of whether the performance of nest building behaviours, the necessity for straw bedding or the perception of a completed farrowing nest are relevant to sow welfare (Baxter, 1981b; Barnett and Hutson, 1987), the function of the behaviours in relation to neonatal survival is not clear. The relevance of sows making a farrowing nest in naturalistic environments is questioned, since even though nests are constructed in an appropriate site and manner, neonatal mortality rates are still high (Jensen, 1986 {20.7%}; Jensen, 1989 {15.7% of liveborn piglets}). Reports of neonatal mortality amongst wild swine are even less known. Kirkwood *et al.* (1987) reported that 21 of 89 liveborn piglets (25.8%) died in the first week. A second question relates to the function of nest building *per se*.

The nest building behaviour performed by domestic sows in naturalistic environments commenced immediately after the wandering (restless) stage. Jensen (1986) has described the behaviour in some detail. Briefly, sows started by digging a shallow oval-shaped hole which was filled with grass and other soft materials, initially scratched from the edge of the hole, but after a short time was gathered from the surrounding area. Occasionally the sow rooted in the grass pile to produce a central depression. Some sows completed the nest with branches torn from small trees and bushes. More elaborate nests were built in colder weather (Jensen, 1989).

Sows which farrow in crates with or without straw bedding are reported to perform behaviours similar to nest building behaviours such as rooting and pawing (Baxter and Petherick, 1980; Lammers and de Lange, 1986). The incidence of these activities shows a steep increase beginning about 30-40 h with a peak about 6-12 h prior to the delivery of the first piglet. Thereafter the incidence declines to a low level and finally ceases 1-2 h after the birth of the first piglet (Vestergaard and Hansen, 1984).

Hutson (1988), using operant conditioning techniques found that sows had an apparent low motivation towards straw presented in a box, suggesting that straw *per se* was not a key stimulus to the performance of nest building behaviours. Preliminary results from a current study (Cronin, unpublished data), indicate that the provision of straw bedding appeared to stimulate the performance of nest-building behaviours. Further, there was an inverse association, although non-significant, between the amount of these nest-building behaviours during the 24 h prior to parturition and the duration of parturition. The importance of a short parturition to reduce incidence of stillbirths (most of which die during the process of parturition) has been established (Sprecher *et al.*, 1975). Vestergaard and Hansen (1984) reported effects of restraint on duration of parturition, but did not report on the correlation between nest-building behaviour and duration of parturition. Sows which farrowed in crates and/or which were housed in tether stalls during pregnancy, took about 100 min longer to farrow than sows which were loose housed during pregnancy and at farrowing. All sows in the study had straw bedding.

How the performance of specific nest-building behaviours influence neonatal mortality is not known. Recent research by Cronin and van Amerongen (unpublished data) investigated the effects of providing nulliparous sows with a simulated farrowing nest on maternal behaviour and mortality and growth of piglets. Sows farrowed in either conventional farrowing crates (control) or in identical crates modified with a hessian cover across and down both sides of the cage and with straw bedding on the concrete floor. Compared to the control sows, sows in the modified crates with the simulated nests tended to perform more suckling behaviour (mostly "neo-nursing") during observation periods on the first day of lactation (mean 15.0 vs 32.60% of observation time, respectively) and were more responsive to the distress vocalizations of their piglets throughout lactation. Further, litters of sows in the modified treatment had fewer pre-weaning mortalities (7 vs 0 mortalities/treatment, respectively) and the causes of death in the control treatment were savaging by the sow (4 piglets), overlying (2 piglets) and illness (1 piglet). In addition, there was a tendency for piglets in the control treatment to have a slower growth rate to weaning (5.57 vs 6.23 kg).

Thus, there is some evidence that the pre-farrowing environment influences neonatal mortality and piglet growth. Whether the effect is via the performance of nest-building behaviours or the perception of a "suitable" farrowing environment is not understood.

Parturition and early lactation

In the last hours before parturition, sows become less active and may remain laterally recumbent (Jones, 1966; Randall, 1972). According to Randall (1972), 93% of births occurred while sows occupied a side-lying posture. Nevertheless sows are

often not passive during parturition and will get up frequently to change lying position and to sniff and grunt at their newborn piglets (Jensen, 1986). Presumably this initiates the process of acceptance of the young. According to Jeppeson (1984), the newborn piglet instinctively emits a "huffing or quacking" sound in response to the appearance of the sow's head. While this sound may inhibit any aggression from her, disturbance of this stimulus-response mechanism may result in avoidance of the newborn by the sow and/or threats or actual savaging attempts by the sow.

The neonate however is very attracted to the sow's udder (Welch and Baxter, 1986), so much so that Petherick (1982/83) found that neonates only sought the heated creep area if the udder was obstructed. The motivation of piglets to reach the udder and remain there is strong. With the udder fully exposed, neonates can gain access to the continuous supply of colostrum available during the initial 10 h or so. The intake of colostrum, particularly in the first 4 h, influences both the probability of survival and efficiency of growth later in lactation (Hartsock and Graves, 1976; Robertson and McCartney, 1980; Varley *et al.*, 1987). Thus to optimize neonatal survival it is essential that sows adequately perform those specific behaviours which facilitate the transfer of colostrum to the piglets.

Two types of suckling behaviour are reported for pigs. The initial form of suckling occurs during and shortly after parturition, is asynchronous and continuous and functions to optimize the transfer of colostrum to the neonates. Following this, cyclic bouts of suckling occur. These bouts are synchronous in nature with a 40-60 min period (Whittemore and Fraser, 1974; Fraser, 1980). There are five phases of suckling behaviour (see Fraser, 1980), and, according to Fraser (1980), the interaction sequence involved in the suckling process has probably evolved to facilitate equal distribution of milk to piglets in the litter and thereby to maximize the potential for survival of individuals.

During the first two days of lactation, sows show a preference for lying with their udder towards the heat source (Titterington and Fraser, 1975). In crates, neonates then may receive the advantage of remaining at the udder while also benefiting from the creep heater. Although time spent at the udder may be important to the intake of colostrum (Fraser, 1984), the neonate remains at risk of being crushed. Careful movements on the part of the sow are therefore required in order that neonates are not overlaid. Although crates have been designed to reduce the incidence of piglet mortality due to crushing, concern has been expressed that the lack of appropriate stimuli reduces the sow's level of maternal care and attention for her litter. Cronin and Cropley (1989) however, found that recently farrowed, primiparous sows in crates responded to piglet distress vocalizations and/or a simulated piglet placed under their udder by taking longer to descend from standing to belly lying.

As mentioned earlier, the pre-farrowing environment influenced the performance of suckling behaviour by sows during early lactation (Cronin and van Amerongen, unpublished data). While these authors also reported concomitant effects ($P > 0.05$) on growth to weaning, Metz and Oosterlee (1980) reported differences in the level of transfer of passive immunity associated with the farrowing environment. Sows farrowed in either a standard farrowing crate without straw bedding or in a large pen bedded with straw. During the neonatal period, piglets in the straw-bedded pen treatment spent about 60% of their time lying at the sow's udder, whereas piglets in the crate treatment spent less than 10% of their time there. The 40% greater level of transference of passive antibodies to piglets in the straw bedded pen treatment was probably related to increased suckling behaviour in that treatment. The function of maternal behaviours elicited by the environment prior to farrowing, are clearly not well understood, but conceivably hold the key to reducing neonatal mortality and improving piglet health and growth.

While the question of providing straw to sows pre-farrowing is not new, a current study (Cronin, unpublished data) is utilizing straw to help elicit a range in the performance of maternal behaviours with the aim of relating this to piglet survival and growth. Preliminary results suggest that the provision of straw bedding affected the performance of maternal behaviours and, in particular, resulted in increased suckling behaviour in early lactation compared to sows which farrowed without straw bedding (4.8 vs 3.3% of observation time, respectively). Ironically in this study there was little effect of straw bedding on neonatal mortality; rather neonatal mortality was mainly influenced by restraint of the sow and/or area available to the neonate. Litters of sows farrowed in crates compared to pens had fewer neonatal mortalities (7.2 vs 14.9% of liveborn piglets in crates and pens respectively, died in the first 72 h). The number of piglets dying over each of the first three days, and the primary cause of death are shown in Figure 1. While the provision of straw and/or space present the peri-parturient sow with the opportunity to perform relevant maternal behaviour, there was no corresponding reduction in piglet mortality levels (7.3 and 7.2% of liveborn piglets died in the first 72 h, respectively, in crates without and with straw bedding). Straw bedding however tended to reduce the level of neonatal mortality in pens (16.8 and 12.9% of liveborn piglets died in the first 72 h, respectively, without and with straw bedding).

The results presented in Figure 1 show that neonatal mortalities due to savaging are at a significant level. While this appears to be attributable to the fact that predominantly gilts were used in the experiment, it is clear that non-restrained gilts savaged more piglets than restrained gilts. According to English *et al.* (1977), a high proportion of gilts attempt to savage at least their first piglet. By housing gilts in crates to restrict movement, gilts that attempted to savage their piglets were less able to inflict serious wounds. The reasons which prompt a sow to attack a newborn piglet or the litter are somewhat obscure but Pomeroy (1960) suggested pain and fear were involved. Observations from video records of savaging incidents tend to support this (Cronin, unpublished data).

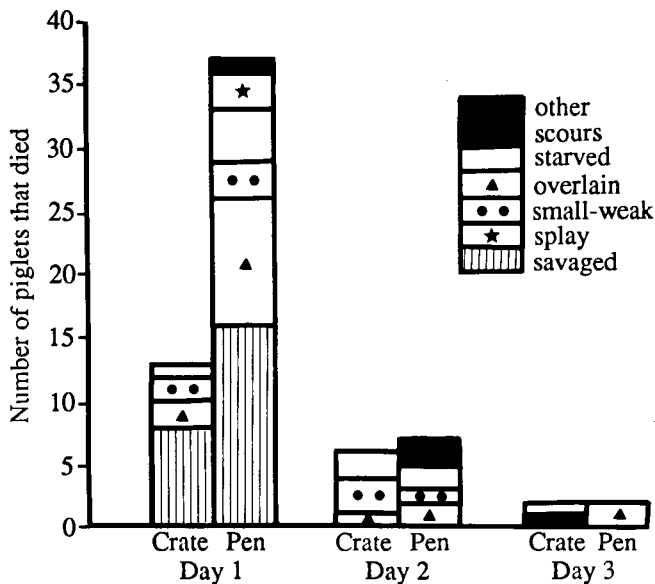


Figure 1. The incidence of neonatal mortality and causes of death in litters farrowed in crates and pens (32 litters/treatment).

Defence of the nest site/litter

According to Pomeroy (1960), most recently farrowed sows will allow almost anyone except a complete stranger to handle their piglets, providing they do not make them squeal. If a piglet squeals most sows will violently attack anyone. Further, Newberry and Wood-Gush (1985) found that in their pig park (a 2.3 ha hilly enclosure in Scotland, containing running streams, open grassy areas, gorse-covered areas and a stand of pine trees), any disruption at the nest site could result in the sow threatening or chasing an intruder (whether it be another pig or a human observer) even during milk ejection. Thus one main reason for restraining sows in crates around farrowing was to reduce the chance that the sow attacks the stockperson. Clearly while the function of nest/litter defence to reduce neonatal mortality from predators or other pigs is no longer appropriate in the controlled environment of the intensive piggery, the effects of culling sows which showed aggression to humans after farrowing (in defence of the nest site/litter) on maternal behaviour in modern sows is not known. However, although the incidence of sows aggressive to humans after farrowing has not been reported, the use of farrowing crates appears to have helped to reduce it. Whether this is due to selection against aggressive sows in breeding programs or an effect of an inappropriate pre-farrowing environment, is not known.

Conclusions

Maternal behaviour in pigs is clearly influenced by the pre-farrowing environment, and does not appear to have been "bred out" of the modern sow through selection for high production in an intensive environment. It is unclear whether the performance of maternal behaviours affect neonatal mortality in farrowing crates, since sows are unable to perform much of the species-specific repertoire that comprises porcine maternal behaviour. There is however, a paradox: While the farrowing crate may provide a lack of appropriate stimuli prior to farrowing, and this may dampen the performance of maternal behaviours compared to domestic sows in naturalistic environments, the level of neonatal mortality reduced by farrowing sows in crates.

For modern pig production though, the most important maternal behaviours would appear to be duration of parturition and suckling behaviour (particularly early in lactation), as these factors affect the fitness, morbidity and growth of the neonate. Further research is clearly required on the factors affecting the development of maternal behaviours relevant to intensive farrowing accommodation.

NEONATAL MORTALITY: THE INFLUENCE OF LACTATION ON PIGLET SURVIVAL

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Introduction

Causes of death in suckling piglets have been well reviewed (Fahmy and Bernard, 1971; Edwards, 1972; Dyck and Swierstra, 1987). A significant proportion of all liveborn piglets (up to 25%) do not survive the lactation period. About half of these piglets die within the first 72 h after birth and later deaths are often a consequence of events which occurred in the first hours of life. Most of the liveborn piglets which die during the lactation period could have survived in a favourable environment, and it is now apparent that the deprivation of colostrum and milk, rather than some innate lack of vigour, has a major influence on the survival rate of neonatal piglets (Hartsock and Graves, 1976). Colostrum and milk provide the neonatal piglets with both immunological protection against pathogenic micro-organisms, and the energy supply that is required to prevent hypoglycaemia.

Protection against microbial infection

Although mammalian neonates (including piglets) are immunocompetent at birth, their isolation in the uterus from environmental antigens has meant that, in terms of protection against microbial infection, neonates are relatively immunodeficient (Murgita and Wigzell, 1981). Consequently, various mechanisms have evolved to allow the passive transfer of humoral immunity from the mother to her offspring (Guidry, 1985). Since there is minimal placental transfer of maternal humoral immunity to the pig foetus throughout gestation, the piglet is agammaglobulinaemic at birth and must obtain its passive immunity from maternal immunoglobulins (IgA, IgG and IgM) secreted into colostrum (Kruse, 1983). Furthermore, the ingestion of immunoglobulins in milk during established lactation provides defence against possible enteric infections in the suckling piglet (Porter, 1981). The piglet also obtains innate protection from "non-antibody factors" (e.g. lactoferrin, lysozyme and lactoperoxidase) in colostrum and milk (Reiter, 1978). Therefore, failure to ingest colostrum and milk predisposes the piglets to infection from pathogens which may exist in the farrowing crates and, in many piggeries, a high percentage of these piglets will die before they reach weaning age.

Innate protection

It has become clear that there are other "non-antibody factors" in sow's colostrum and milk which either enhance the activity of the antibodies or provide a certain amount of protection against pathogens (Reiter, 1978). The concentration of lactoferrin, an iron-binding protein which non-specifically inhibits bacterial growth, was reported by Elliot *et al.* (1984) to be high in colostrum (1.1-1.3 g/l) and then to decrease rapidly over the first week of lactation. Milk also contains other antimicrobial proteins (Reiter, 1985). Lysozyme catalyses the hydrolysis of the glycosidic linkages [$\beta(1-4)$] between the N-acetylglucosamine and N-acetylmuramic acid in the cell walls of certain bacteria and is known to have bactericidal activity in association with other milk components, particularly IgA. Furthermore, lysozyme has an anti-inflammatory action by inhibiting neutrophil chemotaxis and the generation of free radicals. Lactoperoxidase activity is found in milk as well as in saliva. The importance of the activity of

lactoperoxidase in the gastrointestinal tract of the young is unknown.

A variety of cell types has been identified in sow colostrum and milk by Lee *et al.* (1983) including neutrophils, macrophages, epithelial cells, eosinophils and lymphocytes. The presence of these phagocytic and lymphoid cells in colostrum and milk may help to provide protection against infection for the mammary glands of the sow and the gut of the neonatal piglet.

Immunological protection

The concentrations of all the colostral immunoglobulins are high at birth and decline during the first 24 h after parturition. The decline is very rapid initially as Bourne (1969a) has shown that the concentration of whey protein in colostrum declines to 50% of initial levels within 4-6 h after birth of the first piglet. Furthermore, the quantity of immunoglobulin in colostrum has been shown to increase with parity and is influenced by factors such as breed, vaccination, season and farming practices (Inoue *et al.*, 1980; Inoue, 1981). Whereas IgG is the predominant immunoglobulin in colostrum (80%), the predominant immunoglobulin in milk secreted throughout lactation is IgA (70%). In contrast to colostrum, more than 90% of IgA and IgM, and almost 70% of IgG in sow milk is synthesized locally in the mammary glands and there is abundant evidence indicating that a large proportion of the plasma cells which secrete immunoglobulin into milk are derived from B-lymphocytes originally activated to pathogens in the intestinal tract. Thus, the antimicrobial immunity in colostrum resembles that of the maternal blood vascular system, whereas that in milk during established lactation more closely resembles that of the local immunity of the gut (see Hartmann and Holmes, 1989).

Large quantities of immunoglobulins, along with other colostral proteins, are absorbed intact across the small intestine of the piglet during the first 12-24 h of life. Throughout this time the mucosal cells in the intestine, which are mostly of foetal origin, are capable of transporting colostral immunoglobulins into blood by way of the lymphatic circulatory system. By 24-36 h after birth the intestinal tract of the newborn piglet undergoes "closure" and the transport (endocytosis) of macromolecules ceases (Westrom *et al.*, 1984). Changes associated with this closure develop along the small intestine at different times after birth, with transport terminating in the duodenum, jejunum and ileum at about 2 h, 2 days and 3 days after birth, respectively (Murata and Namioka, 1977). After gut closure, the immunoglobulins in ingested milk continue to provide local protection against microbial pathogens in the gastrointestinal tract of the piglet.

The presence of proteolytic enzymes (such as pepsin and trypsin) in the gastrointestinal tract has an influence on the quantities of colostral immunoglobulins absorbed by the piglets. Stone *et al.* (1979) demonstrated that IgA, and to a lesser extent IgG₂, were the most resistant of the porcine immunoglobulins to these proteolytic enzymes. A specific trypsin inhibitor (sow colostrum trypsin inhibitor) was observed at high concentrations (1.3 g/l) in the colostrum at parturition, and then rapidly decreased to very low concentrations (0.04 g/l) over the first 2-3 days of lactation (Laskowski *et al.*, 1957; Jensen and Pedersen, 1979). It has been proposed that the sow colostrum trypsin inhibitor plays a role in protecting the colostral immunoglobulins from proteolytic digestion during the first day or two after birth (Jensen and Pedersen, 1982). Indeed Carlsson *et al.* (1980) observed that the absence of the trypsin inhibitors decreased the intestinal absorption of total colostral proteins in neonatal piglets by 35%. Westrom *et al.* (1985) showed that there was a more efficient transfer of macromolecules into the blood of neonatal piglets fed colostrum with a high protein content compared to those fed a 200 g/l protein solution. They suggested that, in addition to the protease inhibitors and protein content of colostrum, a number of other unknown factors also may enhance the intestinal uptake of macromolecules in the neonatal piglet.

The systemic immunity of the neonatal piglet invariably reflects that of the sow, particularly as a large proportion of the colostral immunoglobulins originate from the maternal circulation (Bourne and Curtis, 1973). Serum concentrations of the acquired immunoglobulins reach peak values in the newborn piglets 12-24 h after birth, with the predominant class being IgG (Porter and Hill, 1970; Curtis and Bourne, 1971). Thereafter, the serum concentrations of IgG, IgA and IgM declined with half lives of 10, 2 and 3 days, respectively (Klobasa *et al.*, 1981). Minimum concentrations of serum IgG, IgA and IgM occurred at about 5, 3 and 2 weeks, respectively, after which time a rise in serum levels indicated that the piglets had established endogenous synthesis of immunoglobulins (Klobasa *et al.*, 1981). Variations in the mean concentrations of serum IgG between different litters were found by De Paille *et al.* (1988) to be associated with differences in the yield of colostrum from the sows. Furthermore, they suggested that litters with low concentrations of serum IgG during the first 12 h of life may be used to select out sows with low milk production.

In order to improve neonatal survival, effective strategies have been employed in many piggeries to actively immunize sows against virulent pathogens to which piglets are particularly susceptible, including enterotoxigenic strains of *E. coli* (Kohler *et al.*, 1975; Chidlow and Porter, 1979; Moon, 1981; Moon *et al.*, 1988), transmissible gastroenteritis (TGE) virus (Bohl and Saif, 1975; Stone *et al.*, 1977) and Aujeszky's disease (McFerran and Dow, 1973; Iglesias and Trujano, 1989). The aim of these immunization strategies was to stimulate an increase in the proportion of maternal antibodies in colostrum and milk directed against the particular pathogen. For example, it has been recommended that sows are vaccinated against enterotoxigenic *E. coli* by the oral administration of antigen given daily from 60 days pre-partum until parturition, along with an intramuscular injection of antigen between 18 and 25 days prepartum (Chidlow *et al.*, 1979). This combination of oral and parenteral immunization of the sow provided the piglets with adequate local and systemic protection against the *E. coli* pathogen. Furthermore, the immunization of sows against enterotoxigenic strains of *E. coli* helped to control the spread of infection throughout the herd by reducing the levels of pathogen in the excretion (Chidlow and Porter, 1979).

Exposing piglets to a cold temperature after birth reduced their acquisition of colostral immunoglobulins and resulted in an increased rate of mortality (Belcha and Kelley, 1981). Cold stress does not decrease the efficiency of absorption of colostral immunoglobulins (Kelley *et al.*, 1982), but may cause the piglets to be less vigorous during suckling which would lower their intake of colostrum. Subjecting sows to extremes of temperature in late pregnancy also can effect the acquisition of immunoglobulins by newborn piglets. An improved absorption of IgG has been observed in piglets reared on sows exposed to a cold temperature (5°C) during late pregnancy (Bate and Hacker, 1985a,b). These sows had elevated blood cortisol during cold stress and it was suggested that the maternal glucocorticoids may have mediated the increased absorptive capacity of IgG in the piglets. On the other hand, Machado-Neto *et al.* (1987) observed that sows exposed to moderate heat stress (32°C) during late pregnancy also had elevated concentrations of blood cortisol, but the concentrations of total protein and IgG in their colostrum were suppressed. Furthermore, the piglets reared on heat-stressed sows had low concentrations of blood IgG during the first three weeks after birth.

Coalson and Lecce (1973) reported that 15% of piglets that were prevented from suckling until 4 h after birth had extremely low levels of serum immunoglobulin. Therefore, it is not surprising that piglets born at the end of farrowing, especially where the litter size was more than eight, were found to have much lower concentrations of serum IgG than their earlier born littermates (De Paille *et al.*, 1988). Although Bourne (1969b) suggested that piglets would have equal access to colostral immunoglobulins if they were removed immediately after birth, and simultaneously returned to

the sow at the end of farrowing, Hendrix *et al.* (1978) demonstrated that such a practice made no difference to either the levels of immunoglobulins or survival rate at 3 weeks of age.

The behaviour of the piglets during the first 24 h after birth also has a major influence on their consumption of colostral immunoglobulins. Individual piglets develop a preference for a particular teat within the first few hours after birth (McBride *et al.*, 1965). Piglets were seen to be quite aggressive towards each other as they were selecting teats (Hartsock and Graves, 1976), but once a teat order was established the level of fighting decreased. A number of studies have reported that piglets with low birth weights competed with their larger and heavier littermates for teats during suckling bouts, and consequently ingested less colostrum (Hendrix *et al.*, 1978; Milon *et al.*, 1983; De Pastille *et al.*, 1988). Since larger litters result in both smaller piglets and a greater amount of fighting for teats, Hartsock and Graves (1976) suggested that maximizing the size of the litters would lead to more piglets dying before they reached weaning age from a combination of inadequate passive immunity and starvation.

Energy balance

Newborn piglets have poor insulation due to a sparse hair covering, a lack of subcutaneous fat (Mellor and Cockburn, 1986) and a high surface/mass exposure (Le Dividich and Noblet, 1983). They are usually housed in a moderate environment (18-26°C) and, despite huddling together with their litter mates, they need to increase their heat production to avoid hypothermia. Shivering thermogenesis is the predominant source of heat, and the energy required for heat production is derived both from the piglets' body reserves and from colostrum (Mellor and Cockburn, 1986). The major energy substrates for heat production are carbohydrate and lipid because protein metabolism is minimal during the first day after birth.

The newborn piglet has a total fat content of 1-2% of body weight of which about half is structural fat and therefore unavailable as an energy source (Le Dividich and Noblet, 1983). Thus the well-being of the newborn unfed piglet is predominantly determined by the availability of carbohydrate and not lipid (Mellor and Cockburn, 1986). However, a limited energy supply can result in both hypothermia and cerebral compromise. In cooler conditions hypothermia precedes cerebral compromise and is the usual cause of death, whereas death resulting from cerebral compromise is more usual in thermoneutral conditions (Mellor and Cockburn, 1986). Foetal growth retardation, caused by either a small placenta or maternal under-nutrition, results in lower lipid levels in piglets but does not affect the quantity of glycogen available to the newborn piglets (Mellor and Cockburn, 1986). Nevertheless, any deficit in lipid availability at birth will result in a greater decline in glycogen reserves by increasing the newborn piglet's use of carbohydrate.

Hypoglycaemia

The concentration of glucose in blood taken from the venae cavae of normal piglets was about 3 mM at birth but increased to over 5 mM by 2 h post-partum (Curtis *et al.*, 1966; Aherne *et al.*, 1969; Pettigrew *et al.*, 1971; Steele *et al.*, 1971). Thus, there is a physiological hypoglycaemia in piglets which is corrected within a few hours of birth. However, it was first reported in the 1940s that an often fatal condition known as "baby pig disease" occurred 24-28 h after birth in piglets and was associated with symptoms such as shivering, lethargy and a weak squeal when disturbed (Graham *et al.*, 1941; Sampson and Graham, 1943). The only biochemical difference between healthy and sick piglets was that the concentration of blood glucose (mean 1.44 mM, range 0.16-3.31 mM) was depressed (Graham *et al.*, 1941). The clinical signs of

hypoglycaemia were observed when the concentration of blood glucose decreased to about 2.7 mM (Pettigrew *et al.*, 1971). Whereas the newborn piglet was susceptible to fasting hypoglycaemia, piglets fed for the first 4 days and then fasted for 72 h did not develop symptomatic hypoglycaemia (Swiatek *et al.*, 1970). This hypoglycaemia results from a failure of the piglet to maintain blood glucose homeostasis by either endogenous glycogenolysis and gluconeogenesis or an exogenous intake of carbohydrate (lactose) from colostrum.

Endogenous glucose

Fasted piglets, maintained at 32-38°C, had severe hypoglycaemia and depleted liver glycogen reserves at about 45 h after birth, whereas in the environment of most piggeries (18-26°C) the liver glycogen was depleted in about 12 h (Mellor and Cockburn, 1986). However in sucking newborn piglets, in a moderate environment, liver glycogen stores were not depleted until 18-24 h after birth (Swiatek, 1970; Pegorier *et al.*, 1981; Pegorier *et al.*, 1984). The depletion of liver glycogen was associated with shivering which provided the newborn piglet with the means of avoiding hypothermia. In the first hours of life the piglet mainly used glycogen stored in the muscles to fuel the shivering process (Mellor and Cockburn, 1986). As the muscle glycogen became depleted, the muscles used blood glucose which was then replaced by glucose from liver glycogenolysis and gluconeogenesis. Since the liver provided about 15% of the available glycogen in piglets, liver glycogen needed to be replenished and gluconeogenesis maintained in order to avoid hypothermia and cerebral compromise (Mellor and Cockburn, 1986). Thus the liver glycogen store is only a short-term back-up for blood glucose homeostasis.

Gluconeogenesis has been estimated to account for 65% of the total glucose entry rate in suckling newborn piglets (Pegorier *et al.*, 1983). The enzymatic capacity for gluconeogenesis was present at birth, but the activities of phosphoenolpyruvate carboxykinase, glucose-6-phosphatase and fructose-1,6-bisphosphatase doubled by 2 days post-partum (Mersmann, 1971). Thus the fed piglet has a substantial capacity to synthesize glucose through gluconeogenesis by the second day after birth. This conclusion is supported by Gentz *et al.* (1970) who showed that the newborn piglet was able to maintain blood glucose at a low but constant level after 48 h of fasting. Furthermore, Flecknell *et al.* (1988) made the important observation that the intake of colostrum by newborn piglets improved the development of the hormonal and metabolic mechanisms involved in the control of blood glucose. In this connection, Pegorier *et al.* (1982) observed that the rate of gluconeogenesis from lactate, pyruvate, alanine, glycerol, dihydroxyacetone and galactose was two to three fold higher in isolated hepatocytes from fed compared to fasted newborn piglets. They concluded that the hypoglycaemia developed in fasted newborn piglets could be explained by this difference. However, the mechanism by which gluconeogenesis is impaired in the hepatocytes of fasted newborns remains unknown.

Exogenous glucose

It has been estimated that the daily intake of lactose accounted for 35-50% of the daily whole body utilization of glucose in sucking piglets (Pegorier *et al.*, 1982). Lactase, the enzyme that hydrolyses lactose to glucose and galactose, had a high activity in the intestinal mucosa at birth and decreased with age (Leibholz, 1986). Whereas the specific activity of lactase declined rapidly over the first week of life (Manners and Stevens, 1972), the total activity of lactase in the intestine reached a maximum at 15 days of age (Leibholz, 1986). Thus the oral administration of lactose resulted in a rise in the concentration of blood glucose from birth onwards (Smith *et al.*, 1988).

Lactase activity is the rate-limiting step in the digestion and absorption of lactose (Kretchmer, 1972). Since intestinal lactase in the foetus increased rapidly in late

gestation (Sprague *et al.*, 1963), premature piglets could have a lower capacity to utilize lactose (Pettigrew *et al.*, 1971). Davidson (1984) noted that, in man, intestinal infection and inflammation decreased lactase activity. Furthermore, intestinal enterocytes were more susceptible to enteropathogens in the younger piglets (Lecce, 1986). This suggests that, even though piglets may continue to suck, a loss of mucosal surface area due to intestinal injury could cause lactose intolerance and carbohydrate malabsorption which may result in hypoglycaemia.

Amylase activity in the pancreatic tissue of piglets was completely absent at birth and the increased activity with age was stimulated by the inclusion of starch in the diet. Both intestinal maltase and sucrase activity were found to be low at birth and increased to appreciable levels by 2 weeks of age (Leibholz, 1986). Thus any disaccharide or polysaccharide other than lactose would not be hydrolysed in the intestine and, therefore, would not be available for the maintenance of blood glucose in the newborn.

Galactose has a different metabolic fate to glucose in several species. In man, absorbed galactose is primarily cleared and metabolized by the Leloir pathway in the liver (Siegal *et al.*, 1988). On the other hand current evidence suggests that much of the absorbed glucose passes through the liver to be metabolized by the peripheral tissues (Katz *et al.*, 1986). Experiments carried out in piglets by Bird *et al.* (1988) supported the concept that most of the absorbed galactose is taken up by the liver while glucose is predominantly taken up by peripheral tissues. Therefore the digestion of lactose by the piglets has the potential to replenish efficiently both liver and muscle glycogen, thereby preventing hypothermia, hypoglycaemia and cerebral compromise.

Conclusions

The acquisition of colostrum and milk is important to ensure that piglets obtain both humoral and surface protection against microbial infections, as well as an adequate external supply of energy to prevent starvation and exposure to cold causing hypoglycaemia and death. However, the benefits derived from colostrum are interdependent. Yaguchi *et al.* (1980) observed that the mortality of heavy piglets with low levels of IgG was similar to that of light piglets with high levels of IgG. Furthermore, De Pastille *et al.* (1988) observed that piglets which died in the first 3 days after birth had both a low level of IgG and weight loss, suggesting that starvation played a major role in the death of these piglets. The factors predisposing the piglets to starvation and hypoglycaemia are associated with the immediate post-partum period and include establishment of teat order, cold stress, failure to suckle, intestinal disease, glycogen stores and the gluconeogenic capacity of the piglet, and lactation failure in the sow. Although the influence of lactation on piglet mortality has been extensively studied, further studies are required to investigate the importance of colostrum intake in relation to piglet morbidity and failure to thrive.

NEONATAL MORTALITY: THE INFLUENCE OF MANAGEMENT

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Introduction

This paper presents pre-weaning production statistics for a range of Australian farms, discusses the benefits of increasing survival and the causes of pre-weaning mortality, and draws from farm studies which have investigated the resolution of pre-weaning mortality problems.

Australian data sets

The knowledge and skills to wean more than 9.5 pigs/litter are available to all Australian pig producers but appear to be applied by relatively few. Data from several sources indicate that average recorded pre-weaning mortality rates on farms range from 11.3-23.5% of piglets born alive (Cleary, 1987; Spicer *et al.*, 1986).

Cost of neonatal mortality

In a study of the cost of disease on Australian pig farms, Cutler and Gardner (1988) provided a guide to calculating the effect of production inefficiencies on profit. They used Auspig and Pigmax computer models to simulate performance and profit for a 100-sow model herd producing 1980 pigs/year.

The model herd produced 10.31 pigs born alive and weaned 8.9. If pre-weaning mortality rate was reduced from 13.6 to 8% and the model herd weaned 9.5 pigs, 10 fewer sows could be carried on the farm for the same production. The feed saving alone for those 10 sows is 12.3 tonnes, a saving of \$2657 for the herd. Alternatively, by constructing extra housing, and using the same resources, the farm could generate additional profit of \$5000 by selling the extra 280 pigs produced which is equivalent to \$50/sow/year. The financial benefits available to producers from improvements in pre-weaning survival are substantial.

Causes of pre-weaning mortality

Spicer *et al.* (1986) described the causes of pre-weaning mortality on a large intensive piggery in Victoria (Table 3). The pre-weaning mortality rate for the herd studied and the cause of death were similar to findings reported elsewhere (Glastonbury, 1976; English and Morrison, 1984a; Svendsen *et al.*, 1986). Further, in additional farm cases investigated by us between 1982 and 1989 (Cutler, Spicer and Prime, unpublished data), similar causes of mortalities have been found. Where mortality rates have been higher or lower than those reported by Spicer *et al.* (1986), changes in the number of pigs dying from overlays, scours or from being small and weak or malnourished have been prominent.

Management approaches to resolving pre-weaning mortality problems

Trauma

English and Wilkinson (1982), Spicer *et al.* (1986) and Svendsen *et al.* (1986) have drawn attention to the importance of trauma as a cause of pre-weaning loss. Spicer *et al.* (1986) found that no pre-existing illness in piglets or sows was found for 56% of the overlain pigs, but the remaining losses were associated with illness in the piglets (26%),

the sow (15%) or both (3%). Pre-existing illness in the sow included inappetence, mastitis, agalactia, purulent vulval discharge, rectal prolapse or aggression. Pre-existing piglet illness included diarrhoea, anaemia, splayleg, weak pigs and pneumonia. The majority of overlays occurred within the first 36 h of life. Most traumas occur when the sow moves to stand up or lie down. About 30% of the traumatic injuries occurred at feeding time (Svendsen *et al.*, 1986).

Spicer *et al.* (1986) determined that savaging of a piglet by the sow accounted for the most significant cause of mortality in parity one litters. Sows which savaged their litter were likely to be those mated at lower body weights (Spicer *et al.*, 1985).

Table 3. Causes of pre-weaning mortality on a large intensive pig farm (Spicer *et al.*, 1986)

Cause of death	% Pigs born	Birth weight
Pre-parturient deaths	2.9	
Parturient deaths	5.4	1.15 ¹
Overlay	2.1	1.31
Scours	1.7	1.27 ¹
Anaemia	1.2	1.24 ¹
Savaged	1.1	1.14 ¹
Non-infectious disease	1.1	1.39
Infectious (other)	1.0	1.24 ¹
Small-weak	0.9	0.82 ¹
Splayleg	0.5	1.24
Nil diagnosis	0.3	1.33
Pre-weaning mortality (% born alive)	11.3	
Total	18.7	18.7

¹Significantly less than the average birth weight for all piglets ($P < 0.05$)

Heat

Crate designs, creep configurations and heater positions influence the time it takes piglets to come under the heat lamp or the amount of time they spend lying under the heat lamp away from the danger zone near the sow. Svendsen *et al.* (1986) and Morrison (personal communication) have demonstrated the importance of heated creep areas in the farrowing crate. In Morrison's experiments different heating-accommodation standards were applied. Lying behaviours and mortality rate were influenced by the treatments. As heating was increased, piglets spent less time lying in the danger zone and mortality rate was reduced from 19.3% to 6.9% in the first 7 days of life. Svendsen *et al.* (1986) reduced mortality rate in the first week of life from 7.0% to 1.5% by providing a moveable heat source.

Morrison (personal communication) and Svendsen *et al.* (1986) demonstrate the importance of properly applied heat. Morrison also demonstrated the effect of farrowing house temperature on survival. When farrowing house temperature was 20.5°C instead of 13.6°C, live weight gain to 7 days increased (169 g/day vs 135 g/day) and 7 day mortality rate fell (10.7% vs 15.1%). Le Dividich and Noblet (1981) reported that colostrum intake was 37% less in pigs reared at 18-20°C compared with 30-32°C. Mortality rate was also substantially higher in the pigs reared at 18-20°C.

English *et al.* (1982) demonstrated the success of "farrowing cradles" to reduce losses from overlays. The farrowing cradles were essentially hinged bars placed parallel to the sides of the farrowing crate. As the sow rose, she pushed the bars upwards, but as her body passed the bars, they fell back to their original position. When the sow came to lie down again, the position of the bars forced her to slow her descent and

gave piglets in the lying area a chance to move to safety. Three-day mortality rate was reduced from 11.1% for controls to 6.2% for the crates fitted with cradles. Muirhead (1987) indicated that crates with movable internal bars were advantageous but that narrow crates about 500 mm wide also assisted superior performance.

Birth weight consideration

Spicer *et al.* (1986) demonstrated that most deaths occurred in piglets which were below the average birth weight. Small piglets (<1.0 kg) took longer to achieve a first suckle than heavier (>1.0 kg) piglets (>86 min vs 38-59 min). The mortality rate for piglets weighing less than 1.0 kg at birth was 33 vs 9.2% for pigs weighing more than 1.0 kg at birth.

Spicer *et al.* (1987), using a heated crib and a colostrum substitute fed by bottle or stomach tube to sick or weak piglets, indicated that first week mortality rates could be kept very low. However, success of the techniques implemented depended on the skill of the farrowing house attendant. Over a 7 week period, one farrowing house attendant lost only 1.3% in the first week of life, while another lost 6.1%. The comparison involved about 1000 litters.

English and Smith (1975) and Fahmy and Bernard (1971) also demonstrated the importance of birth weight variation in a litter. Mortality rate was very much affected by the standard deviation of birth weight within the group rather than the actual birth weight average of the group. Mortality rate in the small pigs was high regardless of weight variation whereas reducing standard deviation of litter weight for the larger pigs reduced mortality rate. Herein, then, is the underlying reason for fostering; fostering is done not only to even up the numbers of pigs per sow, but just as importantly, to equalize the weights of piglets in the litters.

Many authors have demonstrated that piglet birth weight can be increased by increasing feed intake during late pregnancy. Pettigrew (1979) suggested that at least 1 kg of fat should be fed to the sow before farrowing. Moser and Lewis (1981) suggested that feeding fat did lead to an increase in survival, especially in the smaller pigs in the litter. However, Pettigrew (1979) was only able to demonstrate an effect on survival on farms where pre-weaning mortality rate was at least 20%.

Enteric disease

Enteric disease is a small but important cause of pre-weaning mortality (Spicer *et al.*, 1986). In Australia it may be attributed to disease caused by *Escherichia coli*, rotavirus or coccidiosis. Abroad, transmissible gastroenteritis and *Clostridium perfringens* are an issue. By far the most important cause of diarrhoea in the first week of life is *E. coli*. Fahy *et al.* (1987) demonstrated the success of killed *E. coli* vaccines in the prophylaxis of *E. coli* enteric disease (Table 4).

Sanitation

Bille *et al.* (1974) demonstrated that both morbidity and mortality rate associated with gastrointestinal disease was higher in herds with poor hygiene standards. As hygiene standards improved, the prevalence of diarrhoea fell from 28% of litters to 5% during the year in which an organized hygiene program was developed.

Synchronized farrowing

Friendship *et al.* (1986) and Spicer *et al.* (1987) suggested that quality of supervision was a key factor in neonatal survival. Bille *et al.* (1974) demonstrated that increasing the level of supervision during farrowing had a positive effect on neonatal survival.

Table 4. The effect of killed vaccines on *Escherichia coli* disease in neonatal pigs (Fahy *et al.*, 1987)

	Whole cell vaccine	Purified pilus vaccine	Control
Number of sows	97	97	94
Piglets born alive	944	906	902
Litters with diarrhoea (%)	19.6 ^x	21.6 ^x	50 ^y
Scour days	1.3 ^x	1.7 ^x	5.6 ^y
Treatment for diarrhoea/litter	1.4 ^a	1.5 ^a	12.4 ^b
Severity of diarrhoea/litter	0.6 ^a	0.8 ^a	4.1 ^b
Scour related deaths (% of total)	5.0 ^a	6.1 ^a	22.2 ^b

^{x,y}differ at $P < 0.001$ by chi-square test; ^{a,b}differ at $P < 0.01$ by Student's t-test

Induced farrowing using prostaglandins is an effective method of synchronizing farrowing but some caution is worthwhile. Several authors failed to improve piglet survival rates in litters born to synchronized sows. Indeed, piglets born 1-3 days early had lower birth weights and lower survival rates (Downey *et al.*, 1976; Walker, 1977). English *et al.* (1977) reported that piglets induced to farrow on their due date had better survival than controls, whereas those induced to farrow one or two days early had progressively poorer survival.

Field experience in Victoria

Following the work by Spicer *et al.* (1986), efforts were made to apply the results of the study to other farms in Victoria (Prime *et al.*, 1987). One of the major factors contributing to high mortality rates in farrowing houses was lack of attention to detail (Prime *et al.*, 1987). Following an intensive period of staff instruction on 3 farms a 5-7% reduction in neonatal mortality rate was achieved (Table 5). Most of the reduction in mortality rate followed a reduction in overlays. The following measures were instrumental in decreasing neonatal mortality:

- (1) Installation of 100 mm PVC pipes to reduce internal farrowing crate width and slow the descent of the sow. The pipes ran the length of the crate and were suspended by light chains from the top bar of the farrowing crate to rest above the udder of the sow, about 350 mm above the floor.
- (2) Creep area comfort factors. Indoor-outdoor carpet was glued to heavy galvanised metal (350 x 600 mm) and installed in the creep area. The carpet was comfortable, attracted the pigs and did not become excessively hot. In our field studies, excessively high creep temperatures associated with floor heating adjustment or black floors which absorbed heat were just as detrimental as inadequate heating.
- (3) Management of heating systems for the newborn litters. An additional heat lamp was provided toward the rear of the sow during the farrowing period and the immediate 24 h afterward.
- (4) Heated crib for care of sick pigs or a place to house pigs during split suckling sessions.
- (5) Supplementary milk feeding for weak or small pigs or when the pigs in large litters were split suckled and required extra feeding.
- (6) Thorough hygiene programs for the farrowing house and equipment.
- (7) An active and expert fostering program which focused on equalizing litter size and sizes of piglets within the litter.
- (8) Prompt treatments for sick pigs.

Table 5. Changes in neonatal mortality rate following staff training on three farms (Prime *et al.*, 1987)

	Mortality rate (%)					
	Before staff training			After staff training		
	Farm 1	Farm 2	Farm 3	Farm 1	Farm 2	Farm 3
Overlays	7	14.3	5.2	3.5	7.4	2.7
Scours	0.1	0.5	2.5	2.0	0.1	1.2
Small/weak/malnourished	5.8	0	1.5	4.0	0.4	0
Other	6.1	3.25	6.3	2.3	4.6	7.0
Total	19.0	18.0	15.5	11.8	12.5	10.8

Number of sows at farms 1, 2 and 3 were 220, 150 and 240, respectively

Conclusions

The knowledge to decrease neonatal mortality is available. The techniques are simple and easy to apply. What is required is a conviction on the part of the farmers that lower neonatal mortality rates are possible and that they can achieve them, a commitment to recognized approaches (including attention to detail) and supportive training and demonstration from skilled farrowing house attendants or farm advisers.

NEONATAL MORTALITY: CONCLUSIONS

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Throughout this symposium the precarious nature of neonatal existence has been stressed and considering the number of predisposing characteristics of the neonatal pig which seem to discourage survival, it is perhaps remarkable that any piglets survive to weaning. Nevertheless at least 80-90% of liveborn piglets reach weaning, despite being born with only a sparse hair cover, a lack of subcutaneous fat, a high surface/mass ratio and a critical need to acquire colostrum before gut closure occurs. Clearly, environmental factors contribute significantly to neonatal mortality, and thus the task to further reduce mortality largely depends on understanding environmental requirements of the neonate.

In wild swine around the time of farrowing, environmental conditions and the manner in which the sow responds to them via her selection of a nest site and construction of a nest, will be critical to neonatal survival. The functions of the farrowing nest for neonatal survival include protection of the neonates from chilling and predators, and provision of a safe environment in which colostrum is transferred from sow to newborn. Mortality rates, despite the small litter sizes of wild swine, may be considered high.

On the other hand, mortality rates of piglets in commercial herds are clearly lower than those reported for wild swine even though litter sizes of the modern pig are at least twice that of wild swine. The benefits to commercial pig production from increased litter size however, may be lost if in association with larger litter size, the duration of parturition greatly increases and average piglet birth weight is reduced. These factors together for example, may mean that piglets born later in larger litters will have less opportunity to acquire colostrum but will expend more energy in so doing. However, through the development of specialized farrowing accommodation designed to provide the optimum thermal conditions for neonates and the use of simple management techniques, it is possible to save many of the smaller piglets. Further, the importance of an adequate intake of colostrum and milk, especially in the first few hours after birth, for protection against infection and prevention of starvation is recognised and stressed. These factors are also important for reducing piglet morbidity levels and failure to thrive.

In most cases the reduction in mortality levels has been associated with close confinement of sows in farrowing crates and a higher degree of human intervention. Although this may be seen as labour intensive, the approach has often yielded success.

Ironically, improved survival rates amongst neonates due to the use of farrowing crates have often been at the expense of maternal behaviour, which tends to be dampened by the lack of appropriate stimuli. The concern has been expressed however, that further restrictions on the sow may add to, rather than reduce, neonatal mortality, since there is some evidence of a relationship between the conditions in the pre-farrowing environment, the development of maternal behaviour and piglet mortality and morbidity levels. Considering that maternal behaviour evolved for the protection of the piglets, particularly at the neonatal stage, it would seem that future gains to piglet survival may be associated with a better understanding of the role of maternal behaviour.

This symposium has identified the major causes of neonatal mortality, discussed the means to reduce it by the application of easy and simple techniques and in addition indicated that further benefits may flow on via improved piglet health and vitality levels. The use of the farrowing crate to reduce mortality levels has been impressive in many

cases, but there appears to be the potential to further fine-tune existing designs to gain the benefits of maternal behaviour which at present appear to be largely untapped.

References

- AHERNE, F.X., HAYS, V.W., EWAN, R.C. and SPEER, V.C. (1969). Absorption and utilization of sugars by the baby pigs. *Journal of Animal Science*. 29:444-450.
- BARNETT, J.L. and HUTSON, G.D. (1987). Objective assessment of welfare in the pig: Contributions from physiology and behaviour. In "Manipulating Pig Production" pp.1-22, eds. APSA Committee (Australasian Pig Science Association: Werribee, Victoria, Australia).
- BATE, L.A. and HACKER, R.R. (1985a). The influence of the sow's adrenal activity on the ability of the piglet to absorb IgG from colostrum. *Canadian Journal of Animal Science*. 65:77-85.
- BATE, L.A. and HACKER, R.R. (1985b). Influence of environmental temperature during late gestation and soon after birth on IgG absorption by newborn piglets. *Canadian Journal of Animal Science*. 65:87-93.
- BAXTER, M.R. (1981b). The nesting behaviour of sows and its disturbance by confinement at farrowing. In "Disturbed Behaviour In Farm Animals" pp. 101-114, ed. W. Bessei (Verlag Eugen Ulmer: Hohenheim).
- BAXTER, M.R. and PETHERICK, J.C. (1980). The effect of restraint on parturition in the sow. (Proceedings of the International Pig Veterinary Society Congress: Copenhagen), p. 84, eds. N.C. Nielsen, P. Hogh and N. Bille.
- BAXTER, M.R. and SCHWÄLLER, C.E. (1983). Space requirements for sows in confinement. In "Farm Animal Housing and Welfare" pp. 181-195, eds. S.H. Baxter, M.R. Baxter and J.A.C. MacCormack (Martinus Nijhoff Publishers: Boston).
- BAXTER, S.H. (1981a). Welfare and housing of the sow and suckling pigs. In "The Welfare of Pigs" pp. 276-311, ed. W. Sybesma (Martinus Nijhoff Publishers: Boston).
- BAXTER, S.H. (1984a). "Intensive Pig Production: Environmental Management and Design" (Granada Publishing Ltd.: London).
- BAXTER, S.H. (1984b). Chairman's summary and concluding remarks. In "Welfare of Confined Sows" p. 193, eds. A. Aumaitre and R. Dantzer (INRA: Paris)(*Annales de Recherches Veterinaires*. 15:193).
- BAXTER, S.H. and MITCHELL, C.D. (1977). Developments in floor construction in animal production. In "The Veterinary Annual" 17th issue, pp. 286-291, eds. C.S.G. Grunsell and F.W.G. Hill (Wright-Scientifica: Bristol).
- BILLE, N., NIELSON, N.C., LARSEN, J.L. and SVENDSEN, J. (1974). Preweaning mortality in pigs. *Nordisk Veterinær Medecin*. 26:294-313.
- BIRD, P.H., BINNS, S.C. and HARTMANN, P.E. (1988). Monosaccharides in the peripheral blood of piglets after ingestion of lactose and fructose. *Proceedings of the Nutrition Society of Australia*. 13:108.
- BLECHA, F. and KELLEY, K.W. (1981). Cold stress reduces the acquisition of colostral immunoglobulin in piglets. *Journal of Animal Science*. 52:595-600.
- BOHL, E.H. and SAIF, L.J. (1975). Passive immunity in transmissible gastroenteritis of swine: Immunoglobulin characteristics of antibodies in milk after inoculation of virus by different routes. *Infection and Immunity*. 11:23-32.
- BOURNE, F.J. (1969a). Studies on colostral and milk whey proteins in the sow. 1. The transition of mammary secretion from colostrum to milk with natural suckling. *Animal Production*. 11:337-343.
- BOURNE, F.J. (1969b). Studies on colostral and milk whey proteins in the sow. 2. The effect of delayed suckling on colostrum and milk whey proteins. *Animal Production*. 11:345-349.
- BOURNE, F.J. and CURTIS, J. (1973). The transfer of immunoglobulins IgG, IgA and IgM from serum to colostrum and milk in the sow. *Immunology*. 24:157-162.
- BRUCE, J.M. (1977). Conductive heat loss from the recumbent animal. *Farm Building Research and Development Studies*. 8:9-15.
- BRUCE, J.M. (1979). Heat loss from animals to the floor. *Farm Building Progress*. 55:1-4.
- CARLSSON, L.C.T., WESTROM, B.R. and KARLSSON, B.W. (1980). Intestinal absorption of proteins by the neonatal piglet fed on sow's colostrum with either natural or experimentally eliminated trypsin-inhibiting activity. *Biology of the Neonate*. 38:309-320.
- CHIDLOW, J.W., BLADES, J.A. and PORTER, P. (1979). Sow vaccination by combined oral and intramuscular antigen: A field study of maternal protection against neonatal *Escherichia coli* enteritis. *The Veterinary Record*. 105:437-440.
- CHIDLOW, J.W. and PORTER, P. (1979). Intestinal defence of the neonatal pig: Interrelationship of gut and mammary function providing surface immunity against colibacillosis. *The Veterinary Record*. 104:496-500.

- CLARK, M. (1985). Farrowing pen floor abrasiveness measured using a rubber-block drag test. *Farm Building Progress*. 80:29-32.
- CLEARY, G.V. (1987). Current herd performance levels in the Australian pig industry. (Proceedings of the Agrilink Summer Seminar, Agrilink Consulting Group: Shepparton, Victoria).
- CLOUGH, C.E. (1985). "Environmental Design for Piglet Protection" (Master of Science Thesis, University of Aberdeen: Scotland).
- COALSON, J.A. and LECCE, J.G. (1973). Influence of nursing intervals on changes in serum proteins (immunoglobulins) in neonatal pigs. *Journal of Animal Science*. 36:381-385.
- CRONIN, G.M. and CROPLEY, J.A. (1989). The effect of piglet stimuli on the posture changing behaviour of recently farrowed sows. (Proceedings of The Australasian Society for the Study of Animal Behaviour, 16th Annual Conference: Geelong, Victoria), p. 3.
- CURTIS, J. and BOURNE, F.J. (1971). Immunoglobulin quantitation in sow serum, colostrum and milk and the serum of young pigs. *Biochimica et Biophysica Acta*. 236:319-332.
- CURTIS, S.E. (1970). Environmental-thermoregulatory interactions and neonatal pig survival. *Journal of Animal Science*. 31:576-587.
- CURTIS, S.E., HEIDENREICH, C.J. and FOLEY, C.W. (1966). Carbohydrate assimilation and utilization by newborn pigs. *Journal of Animal Science*. 25:655-661.
- CUTLER, R.S. and GARDNER, I. (1988). "A Blue Print for Pig Health Research" (Australian Pig Research Council: Canberra).
- DAVIDSON, G.P. (1984). Lactase deficiency: Diagnosis and management. *Medical Journal of Australia*. September 29th:442-444.
- De PASTILLE, A.B., RUSHEN, J. and PELLETIER, G. (1988). Suckling behaviour and serum immunoglobulin levels in neonatal piglets. *Animal Production*. 47:447-456.
- DOWNEY, B.R., CONLON, P.D., IRVINE, D.S. and BAKER, R.D. (1976). Controlled farrowing program using prostaglandin analogue. *Canadian Journal of Animal Science*. 56:655-659.
- DYCK, G.W. and SWIERSTRA, E.E. (1987). Causes of piglet death from birth to weaning. *Canadian Journal of Animal Science*. 67:543-547.
- EDWARDS, B.L. (1972). Causes of death in new-born pigs. *Veterinary Bulletin*. 42:249-258.
- ELLIOT, J.I., SENFT, B., ERHARDT, G. and FRASER, D. (1984). Isolation of lactoferrin and its concentration in sow's colostrum and milk during a 21-day lactation. *Journal of Animal Science*. 59:1080-1084.
- ENGLISH, P.R. (1969). "Mortality and Variation in Growth of Piglets. A Study of Predisposing Factors with Particular Reference to Sow and Piglet Behaviour" (Doctor of Philosophy Thesis, University of Aberdeen: Scotland).
- ENGLISH, P.R., DIAS, M.F.M. and BAMPPTON, P.R. (1982). Evaluation of an improved design of farrowing crate. (Proceedings International Pig Veterinary Society Congress: Mexico), paper 289.
- ENGLISH, P.R. and MORRISON, V. (1984a). Causes and prevention of piglet mortality. *Pig News and Information*. 5:369-376.
- ENGLISH, P.R. and MORRISON, V. (1984b). Improving piglet survival. *Pig Farming*. September 1984: 22-26.
- ENGLISH, P.R. and MORRISON, V. (1985). Success story on survival. *Pig International*. 15:6-8.
- ENGLISH, P.R. and SMITH, W.J. (1975). Some causes of death in neonatal pigs. *Veterinary Annual*. 15:95-104.
- ENGLISH, P.R., SMITH, W.J. and MACLEAN, A. (1977). "The Sow - Improving Her Efficiency" (Farming Press Ltd.: Ipswich, Suffolk).
- ENGLISH, P.R. and WILKINSON, V. (1982). Management of the sow and litter in late pregnancy and lactation in relation to piglet survival and growth. In "Control of Pig Reproduction", pp. 478-506, eds. D.J.A. Cole and G.R. Foxcroft (Butterworths: London).
- FAHMY, M.H. and BERNARD, C. (1971). Causes of mortality in Yorkshire pigs from birth to 20 weeks of age. *Canadian Journal of Animal Science*. 51:351-359.
- FAHY, V.A., CONNAUGHTON, I.D., DRIESEN, S.J. and SPICER, E.M. (1987). Neonatal diarrhoea update. In "Pig Production" (Proceedings Number 95, Post-Graduate Committee in Veterinary Science, University of Sydney), pp. 965-977.
- FLECKNELL, P.A., WOOTTON, R., ROYSTON, P. and JOHN, M. (1988). Glucose homeostasis in the newborn. *Biology of the Neonate*. 54:356-362.
- FORSTER, E.S. and HEFFNER, E.H. (1954). "Columella - De Re Rustica" (Wm. Heinemann: London).
- FRASER, D. (1980). A review of the behavioural mechanism of milk ejection of the domestic pig. *Applied Animal Ethology*. 6:247-255.
- FRASER, D. (1983/84). The role of behaviour in swine production: A review of research. *Applied Animal Ethology*. 11:317-339.
- FRASER, D. (1984). Some factors influencing the availability of colostrum to piglets. *Animal Production*. 39:115-123.
- FRIENDSHIP, R.M., WILSON, M.R. and McMILLAN, I. (1986). Management and housing factors associated with preweaning mortality. *Canadian Veterinary Journal*. 2:307-311.

- GEERS, R., GOEDSEELS, V., PARDUYN, G. and VERCRUYSE, G. (1986). The group postural behaviour of growing pigs in relation to air velocity, air and floor temperature. *Applied Animal Behaviour Science*. 16:353-362.
- GENTZ, J., BENGTSAN, G., HAKKARAINEN, J., HELLSTROM, R. and PERSSON, B. (1970). Metabolic effects of starvation during neonatal period in the piglet. *American Journal of Physiology*. 218:662-668.
- GLASTONBURY, J.R.W. (1976). A survey of preweaning mortality in the pig. *Australian Veterinary Journal*. 52:272-276.
- GLASTONBURY, J.R.W. (1977). Preweaning mortality in the pig. Pathological findings in piglets dying between birth and weaning. *Australian Veterinary Journal*. 53:310-314.
- GRAHAM, R., SAMPSON, J. and HESTER, H.R. (1941). Acute hypoglycaemia in newborn pigs so called "baby pig disease". *Proceedings of the Society for Experimental Biology and Medicine*. 47:338-339.
- GROMMERS, F.J., CURTIS, S.E., ANTONISSE, H.W. and CHRISTISON, G.I. (1970). Swine-floor contact area as a function of body weight and posture. *Journal of Animal Science*. 31:1232-1234.
- GUIDRY, A.J. (1985). Mastitis and the immune system of the mammary gland. In "Lactation" pp. 229-262, ed. B.L. Larson, written by R.R. Anderson (The Iowa State University Press: Iowa).
- GUNDLACH, H. (1968). Brutfursorge, Brutpflege, Verhaltensontogenese und Tagesperiodik beim Europäischen Wildschwein (*Sus scrofa* L.). *Zeitschrift für Tierpsychologie*. 25:955-995.
- GUSTAFSSON, G. (1985). "Plane Infra-red Heaters for Piglets - Heat Balance Analysis and Initial Trials" (Report 45, Department of Farm Buildings, Swedish University of Agricultural Sciences: Lund).
- HANSON, R.P. and KARSTAD, L. (1959). Feral swine in south-eastern United States. *Journal of Wildlife Management*. 23:64-74.
- HARRIS, J. (1891). "Harris on the Pig" (Orange Judd Co.: 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, Victoria, Australia).
- HARTSOCK, T.G. and GRAVES, H.B. (1976). Neonatal behaviour and nutrition-related mortality in domestic swine. *Journal of Animal Science*. 42:235-241.
- HASKELL, M.J. (1989). Pre-farrowing restlessness and sow welfare: A preliminary study. (Proceedings of The Australasian Society for the Study of Animal Behaviour, 16th Annual Conference: Geelong, Victoria), p.10.
- HENDERSON, R. (1811). "Treatise on the Breeding of Swine" (A. Allardice: Leith).
- HENDRIX, W.F., KELLEY, K.W., GASKINS, C.T. and HINRICHS, D.J. (1978). Porcine neonatal survival and serum gamma immunoglobulins. *Journal of Animal Science*. 47:1281-1287.
- HOOPERBRUGGE, A. (1983). Biggensterfte. (Syllabus van de Voordrachten Tijdens de Contactdagen voor het Welzijnsonderzoek: Wageningen), October 18-19, pp. 92-97.
- HOOPER, W.D. (1934). "Cato and Varro - De Re Rustica" (Wm. Heinemann: London).
- HUNT, K. and PETCHEY, A.M. (1987). A study of environmental preferences of sows around farrowing. *Farm Building Progress*. 89:11-14.
- HUTSON, G.D. (1988). Do sows need straw for nest-building? *Australian Journal of Experimental Agriculture*. 28:187-194.
- IGLESIAS, G. and TRUJANO, M. (1989). Studies on maternally derived antibodies to Aujeszky's disease virus in piglets born to naturally or experimentally infected sows. *Journal of Veterinary Medicine*. 36:57-62.
- INOUE, T. (1981). Possible factors influencing immunoglobulin A concentration in swine colostrum. *American Journal of Veterinary Research*. 42:533-536.
- INOUE, T., KITANO, K. and INOUE, K. (1980). Possible factors influencing the immunoglobulin G concentration in swine colostrum. *American Journal of Veterinary Research*. 41:1134-1136.
- JENSEN, P. (1986). Observations on the maternal behaviour of free-ranging domestic pigs. *Applied Animal Behaviour Science*. 16:131-142.
- JENSEN, P. (1989). Nest site choice and nest building of free-ranging domestic pigs due to farrow. *Applied Animal Behaviour Science*. 22:13-21.
- JENSEN, P., FLOREN, K. and HOBROH, B. (1987). Peri-parturient changes in behaviour in free-ranging domestic pigs. *Applied Animal Behaviour Science*. 17:69-76.
- JENSEN, P. and REDBO, I. (1987). Behaviour during nest leaving in free-ranging domestic pigs. *Applied Animal Behaviour Science*. 18:355-362.
- JENSEN, P.T. and PEDERSEN, K.B. (1979). Studies on immunoglobulins and trypsin inhibitor in colostrum and milk from sows and the serum of their piglets. *Acta Veterinaria Scandinavica*. 20:60-72.
- JENSEN, P.T. and PEDERSEN, K.B. (1982). The influence of sow colostrum trypsin inhibitor on the immunoglobulin absorption in newborn piglets. *Acta Veterinaria Scandinavica*. 23:161-168.

- JEPPESON, L.E. (1984). Environment and the behaviour of the sow and her young. *Proceedings of the Pig Veterinary Society*. 11:18-26.
- JONES, J.E.T. (1966). Observations on parturition in the sow: Part II: The parturient and post-parturient phases. *British Veterinary Journal*. 122:471-478.
- KATZ, J., KUWAJIMA, M., FOSTER, D.W. and MCGARRY, J.D. (1986). The glucose paradox: New perspectives on hepatic carbohydrate metabolism. *Trends in Biological Sciences*. 3:135-140.
- KELLEY, K.W., BLECHA, F. and REGNIER, J.A. (1982). Cold exposure and absorption of colostral immunoglobulins by neonatal piglets. *Journal of Animal Science*. 55:363-368.
- KIRKWOOD, J.K., GASKIN, C.D. and MARKHAM, J. (1987). Perinatal mortality and season of birth in captive wild ungulates. *The Veterinary Record*. 120:386-390.
- KLOBASA, F., WERHAHN, E. and BUTLER, J.E. (1981). Regulation of humoral immunity in the piglet by immunoglobulins of maternal origin. *Research in Veterinary Science*. 31:195-206.
- KOHLER, E.M., CROSS, R.F. and BOHL, E.H. (1975). Protection against neonatal enteric colibacillosis in pigs suckling orally vaccinated sows. *American Journal of Veterinary Research*. 36:757-764.
- KRETCHMER, N. (1972). Lactose and lactase. *Scientific American*. 227:71-78.
- KRUSE, P.E. (1983). The importance of colostral immunoglobulins and their absorption from the intestine of newborn animals. *Annales de Recherches Veterinaires*. 14:349-353.
- LAMMERS, G.J. and de LANGE, A. (1986). Pre- and post-farrowing behaviour in primiparous domesticated pigs. *Applied Animal Behaviour Science*. 15:31-43.
- LASKOWSKI, M., KASSELL, B. and HAGERTY, G. (1957). A crystalline trypsin inhibitor from swine colostrum. *Biochimica et Biophysica Acta*. 24:300-305.
- Le DIVIDICH, J. and NOBLET, J. (1981). Colostrum intake and thermoregulation in the neonatal pig. *Biology of the Neonate*. 40:167-174.
- Le DIVIDICH, J. and NOBLET, J. (1983). Thermoregulation and energy metabolism in the neonatal pig. *Annales de Recherches Veterinaires*. 14:375-381.
- LECCE, J.G. (1986). Diarrhoea: The nemesis of the artificially reared, early weaned piglet and a strategy for defence. *Journal of Animal Science*. 63:1307-1313.
- LEE, C.S., MCCAULEY, I. and HARTMANN, P.E. (1983). Light and electron microscopy of cells in pig colostrum, milk and involution secretion. *Acta Anatomica*. 116:126-135.
- LEIBHOLZ, J. (1986). Some aspects of digestion in the pig from birth to 56 days of age. *Proceedings of the Nutrition Society of Australia*. 11:32-39.
- LEWIS, N.J. and HURNIK, J.F. (1985). The development of nursing behaviour in swine. *Applied Animal Behaviour Science*. 14:225-232.
- LONGWILL, A. (1952). Survey of losses in pigs on New Zealand farms, 1949/51. *New Zealand Journal of Science and Technology*. 34:294-305.
- MACHADO-NETO, R., GRAVES, C.N. and CURTIS, S.E. (1987). Immunoglobulins in piglets from sows heat-stressed prepartum. *Journal of Animal Science*. 65:445-455.
- MANNERS, M.J. and STEVENS, J.A. (1972). Changes from birth to maturity in the pattern of distribution of lactase and sucrose activity in the mucosa of the small intestine of pigs. *British Journal of Nutrition*. 28:113-127.
- McBRIDE, G., JAMES, J.W. and WYETH, G.S.F. (1965). Social behaviour of domestic animals. VIII. Variations in the weaning weight in pigs. *Animal Production*. 7:67-74.
- McFERRAN, J.B. and DOW, C. (1973). The effect of colostrum derived antibody on mortality and virus excretion following experimental infection of piglets with Aujeszky's disease virus. *Research in Veterinary Science*. 15:208-212.
- MECHI, J.J. (1852). Feeding on open-boarded floors. *Practical Mechanics Journal*. 4:68.
- MELLOR, D.J. and COCKBURN, F. (1986). A comparison of energy metabolism in the newborn infant, piglet and lamb. *Quarterly Journal of Experimental Physiology*. 71:361-379.
- MERSMANN, H.J. (1971). Glycolytic and gluconeogenic enzyme levels in pre- and post-natal pigs. *American Journal of Physiology*. 222:1297-1302.
- METZ, J.H.M. and OOSTERLEE, C.C. (1980). Immunologische und ethologische Kriterien für die artgemasse Haltung von Sauen und Ferkeln. In "Aktuelle Arbeiten zur artgemassen Tierhaltung", KTBL-Schrift 264, pp. 39-50 (KTBL: Darmstadt, West Germany).
- MILON, A., AUMAITRE, A., Le DIVIDICH, J., FRANZ, J. and METZGER, J.J. (1983). Influence of birth prematurity on colostrum composition and subsequent immunity in piglets. *Annales de Recherches Veterinaires*. 14:533-540.
- MITCHELL, C.D. and SMITH, W.J. (1978). Piglet foot dimensions for design of slotted floors. *Farm Building Progress*. 51:7-9.
- MOON, H.W. (1981). Protection against enteric colibacillosis in pigs suckling orally vaccinated dams: evidence for pili as protective antigens. *American Journal of Veterinary Research*. 42:173-177.
- MOON, H.W., ROGERS, D.G. and ROSE, R. (1988). Effects of an orally administered live *Escherichia coli* pilus vaccine on duration of lacteal immunity to enterotoxigenic *Escherichia coli* in swine. *American Journal of Veterinary Research*. 49:2069-2071.

- MORRISON, V., ENGLISH, P.R. and LODGE, G.A. (1983). The effect of alternative creep heating arrangements at two house temperatures on piglet lying behaviour and mortality in the neonatal period. *Animal Production*. **36**:530-531.
- MOSER, B.D. and LEWIS, A.J. (1981). Fat additions to sow diets - a review. *Pig News and Information*. **2**:265-269.
- MOUNT, L.E. (1968). "The Climatic Physiology of the Pig" (E. Arnold: London).
- MUIRHEAD, M.R. (1987). Mortality. In "Pig Production" (Proceedings Number 95, Post-Graduate Committee in Veterinary Science, University of Sydney: Sydney), pp. 525-558.
- MURATA, H. and NAMIOKA, S. (1977). The duration of colostral immunoglobulin uptake by the epithelium of the small intestine of neonatal piglets. *Journal of Comparative Pathology*. **87**:431-439.
- MURGITA, R.A. and WIGZELL, H. (1981). Regulation of the immune response in the fetus and newborn. *Progress in Allergy*. **29**:54-133.
- NAAKTGEBOREN, C. (1979). Behavioural aspects of parturition. *Animal Reproduction Science*. **2**:155-166.
- NEWBERRY, R.C. and WOOD-GUSH, D.G.M. (1985). The suckling behaviour of domestic pigs in a semi-natural environment. *Behaviour*. **95**:11-25.
- NIELSEN, N.C., CHRISTENSEN, K., BILLE, N. and LARSEN, J.L. (1974). Prewaning mortality in pigs. 1. Herd investigations. *Nordisk Veterinaermedicin*. **26**:137-150.
- PEGORIER, J.P., DUEE, P.H., ASSAN, R., PERET, J. and GIRARD, J.R. (1981). Changes in circulating fuels, pancreatic hormones and liver glycogen concentration in fasting or suckling newborn pigs. *Journal of Developmental Physiology*. **3**:203-217.
- PEGORIER, J.P., DUEE, P.H., GIRARD, J.R. and PERET, J. (1982). Development of gluconeogenesis in isolated hepatocytes from fasting or suckling newborn piglets. *Journal of Nutrition*. **112**:1038-1046.
- PEGORIER, J.P., DUEE, P.H., GIRARD, J.R. and PERET, J. (1983). Rate of glucose turnover in suckling or fasting newborn pigs. *Federation Proceedings*. **42**:1328.
- PEGORIER, J.P., DUEE, P.H., NUNES, C.S., PERET, J. and GIRARD, J.R. (1984). Glucose turnover and recycling in unrestrained and unanaesthetized fasting or post absorptive newborn pigs. *British Journal of Nutrition*. **52**:277-287.
- PENNY, R.H.C., EDWARDS, M.J. and MULLEY, R. (1971). Clinical observations of necrosis of the skin of suckling pigs. *Australian Veterinary Journal*. **47**:529-537.
- PETHERICK, J.C. (1982). "A Biological Basis for the Design of Space in Livestock Housing" (Master of Science Thesis, University of Aberdeen: Scotland).
- PETHERICK, J.C. (1982/83). A note on nursing termination and resting behaviour of suckling piglets. *Applied Animal Ethology*. **9**:359-365.
- PETHERICK, J.C. (1983). A note on allometric relationships in Large White x Landrace pigs. *Animal Production*. **36**:497-500.
- PETTIGREW, J.E. (1979). Fat in gestation and lactation diets for sows. (Proceedings 71st Annual Meeting, American Society of Animal Science: Tucson), pp. 1-10.
- PETTIGREW, J.E., ZIMMERMANN, D.R. and EVAN, R.C. (1971). Plasma carbohydrate levels in the neonatal pig. *Journal of Animal Science*. **32**:895-899.
- POMEROY, R.W. (1960). Infertility and neonatal mortality in the sow. III. Neonatal mortality and foetal development. *Journal of Agricultural Science (Cambridge)*. **54**:31-56.
- PORCIUS (pseudonym). (1850). "The Rev. Haxtable and His Pigs" (Wm. Blackwood and Sons: Edinburgh).
- PORTER, P. (1981). Immune system. In "Diseases of Swine", fifth edition, pp. 41-51, eds. A.D. Lemar, R.D. Glock, W.L. Mengeling, R.M.C. Penny, E. Scholl and B. Straw (The Iowa State University Press: Iowa).
- PORTER, P. and HILL, I.R. (1970). Serological changes in immunoglobulins IgG, IgA and IgM and *Escherichia coli* antibodies in the young pig. *Immunology*. **18**:565-573.
- PRIME, R.W., FAHY, V.A., RAY, W., CUTLER, R.S. and SPICER, E.M. (1987). On farm validation of research - lowering preweaning mortality rates in pigs. (Report to Pig Research Council, Department of Primary Industries and Energy: Canberra).
- RANDALL, G.C.B. (1972). Observations on parturition in the sow. I. Factors associated with the delivery of the piglets and their subsequent behaviour. *The Veterinary Record*. **90**:178-182.
- REITER, B. (1978). Review of the progress of dairy science: antimicrobial systems in milk. *Journal of Dairy Research*. **45**:131-147.
- REITER, B. (1985). The biological significance and exploitation of the non-immunoglobulin protective proteins in milk: Lysozyme, Lactoferrin, Lactoperoxidase, Xanthineoxidase. *Bulletin of the International Dairy Federation*. **191**:2-35.
- ROBERTSON, A.M. (1977). Accommodation for farrowing and lactating sows. *Farm Building Progress*. **48**:15-18.

- ROBERTSON, A.M. and CLARKE, J.J. (1980). Where skill saves lives. *Pig International*. 10:14-21.
- ROBERTSON, A.M. and McCARTNEY, A. (1980). Piglet farrowing boxes. *Farm Building Progress*. 59:15-16.
- SAINSBURY, D. (1963). "Pig Housing" (Farming Press (Books) Ltd.: Ipswich).
- SAMPSON, J. and GRAHAM, R. (1943). Studies on baby pig mortality. 3. A note on experimental insulin hypoglycaemia in the pig. *Journal of American Veterinary Medical Association*. 102:176.
- SCHWALLER, C.E. (1981). "Space Utilization by Sows Standing Up and Lying Down in Confinement" (Bachelor of Science, Thesis, Trent Polytechnic: England).
- SHARPE, H.B.A. (1966). Pre-weaning mortality in a herd of Large White pigs. *British Veterinary Journal*. 122:99-111.
- SIEGAL, C.D., SPARKS, J.W. and BATTAGLIA, F.C. (1988). Patterns of serum glucose and galactose concentrations in term newborn infants after milk feeding. *Biology of the Neonate*. 54:301-306.
- SMITH, N.A., MORRIS, S.J. and HARTMANN, P.E. (1988). The ingestion and absorption of carbohydrates in newborn piglets. *Proceedings of the Nutrition Society of Australia*. 13:108.
- SMITH, W.J. (1978). The effects of environmental factors, floor design and materials on foot and limb disorders in baby pigs. In "Animal Housing - Injuries Due to Floor Surfaces", pp. 49-56 (Cement and Concrete Association: Slough).
- SMITH, W.J. and MITCHELL, C.D. (1976). Observations on injuries to suckled pigs confined on perforated floors with special reference to expanded metal (2073f). *Pig Veterinary Society Proceedings*. 1:91-104.
- SPICER, E.M., DRIESEN, S.J., FAHY, V.A. and HORTON, B.J. (1985). Trauma, overlay and savaging. *Australian Advances in Veterinary Science*. p. 122.
- 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). Causes of preweaning mortality on a large intensive piggery. *Australian Veterinary Journal*. 63:71-75.
- SPICER, E.M., DRIESEN, S.J., FAHY, V.A., WILLIAMSON, P.L. and CONNAUGHTON, I.D. (1987). Pre-weaning mortality in pigs. In "Pig Production" (Proceedings Number 95, Post-Graduate Committee in Veterinary Science, University of Sydney), pp. 979-985.
- SPOONER, E.O. (1850). "The Adventures and Transformations of Nitrogen and Ammonia" (Ridgeway: London).
- SPRAGUE, J.T., ULLREY, D.E., WADDILL, D.G., MILLER, E.R., ZUTAUT, C.L. and HOEFER, J.A. (1963). Intestinal lactase, alkaline and acid phosphatase in the swine fetus and newborn pig. *Journal of Animal Science*. 22:121.
- SPRECHER, D.J., LEMAN, A.D. and CARLISLE, S. (1975). Effects of parasymphomimetics on porcine stillbirth. *American Journal of Veterinary Research*. 36:1331-1333.
- STANISLAW, C.M. (1971). Skin and knee abrasions. *Hog Farm Management*. 8:26, 30, 35 and 52.
- STEELE, N.C., FROBISH, L.T., MILLER, L.R. and YOUNG, E.P. (1971). Certain aspects of the utilization of carbohydrates by the neonatal pig. *Journal of Animal Science*. 33:983-986.
- STONE, S.S., KEMENY, L.J., WOODS, R.D. and JENSEN, M.T. (1977). Efficacy of isolated colostrum IgA, IgG and IgM to protect neonatal pigs against the corona-virus of transmissible gastroenteritis. *American Journal of Veterinary Research*. 40:607-612.
- STONE, S.S., PHILLIPS, M. and KEMENY, L.J. (1979). Stability of porcine colostrum immunoglobulins IgA, IgG₂ and IgM to proteolytic enzymes. *American Journal of Veterinary Research*. 40:607-612.
- SVENDSEN, J., BENGTTSSON, A.Ch. and SVENDSEN, L.S. (1986). Occurrence and causes of traumatic injuries in neonatal pigs. *Pig News and Information*. 7:159-170.
- SVENDSEN, J. and BILLE, N. (1981). Reducing baby pig mortality. In "Diseases of Swine", pp. 729-736, compiled by A.D. Leman (Iowa State University Press: Ames).
- SWIATEK, K.R., CHAO, K., HSIANG-LIN, C., CORNBLATH, M. and TILDON, J.T. (1970). Enzymatic adaptations in newborn pig liver. *Biochimica et Biophysica Acta*. 222:145-151.
- THACKER, P.A. (1986). Use of the "Metadata Blowaway System" in an attempt to reduce pre-weaning mortality in baby pigs. *Farm Building Progress*. 84:13-15.
- THOMSON, M.H. (1983). "The Behaviour of Piglets in Relation to Creep Heating" (Bachelor of Science Thesis, University of Aberdeen: Scotland).
- TITTERINGTON, R.W. and FRASER, D. (1975). The lying behaviour of sows and piglets during early lactation in relation to the position of the creep heater. *Applied Animal Ethology*. 2:47-53.
- VARLEY, M.A., WILKINSON, R.G. and MAITLAND, A. (1987). Artificial rearing of baby piglets: The effects of colostrum on survival and plasma concentrations of IgG. *British Veterinary Journal*. 145:369-378.
- VESTERGAARD, K. and HANSEN, L.L. (1984). Tethered versus loose sows: Ethological observations and measures of productivity. I. Ethological observations during pregnancy and farrowing. *Annales de Recherches Veterinaires*. 15:245-256.
- WALKER, N. (1977). The effects of induction of parturition in sows using an analogue of prostaglandin F₂. *Journal of Agricultural Science (Cambridge)*. 89:267-271.

- WEBB, N.G. (1984). Compressive stresses on, and the strengths of, the inner and outer digits of pigs' feet, and the implications for injury and floor design. *Journal of Agricultural Engineering Research*. 30:71-80.
- WEBB, N.G. and NILSSON, C. (1983). Flooring and injury - an overview. In "Farm Animal Housing and Welfare", pp. 226-259, eds. S.H. Baxter, M.R. Baxter and J.A.C. MacCormack (Martinus Nijhoff: Boston).
- WELCH, A.R. (1986a). "Environmental Control of Piglet Behaviour During the Suckling Period" (Doctor of Philosophy Thesis, University of Aberdeen: Scotland).
- WELCH, A.R. (1986b). Pathways for newborn piglets from sow to creep area. *Farm Building Progress*. 85:27-28.
- WELCH, A.R. and BAXTER, M.R. (1986). Responses of newborn piglets to thermal and tactile properties of their environment. *Applied Animal Behaviour Science*. 15:203-215.
- WESTROM, B.R., OHLSSON, B.G., SVENDSEN, J., TAGESSON, C. and KARLSSON, B.W. (1985). Intestinal transmission of macromolecules (BSA and FITC-dextran) in the neonatal pig: Enhancing of colostrum, proteins and proteinase inhibitors. *Biology of the Neonate*. 47:359-366.
- WESTROM, B.R., SVENDSEN, J., OHLSSON, B.G., TAGESSON, C. and KARLSSON, B.W. (1984). Intestinal transmission of macromolecules (BSA and FITC-labelled dextrans) in the neonatal pig: Influence of age of piglet and molecular weight of markers. *Biology of the Neonate*. 46:20-26.
- WHITTEMORE, C.T. and FRASER, D. (1974). The nursing and suckling behaviour of pigs. II. Vocalization of the sow in relation to suckling behaviour and milk ejection. *British Veterinary Journal*. 130:46-356.
- YAGUCHI, H., MURATA, H., KAGOTA, K. and NAMIOKA, S. (1980). Studies on the relationship between serum gamma globulin levels of neonatal piglets and their mortality during the first two months of life: An evaluation for the ammonium sulphate reaction. *British Veterinary Journal*. 136:63-70.
- YOUATT, W. (1847). "The Pig" (London).

THE EFFECT OF VITAMIN E SUPPLEMENTATION ON THE PERFORMANCE OF GROWING PIGS

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The major function of vitamin E in the body is as an antioxidant to protect biological membranes from peroxidation. Post-weaning is the most susceptible period for pigs to develop symptoms of vitamin E deficiency. In the present study, the requirement of vitamin E was investigated.

Three pigs were weaned from each of 4 litters at 21 days of age and allotted in 3 experimental groups. They were fed a basal diet of wheat starch, casein, soyabean meal, lard and minerals and vitamins, which contained 0.6 mg vitamin E/kg. The diet was supplemented with 0, 20 or 100 mg/kg dl- α -tocopherol acetate (Table 1). The pigs were reared in individual pens and were offered the diets *ad libitum* for 63 days.

Pigs were bled weekly and α -tocopherol was measured in plasma. Two estimations of lipid peroxidation in pigs were conducted to indirectly evaluate vitamin E status at the end of the experiment: (1) thiobarbituric acid (TBA) reaction to determine lipid peroxidation level in red cells of blood (Fontaine and Valli, 1977); (2) ethane and pentane in exhaled gas (Riely *et al.*, 1974).

Table 1. Effect of vitamin E supplement of diets for pigs (21-84 days of age)

	Week of experiment	Vitamin E supplement (mg kg/diet)			SEM
		0	20	100	
Ethane (pmol/l)	9	177 ^y	97 ^x	70 ^x	18.1
Pentane (pmol/l)	9	149 ^b	85 ^a	55 ^a	18.4
TBA (μ mol 100 g/Hb)	9	11.9 ^b	7.0 ^a	9.0 ^a	0.96
Plasma tocopherol (μ g/ml) -	1	1.59	1.28	1.19	0.343
	4	0.10 ^x	0.31 ^x	1.14 ^y	0.074
	6	0.13 ^x	0.77 ^y	1.85 ^z	0.145
Growth rate (g/day)	0-9	592	628	524	40.4
Food conversion ratio	0-9	1.34	1.29	1.36	0.038

^{a,b}differ at $P < 0.05$; ^{x,y,z}differ at $P < 0.01$

Lipid peroxidation levels as indicated by ethane and pentane exhalation and TBA reaction in red cells were significantly greater in the pigs which were not supplemented with vitamin E, but no significant differences were found between the other two groups. The plasma α -tocopherol level of pigs receiving no supplemental vitamin E fell to 0.1 μ g/ml within four weeks and remained low thereafter. Although the plasma α -tocopherol level in Group II pigs decreased initially, six weeks after treatment began the level increased above 0.4 μ g/ml, which is considered adequate for growing pigs (van Vleet, 1982).

Under the present experimental conditions, it may be concluded that a supplement of 20 mg of dl- α -tocopherol acetate/kg of diet is adequate to meet the requirements of young pigs.

References

- FONTAINE, M. and VALLI, V.E.O. (1977). *Canadian Journal of Comparative Medicine*. 41:52-66.
 RIELY, C.A., CHOEN, G. and EVANS, G.D. (1974). *Science*. 183:208-210.
 Van VLEET, J.F. (1982). *American Journal of Veterinary Research*. 43:1180-1189.

THE EFFECTS OF FOLIC ACID SUPPLEMENTATION ON THE PERFORMANCE OF GROWING PIGS

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Folic acid is required for the metabolism of single carbon compounds, and a deficiency may result in poor growth and anaemia (NRC, 1988). The contribution of folic acid in feeds commonly fed to pigs, combined with bacterial synthesis in the intestinal tract, is thought to meet the requirement of pigs. Therefore, folic acid is not usually added to pig diets. In the present experiment pigs were given a purified diet with and without a folic acid supplementation.

Twelve pigs which were weaned at 3 days of age were allocated to three groups: A control diet of casein, cornflour and a vitamin and mineral premix free of folic acid (Control); the same diet supplemented with an antibiotic, phthalylsulfacetamide, at the rate of 200 mg/kg live weight/day (AB); and the control diet with a supplement of 0.36 mg folic acid/kg diet (FA). The antibiotic treatment was included to reduce the microbial synthesis of folic acid. The control diet contained 0.05 mg folic acid/kg.

Growth rates and feed conversion ratios (FCR) were measured between 3 and 106 days of age, blood samples taken by jugular venipuncture on day 54 for plasma folates and a histidine load test (Luhby *et al.*, 1959) was performed at 98 days.

Table 1. Growth performance (3-106 days), plasma folate levels and FIGLU levels in urine of pigs supplemented or not supplemented with folic acid

Diet	FCR	Weight gain (g/day)	Plasma folates (ng/ml)	FIGLU* ($\mu\text{mol}/8\text{ h}$)		SEM
				Before load	After load	
Control	1.67	481	9.9 ^a	64.7 ^{ab1}	182.8 ^{a2}	25.9
+AB	1.60	482	7.0 ^a	135.9 ^{b1}	555.2 ^{b2}	115.0
+FA	1.65	424	41.9 ^b	0.6 ^{a1}	2.5 ^{a1}	0.8
SEM	0.034	18.2	0.75	31.5	93.79	

*FIGLU (forminoglutamic acid) is an intermediate metabolite in the conversion of histidine to glutamic acid. This reaction requires folic acid and if not present FIGLU excretion in the urine is increased, especially after loading with histidine.

^{a,b}in same column differ at $P < 0.05$; ^{1,2}in same row differ at $P < 0.05$

There was no significant effect of folate supplementation on the performance of the pigs. However, the concentration of folate in plasma was low in the pigs which did not receive supplemental folic acid. The results also revealed an elevated excretion of FIGLU in the pigs without folic acid supplementation, both before and after dosing the pigs with 8 g of L-histidine. These results confirm earlier observations in humans and rats (Luhby *et al.*, 1959; Tabor and Wyngarden, 1965).

Plasma folate and FIGLU excretion may be useful indicators of folate deficiency.

References

- LUHBY, A.L., COOPERMAN, J.M. and TELLER, D.N. (1959). *Proceedings of the Society of Experimental Biology and Medicine*. **101**:350-352.
- NATIONAL RESEARCH COUNCIL. (1988). "Nutrient Requirements of Swine" Ninth revised edition (National Academy Press: Washington DC).
- TABOR, H. and WYNGARDEN, L. (1958). *Journal of Clinical Investigation*. **37**:824-828.

CITRIC ACID SUPPLEMENTATION OF CREEP-WEANER DIETS

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The addition of an organic acid, such as citric acid (CA), to creep-weaner diets can reduce stomach pH, thereby reducing the incidence of post-weaning scours (Bolduan *et al.*, 1988). This experiment was conducted to examine the effects of CA supplementation on feed intake and the incidence of post-weaning scours in piglets fed a conventional wheat-based diet.

The control diet containing wheat, fish meal, meat and bone meal, soyabean meal, L-lysine and tallow was formulated to 15 MJ digestible energy (DE)/kg and 0.67 g available lysine/MJ DE. The CA diet was produced by supplementation of the control diet with 10 g CA/kg. The control and CA diets were offered *ad libitum* to 12 and 11 litters respectively between 10 and 49 days of age. Litters of comparable size were allocated to each diet. Weaning occurred at 28 days of age; feed intake and the occurrence and severity of post-weaning scours were recorded daily.

Table 1. Performance of piglets given citric acid (CA) in their creep-weaner diets

	Control diet	+CA diet	SEM
Live weight (kg/piglet)			
10 days	2.97	3.06	0.121
28 days	6.77	6.88	0.332
49 days	14.35	14.74	0.826
Feed intake (g/piglet/day)			
10-28 days	19	36	4.9
29-49 days	522	560	42.1
10-49 days	276	304	23.2
Feed conversion ratio			
29-49 days	1.40	1.41	0.046
Incidence of scouring (days/litter)			
29-49 days	1.5	1.0	0.62

Inclusion of CA in the creep-weaner diet had no effect on growth performance but did improve feed intake prior to weaning. This is consistent with the results obtained by Magee *et al.* (1987). The acidified diet had no effect on either the number of days that scouring occurred (Table 1) or on the severity of scouring.

These results indicate that although CA supplementation increased creep intake, there was no apparent effect on growth performance or on the incidence of scouring.

References

- BOLDUAN, G., JUNG, H., SCHNABEL, E. and SCHNEIDER, R. (1988). *Pig News and Information*. 9:382-385.
- MAGEE, M.H., WILLIAMS, K.C. and NEILL, A.R. (1987). In "Manipulating Pig Production" p. 150, eds. APSA Committee (Australasian Pig Science Association: Werribee, Victoria, Australia).

A SYMPOSIUM - DIET AND MANAGEMENT OF WEANER PIGS

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Introduction

There is considerable variation between the different types of feeding and management systems for weaner pigs which are being used successfully around the world. However, the decision on which system a primary producer should adopt to suit his particular production unit is a difficult one, not only for the producer, but also for his advisers, e.g. extension officers, pig scientists, stockfeed manufacturers and veterinarians. Despite the remarkable improvements in efficiency of pig production over the years, which have been brought about by the application of advances in scientific knowledge, the variable performance and health of piglets following weaning remains a particularly important problem in the pig industry.

For a pig production enterprise to remain financially viable there is continuous pressure on the producer to increase the number of pigs sold/sow/year, to produce top quality carcasses rapidly and with good feed conversion efficiency, and to make optimal use of housing. In an attempt to maximise the number of pigs sold/sow/year, age at weaning has been reduced so that nowadays, weaning at 28 days is normal and weaning at 21 days can be successful, provided the quality of management is excellent. However, the best of the producers who wean at 28 days produce more pigs and are more profitable than the poorer producers who wean at 21 days. Consequently, reducing age at weaning on its own does not necessarily result in an increase in the productivity or profitability of a particular enterprise. It is important that factors such as weight at weaning, the quality of the post-weaning diet and the suitability of the post-weaning environment are given due consideration. The growth performance of pigs from weaning to 3 months of age is critical in determining subsequent performance. There is some evidence that the ability to produce quality carcasses from fast-growing finishing pigs fed *ad libitum* is enhanced by rapid early growth, with genetic and environmental factors also having an important influence.

The aims of this symposium are to investigate the factors which are known to influence weaner growth performance and to determine how these factors interact to influence the choice of ingredients, diets and feeding systems needed to achieve a target weight of 30 kg at 10 weeks. Recommendations for nutrient specifications of diets are very variable with high protein, high energy, low fibre diets being recommended on the one hand and relatively high fibre diets and low protein diets as alternatives. These apparent anomalies can be largely explained in terms of interactions with feeding management, weaning age, live weight and physiological development of the digestive tract. However, there is still much to learn about dietary amino acid availability, optimum energy levels, type of fat source and type of fibre in baby pig feeds.

Recommendations for restrictions of different raw materials in weaner diets are also variable. The relative efficiency of utilization of animal and vegetable proteins, from different sources and the apparent special role of processed milk products in weaner diets are areas in need of clarification. The use of added fat and precooked cereals also needs clarification, while the role of flavours and flavour enhancers in increasing feed intake of baby pigs, and of organic acids in reducing digestive upsets is of interest.

Another potential source of problems in commercial practice is the wide range in the weights of piglets at weaning and mixing, and the way in which this can affect

performance of individuals due to competition within the group as a whole and the interaction with stocking rate. Finally, we need to know if sophisticated and expensive creep and weaner diets and feeding management systems based on milk products, high quality animal protein sources, cooked cereals, with added fats, flavours and organic acids can be justified financially, or whether lower cost diets and simpler feeding management systems are preferable. In particular we need to know how these various factors might interact with age at weaning and the animal's environment.

In this symposium we shall look at the main reasons for the successes and failures of various feeding management systems for baby pigs under different piggery situations. Of overriding significance is the stage of physiological development of the digestive system of the baby pig at weaning. The first paper will deal with this area in detail and outline the biological limitations imposed by the developing digestive system on the growth performance of weaned pigs. The second paper looks specifically at the development of alternative feeding strategies for weaner pigs, reviewing information from around the world on feeding systems, feed specifications and feed formulation in relation to the developing digestive system of the baby pig. The final speaker discusses the nutritional management of weaner pigs, drawing largely on his own results from experiments and trials carried out in a controlled research and development environment combined with more recent personal experience from the largest commercial intensive piggery complex in Australia.

BIOLOGICAL LIMITATIONS IMPOSED BY THE DIGESTIVE SYSTEM TO THE GROWTH PERFORMANCE OF WEANED PIGS

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Introduction

Sow's milk is "tailor-made" for the young pig and the natural development of the gastrointestinal system proceeds "in-concert" with the frequent and regular ingestion of small quantities of milk "round-the-clock". When a piglet is weaned on to a diet of solid food, its gastrointestinal system has to adapt to changes in the nature of its diet and to changes in the pattern of intake. The ability of the piglet to undergo this adaptation is directly dependant on its size and age at weaning. Thus the smaller and younger a piglet is at weaning the poorer will be its growth rate in the post-weaning period (Campbell, 1989). If, however, the physiological limitations of the gastrointestinal system of the newly weaned pig are understood then diets can be formulated and management procedures devised and adopted which will significantly improve its post-weaning growth performance. In this paper we have attempted to highlight the important events which occur in the development of the gastrointestinal system of the sucking pig and identify some of the factors which may limit the animal's adaptation to the weaned state. It is hoped that this will stimulate discussion between physiologists, nutritionists and those responsible for the management of the pig at weaning. Rather than blame the victim, the weaned pig, for poor post-weaning performance, we should be considering ways of improving the weaning procedure, the post-weaning environment and the pre- and post-weaning diets to ensure that each pig has a chance of achieving the target weight of 28-30 kg at 10 weeks of age mentioned by Campbell (1989).

Porcine milk

Following the ingestion of a protein-rich colostrum immediately after birth, piglets are suckled by the sow and receive milk, a secretion developed during many centuries of natural selection, for adequate nurture. The chemical composition of sow's milk is given in Table 1. Milk supplies the piglet with the nutrients required for growth and development, along with a ready supply of relatively un-contaminated water. Milk not only supplies the piglet with energy and essential amino acids but also with essential fatty acids, minerals, trace-elements and vitamins.

Piglets are suckled by the sow approximately every hour (Hartman *et al.*, 1962; Hartmann *et al.*, 1984; Pluske and Williams, 1988), throughout the day and night. The average milk consumption over an eight-week lactation is 800 g/piglet/day (Lucas and Lodge, 1961) with intake rising to a peak (1000 g/day) at between three and five weeks of age (Elsley, 1970). However, as milk yield is affected by a variety of factors, there is considerable variation in the amount of milk ingested/pig, both within and between litters.

Milk is particularly well digested by the neonatal pig as is indicated by the data presented by Lucas and Lodge (1961) for the digestibility of nutrients in sow's milk by two three-week-old piglets (Table 2). Results from several studies indicate that the components of cow's milk are also well digested by the piglet. For example, Braude and Newport (1973) gave whole cow's milk to piglets (2-26 days of age) and found an

apparent digestibility of lipid of 96%. Similarly, the apparent digestibility of protein determined over the entire digestive tract of piglets (4-40 days of age) given cow's milk, ranges from 95-99% (Terroine and Spindler, 1925; Braude *et al.*, 1970). Recently values have been obtained for the digestibility of amino acids in bovine milk protein using the ileal method (Table 3). These results confirm the almost complete digestion and absorption of milk protein.

Table 1. The chemical composition of sow's milk¹

Component	Concentration	Amino acid	(g/100 g protein)	Fatty acid (% TFA) ²	
Protein ³ (%)	5.96	Lysine	7.4	Capric	0.2
Lactose (%)	4.73	Methionine	1.4	Lauric	0.3
Fat (%)	8.50	Cystine	1.4	Myristic	3.3
Water (%)	79.80	Histidine	2.3	Palmitic	30.3
Calcium (%)	0.21	Phenylalanine	3.6	Palmitoleic	9.9
Phosphorus (%)	0.15	Tyrosine	4.5	Stearic	4.0
Potassium (%)	0.10	Threonine	3.6	Oleic	35.3
Sodium (%)	0.032	Leucine	8.0	Linoleic	13.0
Magnesium (%)	0.02	Isoleucine	4.3	Linolenic	2.5
Iron (mg/100 ml)	0.133	Valine	5.1		
Zinc (mg/100 ml)	0.494	Arginine	5.8		
Sulphur (%)	0.08	Tryptophan	1.2		
Vitamin A (mg/100 ml)	0.154				
Vitamin D (i.u./100 ml)	9.25				
Vitamin E (mg/100 ml)	0.140				
Thiamine (mg/100 ml)	0.071				
Riboflavin (mg/100 ml)	0.277				
Nicotinic acid (mg/100 ml)	0.739				
Pantothenic acid (mg/100 ml)	0.462				
Folic acid (μ g/100 ml)	0.390				
Biotin (μ g/100 ml)	1.40				
Vitamin B ₁₂ (μ g/100 ml)	0.150				
Vitamin C (mg/100 ml)	12.07				

¹Derived from data presented by Lucas and Lodge (1961), Hafez (1974), Pond and Houpt (1978);
²total fatty acids; ³(N x 6.38)

Table 2. The apparent digestibility of several chemical components of sow's milk by 3-week-old piglets¹

Component	Coefficient of digestibility
Dry matter	0.97
Crude protein	0.98
Ether extract	0.99
Ash	0.94
Calcium	0.92
Phosphorus	0.98

¹From Lucas and Lodge (1961)

It can be concluded that milk is an extremely highly digestible food for piglets. Moreover, it also appears that the absorbed nutrients are utilized with a high degree of efficiency. Lucas and Lodge (1961) reviewed the results of several studies and reported a mean food conversion ratio of 0.8:1 (kg milk dry matter:kg live weight gain) for suckled piglets during the first four weeks of life. This very high efficiency of

conversion of milk to body weight reflects the well-balanced and highly available nutrient content of milk. Moughan *et al.* (1989a) determined daily urinary urea excretion and biological value (BV) for five-week-old piglets given a semi-synthetic diet based on maize-starch and containing bovine skim-milk as the sole source of protein. A very low mean excretion of urinary urea (126 mg/kg^{0.75}/day) and a high BV (98%) were found. The BV indicates that the absorbed amino acids from milk protein were almost completely utilized by the piglet.

Table 3. The apparent ileal digestibility of amino acids in bovine milk protein for 5-week-old piglets¹

Amino acid	Coefficient of digestibility	
	Skim-milk powder	60/40 whey/casein ²
Lysine	0.94	0.96
Histidine	0.92	0.96
Arginine	0.92	0.93
Threonine	0.79	0.89
Valine	0.87	0.94
Methionine	0.96	0.97
Isoleucine	0.88	0.96
Leucine	0.92	0.97
Phenylalanine	0.96	0.95
Tyrosine	0.96	0.96
Tryptophan	-	0.94
Cystine	-	0.92

¹Moughan *et al.* (1989a); ²Moughan *et al.*, (1989c).

The frequent sucking behaviour of the piglet coupled with a rapid rate of emptying of milk from the piglet's stomach (Moughan *et al.*, 1989b) ensure a continuous and relatively even supply of this nutritionally highly available fluid to the small intestine. The frequent ingestion of small amounts of a highly digestible milk has major advantages for the piglet, ensuring that the gut is not overloaded, thus predisposing to the development of diarrhoea. The practice of early-weaning (at less than three weeks of age) with free access to solid food, has in the past, been frequently associated with the development of digestive disorders in the piglet (Brent *et al.*, 1975).

Although being a highly available source of most nutrients, milk is known to be deficient in iron and copper (Pond and Houpt, 1978) and if piglets are raised indoors there is a need to provide extra amounts of these minerals. Also, there is evidence that suckled pigs do not necessarily grow to their full potential (Lucas and Lodge, 1961; Williams, 1976). If nutrient intake is increased artificially, the amount of dietary nitrogen retained within the body may increase.

As the piglet grows, the gap between potential growth and the growth actually sustained by milk intake from the sow widens. It is not surprising, therefore, that there is pressure to wean piglets from their dam as soon after birth as possible. In this context, however, it should be realized that milk is not only a food but that it is a biological secretion having several accessory roles.

Components of milk with biological functions other than the provision of nutrients, have been the topic of intensive study in humans. Many compounds have been identified and their physiological functions elucidated. Some of the factors present in human milk are listed in Table 4. Information about the occurrence and function of these compounds in sow's milk is not as readily available as is the case for man. Nevertheless, the importance of several factors is now recognized.

Table 4. Some constituents of human milk possessing non-nutritional roles¹

Protective factors	
Cells	- lymphocytes monocytes macrophages polymorphonuclear leukocytes
Antibodies	- IgG, IgM, secretory IgA
"Non-antibody" factors	- lactoferrin, transferrin, lysozyme, lactoperoxidase, bifidus factor, vitamin B ₁₂ binding protein, anti-viral and anti-protozoal agents
Digestive enzymes	- lipase, α -amylase, bile-salt-stimulated lipase
Growth promoting/ regulatory factors	- epidermal growth factor, insulin, thyroxine, prostaglandins etc.

¹Goldman and Goldblum (1980), Hamosh (1986), Morriss (1986), Pickering and Kuhl (1986), Buescher and Pickering (1986), May (1988)

Immunological and non-immunological host defence mechanisms at the mucosa are underdeveloped during the neonatal period. The intestinal epithelial cells lack a well-defined brush border and retain a primitive transport mechanism for endocytosis of large molecules. The immunological factors present in milk may compensate for this transient deficiency of mucosal immaturity in the neonate (Mandyla and Xanthou, 1986). The milk immunoglobulins are synthesized by plasma cells, some of which are located in the mammary tissue. A large proportion of these plasma cells (and ultimately the antibodies secreted in the milk) are derived from B lymphocytes originally sensitized in the gut against intestinal pathogens (Hartmann *et al.*, 1984).

In mature sow's milk, IgG is found at 3.0, IgM at 0.3 and IgA at 7.7 mg/ml (Jenness, 1986). The immunoglobulins are glycoproteins and consequently are not readily hydrolyzed in the gastrointestinal tract of the piglet (Hartmann *et al.*, 1984). As may be expected the quantity of immunoglobulins in sow's colostrum and presumably mature milk increases with parity (Inoue, 1981) and there may also be an effect of breed of sow (Smith, Schollum, Moughan, unpublished data). Secretory IgA from the milk may be particularly important to the new-born piglet's defence system. It is not until around the third week of life that the piglet produces its own secretory IgA in significant amounts. The immunoglobulins in milk protect against bacterial and viral pathogens primarily by preventing adhesion of the pathogens to mucosal epithelial cells. In the pig, milk immunoglobulins have been shown to inhibit the growth of *E. coli*, block its adhesion to enterocytes and neutralize the heat labile toxin produced by the bacteria (Hampson, 1987).

The commercial production of milk immunoglobulin concentrate may offer a means of providing some protection to early-weaned piglets. It is now possible to vaccinate cows with specific pathogens to induce the production of specific antibodies, which can then be concentrated and administered to the piglet (Reiter, 1985). More recently, Drew and Owen (1988) have shown that concentrated porcine serum immunoglobulins obtained from abattoir blood can be used as an addition to the diet to provide protection for early-weaned pigs.

Non-antibody factors such as lactoferrin, transferrin, vitamin B₁₂-binding protein and the bifidus factor have been shown to be present in sow's milk (Gyorgy *et al.*, 1954; Trugo and Newport, 1983; Elliott *et al.*, 1984; Jenness, 1986) and are likely to play a

part in the piglet's defence against disease. Lysozyme has also been found in sow's milk and is believed to have a significant effect on the bacterial colonization of the gastrointestinal tract of the unweaned piglet (Schulze and Muller, 1980). The existence of lactoperoxidase in sow's milk appears not to have been investigated (Jenness, 1986). Lactoferrin seems to be resistant to proteolysis in the gut and in the presence of bicarbonate binds ferric ions. It has been shown in *in vitro* studies to exert a strong bacteriostatic effect towards iron-requiring strains of bacteria (Friend *et al.*, 1983). Lactoferrin obtained from milk has also been shown to be an essential growth factor for human B and T lymphocytes and may have a further role in stimulating growth of intestinal cells (Duke and Headon, 1988). Lonroth *et al.* (1988) have reported that a hormone-like protein (antisecretory factor) is present in sow's milk and that it is capable of reversing intestinal hypersecretion and seems to provide protection against diarrhoea in sucking piglets.

The bifidus factor in sow's milk may promote the selective growth of the bifidobacteria (*Bifidobacterium thermophilum* and *Bifidobacterium pseudolongum*; Mitsuoka, 1981) in the intestine of the piglet, as is the case for the human infant. These acid-producing organisms are capable of lowering the pH of the gut, thus retarding the growth of harmful bacteria such as *E. coli*, bacterioides and clostridia. Lactoferrin may indirectly promote the growth of bifidobacteria by inhibiting the growth of competing bacteria. Bifidobacteria do not require iron for growth (Duke and Headon, 1988).

Sow's milk contains appreciable quantities of lipase (Chandan *et al.*, 1968) which may aid the digestion of milk lipids in piglets. Also, sow's milk may yet be found to contain a bile-salt stimulated lipase (Freed *et al.*, 1986), which was previously thought to be found only in human and gorilla milk. Milk also contains hormones and growth-promoting factors and other compounds that may have regulatory roles, the physiological functions of which are only poorly understood. For example, Jaeger *et al.*, (1987) have detected measurable quantities of epidermal growth factor and insulin in sow's colostrum and in sow's milk throughout a 27 day lactation. These compounds may have a range of specific physiological functions in the piglet and may also be important in the maturation of the digestive system (James *et al.*, 1981a) and in the induction and modulation of hormones and peptides secreted by the gut (Lucas, 1986). The pioneering work of Widdowson *et al.*, (1976) suggests a growth promotional effect of sow's milk; they found a dramatic increase in the mass of gastrointestinal tract of suckled newborn pigs compared to starved littermates 24 h after birth.

Finally, it is becoming increasingly clear that there are complex interactions between the non-nutritional factors in milk. An example of synergism relevant to the piglet has been described by Reiter (1985). Newborn piglets were dosed with *E. coli* and then given bovine colostrum with or without activated lactoperoxidase. The four piglets receiving the colostrum plus lactoperoxidase remained in good health while three of the four control animals developed severe diarrhoea. It is concluded that milk is far more than a source of nutrition. It is a natural secretion with a complex composition, having a wide range of potentially important physiological functions. When piglets are weaned from their dam, they not only have to adapt to a new form of food, but also to the loss of supply of a range of biologically active compounds involved in the development and maintenance of a healthy and functional digestive system.

Development of the gastrointestinal system

The components of the digestive system which are mainly responsible for the digestion and absorption of food are the stomach, small intestine, pancreas and liver. The ability of the pig to carry out digestive and absorptive functions will depend on the physical capacity of the gut, the nature and amount of the secretions it can provide, e.g.

acid, enzymes, bicarbonate and bile, the development of mechanisms to control these secretions, and the digestive and absorptive capacity of the mucosal surface of the small intestine. While information about the development of all these functions of the gastrointestinal system of the sucking pig is not readily available, those components that have been reasonably well studied provide an insight into the limitations of the gut of pigs at 3-5 weeks of age when they are normally weaned. In this section we will describe some aspects of gut development which are important.

Size and capacity in relation to body size

From 28-36 days gestation until birth (115 ± 3 days gestation) the growth rates of the stomach, intestines and pancreas are positively allometric relative to that of the whole foetus (Marrable, 1971). Provided that the newborn pig is suckled by the sow, this pattern of growth continues for up to 3 days after birth (Table 5). After this time, and while the pig's sole source of food is sows' milk (up to 28-36 days of age), the rate of growth of the stomach, small intestine and pancreas are isometric or negatively allometric relative to that of body weight (Table 5). In contrast, the growth rate and the weight of these organs in relation to body weight in pigs which have been weaned for at least two weeks is considerably greater than the values shown in Table 5.

Table 5. The relationship between the weight (stomach, small intestine, pancreas) or length (small intestine) and body weight (BW) in sucking pigs from birth to 38 days of age

	Age (days)						
	0	1-3	5-12	14-18	21-23	24-28	29-38
Stomach (g/kg BW)							
Widdowson <i>et al.</i> (1976) ¹	4.0	4.5	4.2	2	-	-	-
Braude <i>et al.</i> (1981) ¹	4.5	5.5	5.1	4.8	-	5.1	-
Cranwell (1985a) ¹	-	-	5.9	4.7	-	5.2	-
Cranwell (1985b) ¹	-	5.3	-	4.3	-	4.7	-
Xu (1989) ¹	4.9	5.2	5.3	4.6	4.2	4.5	4.0
Small Intestine (g/kg BW)							
Widdowson <i>et al.</i> (1976)	23	33	29	-	-	-	-
Braude (1981)	24	27	26	27	-	26	-
Efird <i>et al.</i> (1982a) ¹	-	34	33	33	26	-	-
Cera <i>et al.</i> (1988) ¹	-	34	32	-	30	36	41
Small Intestine (m/kg BW)							
Vodovar <i>et al.</i> (1964)	2.1	2.6	1.9	1.7	-	-	0.8
Widdowson <i>et al.</i> (1976)	3.0	3.3	1.8	-	-	-	-
Shields <i>et al.</i> (1980) ¹	3.1	-	-	1.6	-	-	-
Pancreas (g/kg BW)							
Friend <i>et al.</i> (1970) ¹	-	-	-	-	1.1	-	1.1
Widdowson and Crabb (1976) ¹	1.0	1.6	1.4	-	-	-	-
Corring <i>et al.</i> (1978) ³	1.0	-	1.5	1.4	1.4	1.6	1.7
Shields <i>et al.</i> (1980)	1.7	-	-	0.8	-	-	-
Efird <i>et al.</i> (1982a)	-	1.0	1.2	1.1	1.0	-	-
Lindemann <i>et al.</i> (1986) ¹	0.8	-	1.4	1.1	1.2	1.1	-
Owsley <i>et al.</i> (1986a) ⁴	1.0	-	-	1.2	-	1.2	-

¹No creep-feed; ²no observations at this age; ³creep-feed from 10 days of age; ⁴creep-feed from 14 days of age

In weaned pigs, the growth of the stomach from 35-115 days of age and 10-40 kg body weight was positively allometric relative to body weight. Stomach weight in

relation to body weight increased from 5.9-6.4 g/kg over this weight range (Cranwell, 1985b). In another experiment with pigs of 40-41 kg body weight, Koong *et al.* (1982) reported stomach weight to body weight ratios of 6.5-8.4 g/kg. The relative weight of the mucosa of the stomach also undergoes a significant increase following weaning. Lindemann *et al.* (1986) reported gastric mucosa weight to body weight ratios of 1.5-2.2 g/kg during the four weeks prior to weaning compared with values of 3.0-3.8 g/kg in the two weeks following weaning.

For the small intestine the difference is even more dramatic and is illustrated by the results of Cera *et al.* (1988) who reported relative small-intestine weights of 55-60 g/kg in 42 day old pigs which were 9.8-10.7 kg body weight and had been weaned for 21 days. These results represent an 84-98% increase in the relative size of the small intestine during the 21 day post-weaning period or an actual increase of 406-432 g of small intestinal tissue. Similar results were reported in one of the studies by Efird *et al.* (1982b) in which pigs of 35 days of age, weaned at 21 days, had relative small-intestine weights of 55-57 g/kg. The length of the small intestine relative to body weight continues to decrease with age in weaned pigs as it does in sucking pigs (Table 5). This can be seen from the results of Vodovar *et al.* (1964) and Shields *et al.* (1980) who reported values of 0.56 m/kg in pigs 8-10 weeks of age. However, the diameter of the small intestine and its capacity were found to undergo 3.5 fold and 43 fold increases from birth to 8-10 weeks of age respectively (Vodovar *et al.*, 1964).

In the two studies by Lindemann *et al.* (1986) and Owsley *et al.* (1986a), the relative size of the pancreas in pigs, 7-17 kg body weight, at 2 and 4 weeks after weaning was 1.54-2.19 g/kg, which is significantly greater than that in pigs prior to weaning (Table 5). The older pigs were found to have 38-81% more pancreatic tissue/unit body weight than pigs at weaning. In the study by Shields *et al.* (1980) the relative size of the pancreas at 2, 4 and 6 weeks after weaning at either 2 or 4 weeks of age was 117-187% greater than that in 2 week old sucking-pigs (Table 5). Other values for the relative weight of the pancreas have been obtained from papers by Efird *et al.* (1982b), 1.5-2.1 g/kg in pigs 2-3 weeks after weaning, and Koong *et al.* (1982), 1.3-2.0 g/kg in pigs of 41 kg body weight.

These observations serve to illustrate that the weaned pig requires a relatively larger digestive system than a sucking-pig if it is to satisfactorily digest and absorb the inherently less digestible post-weaning diets and maintain a satisfactory growth rate. The period of time it takes the pig to upgrade its digestive system is thus one of the limitations affecting its post-weaning performance. The development of the components of the gastrointestinal system dealt within this section will now be considered in more detail.

The stomach

Anatomy and histology

The gastric mucosa of the pig consists of four distinct zones (Nickel *et al.*, 1973) which are readily identifiable macroscopically from about 10-21 days of age (Table 6; Noakes, 1971). The proportion of mucosa (including epithelium, *lamina propria* and *muscularis mucosae*) to muscle (including sub-mucosa, *muscularis externa* and serosa) is 56:44 (Cranwell, 1985b). The fundic zone has the greatest weight and surface area and is thicker than the other zones. With the exception of the *pars oesophagea*, which is lined with stratified squamous, nonglandular epithelium, the luminal surface and gastric pits of the other three regions are lined with simple columnar epithelial cells which secrete mucus (Ito, 1987) and bicarbonate (Flemstrom, 1987). The types of cells found in the gastric glands of each zone of mucosa and the nature of their secretions are presented in Table 7. The principle digestive secretions produced by the stomach are hydrochloric acid and various proteolytic enzymes.

Regulation of gastric secretion

Regulation of gastric secretion is complex, with the chemical signals mediating regulation, emanating from three major routes: Neurocrine, endocrine and paracrine (Sanders and Soll, 1986). The major endocrine peptides involved in the control of gastric secretion in the pig are gastrin (stimulatory) and somatostatin (inhibitory) although the latter also acts as a paracrine and possibly a neurocrine transmitter (Holst, 1985, 1986). Another important paracrine substance involved in gastric secretion is histamine which is delivered from mast cells located in the *lamina propria*; it is a potent stimulator of the parietal cell (Sanders and Soll, 1986). The most important neurocrine transmitter is acetylcholine which, apart from its stimulatory action on the parietal cell, also stimulates proteolytic enzyme, bicarbonate and mucous secretion. Inhibitory neurocrine effects on gastric secretion are indirect and are mediated by β -adrenergic agents which stimulate somatostatin release and inhibit histamine release (Sanders and Soll, 1986). Another neurocrine transmitter of considerable importance is gastrin-releasing peptide (GRP) which is released following vagal stimulation and causes the secretion of gastrin by gastrin cells (G-cells) (Knuhtsen *et al.*, 1984).

Table 6. The surface area and the proportion of the total mucosal weight of the four zones of the gastric mucosa in sucking-pigs (10-56 days of age)

Zone of mucosa	Surface area (%) ¹		Weight (% of total mucosa) ²	
	Mean	SE	Mean	SE
<i>Pars oesophagea</i>	5.6	0.3	4.8	0.5
Cardiac	30.1	0.8	19.8	0.7
Fundic (proper gastric)	44.4	0.9	55.9	1.4
Pyloric (antrum)	20.0	0.7	19.5	1.0

¹Noakes (1971); ²Cranwell (1985b)

Table 7. The gastric glands, secretory cells and exocrine and endocrine secretions of the stomach of the pig

Glands	Cells	Secretions
Cardiac	Mucous neck	Mucus
		Proteases Lipase?
Proper gastric (fundic)	Mucous neck	Mucus
		Proteases
	Parietal (oxyntic) Chief (zymogen) Enteroendocrine	HCl
		Proteases
		Somatostatin
Pyloric (antrum)	Mast cells (<i>lamina propria</i>)	Serotonin (5-hydroxy tryptamine)
		Histamine
	Mucous neck	Mucus
		Proteases
		Proteases
Chief (zymogen) Enteroendocrine	D-cells G-cells	Somatostatin
		Gastrin

The phases of regulation of gastric secretion are cephalic stimulation, gastric distension, chemical stimulation and chemical inhibition in the stomach, and postgastric inhibition. Of these, chemical stimulation probably accounts for the greatest proportion of the gastric secretory response (Walsh, 1984). It is mediated mainly by direct contact between the chemical stimulants in the ingested and partially digested food in the lumen of the stomach and the brush borders on the luminal surface of G-cells found in the pyloric antrum. The most potent chemical stimulators of gastrin secretion appear to be ammonia, amines and amino acids while intact proteins are relatively poor gastrin secretory stimulants, and neither fat nor carbohydrate are effective stimulants of gastrin release (Lichtenberger, 1982).

Chemical inhibition of gastric secretion occurs in response to the presence of acid in gastric contents. When the gastric pH is lowered below 3 gastrin release is inhibited by two mechanisms. Firstly, free acid has a direct inhibitory effect on G-cell secretion via its luminal surface (Walsh, 1984). Secondly, free acid, detected by the luminal surface of the antral D-cell, stimulates the secretion of somatostatin which in turn inhibits the secretion of neighbouring G-cells (Holst, 1985). Of all the mechanisms controlling gastric secretion, both stimulatory or inhibitory, Walsh (1984) considers those which involve the control of gastrin release to be the most important.

Development of HCl secretion

In a recent study with foetal pigs Foltmann *et al.* (1987a) presented evidence that acid secretion commences at least 11 days before term. No information is available as to how the gastric secretory response is mediated in the foetal pig. However it is known that the stomach of the newborn pig is capable of secreting acid in response to histamine (Forte *et al.*, 1975), its analogue, histalog (Tudor, 1983; Cranwell, 1985c), pentagastrin (Cranwell and Xu, 1986; Cranwell *et al.*, 1987) and feeding (Cranwell and Titchen, 1974; Cranwell *et al.*, 1976). The capacity of the stomach to secrete acid in response to either pentagastrin or histalog, though is much lower in the newborn than in 1-2 week-old sucking pigs (Cranwell, 1985c; Xu, 1989). For example pentagastrin-stimulated acid output/unit stomach weight in sucking-pigs undergoes a five-fold increase during the first week of life compared with an overall seven-fold increase during the first 5 weeks of life (Xu, 1989).

Positive linear relationships between maximal acid output and body weight have been reported by Xu (1989) and Cranwell (1985c) in pigs from birth to 5-6 weeks of age. In the study by Cranwell (1985c), in which histalog was the secretagogue, sucking pigs which had received no creep-feed were compared with litter-mates which were reared by the sow for 21 days, but were allowed access to solid food (creep-feed) at 14 days and were entirely dependant on solid food after weaning. The results from this experiment are presented in Figure 1. The slope of the regression line for the pigs fed solid food (C-pigs) was significantly greater ($P < 0.05$) than that for the sow-reared pigs (M-pigs). This indicates that the gastric acid secretory capacity develops more rapidly in animals provided with creep feed and weaned on to solid food.

From the regression equations in Figure 1 it is possible to calculate the acid secretory capacity of both M- and C- pigs of similar body weights at 3 (4kg) and 6 weeks (13 kg) of age. During the intervening 3 week period the maximal acid output/unit body weight of the M-pigs remained constant (0.76-0.77 mmol/kg/h) whereas that for the C-pigs increased from 1.00 mmol/kg/h at 3 weeks to 1.15 mmol/kg/h at 6 weeks. Thus, the acid secretory capacity of the C-pigs was 32% and 49% greater than the M-pigs at the two ages respectively. The greater acid secretory capacity of the C-pigs was related to their having more stomach tissue/unit body weight and to the stomach tissue itself being capable of producing more acid/unit stomach weight (Cranwell, 1985a).

Apart from a brief report by Abin *et al.* (1983), whose results are consistent with those of Cranwell (1985c), no other reports on acid secretion in weaned pigs up to 6 weeks of age have been found in the literature. Estimates of acid secretory capacity in older pigs 14 to 36 kg body weight by Muggenburg *et al.* (1967), Barbezat *et al.* (1974), Fujita *et al.* (1980) and Watson *et al.* (1985) are of limited value because outputs were not expressed on a per unit body weight basis, the precise body weights of the animals at the time of the experiments were not provided, and only the range in body weights of the animals used was given. Bearing in mind these limitations, the imprecision of the calculations used to determine outputs/unit body weight and the differences in the experimental techniques used, there is reasonably close agreement between the maximal acid output in these older pigs (0.5-1.5 mmol/kg/h) with that in weaned pigs 6-13 kg body weight, (1.13-mmol/kg/h) reported by Cranwell (1985c). This similarity in acid output/unit body weight suggests that as pigs grow older gastric secretory capacity remains correlated with body weight in a manner similar to that found for weaned pigs by Cranwell (1985c). However further more precise research is necessary to confirm this and to determine which are the important factors involved in the development of gastric secretion in the pig.

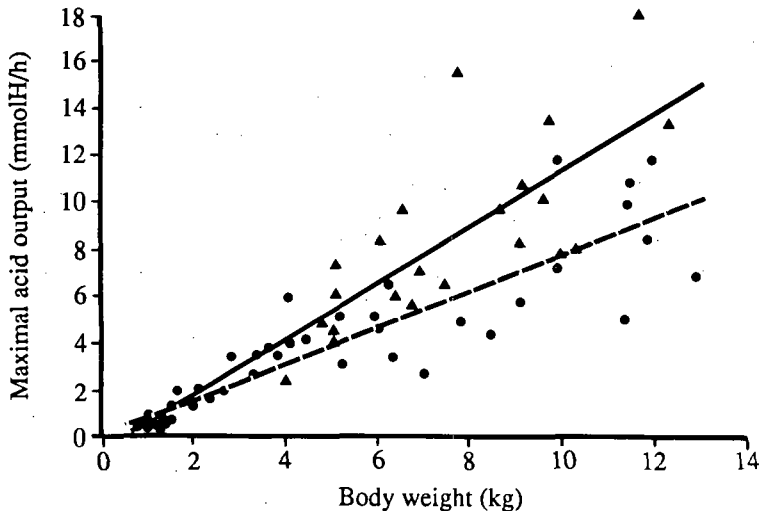


Figure 1. Linear regression of maximal gastric acid output in response to intravenous histalog infusion at 3 mg/kg/h vs. body weight in 70 pigs (35 littermate pairs from 12 litters, <24 hours to 42 days old, 1-13 kg body weight). Using a split litter technique one pig from each pair was fed solely by the sow (M-pig; circles) whereas the other pig was fed by the sow for 21 days, but allowed access to solid food at 12-14 days and fed only solid food from 21 days (C-pig; triangles). The regression equations were:

$$M\text{-pigs (broken line)} Y = 0.77X - 0.06 \quad (r^2 = 0.81; P < 0.001; df = 34) \text{ and}$$

$$C\text{-pigs (solid line)} Y = 1.21X - 0.83 \quad (r^2 = 0.76; P < 0.001; df = 34),$$

where Y = Maximal acid output (mmol/h) and X = body weight (kg) (from Cranwell, 1985c).

Although the gastric secretory capacity of pigs can be determined quite accurately it is much more difficult to measure how much acid (or gastric juice) is actually secreted in response to the intake of a known quantity of food. No precise information on this aspect of gastric secretion could be found in the literature.

Ontogeny of the gastric proteases

The three major proteases found in the gastric mucosa of the young pig are chymosin (rennin, EC 3.4.23.4), pepsin (pepsin A, EC 3.4.23.1) and gastricsin (pepsin C, EC 3.4.23.3) (Foltmann, 1981a; Foltmann *et al.*, 1981).

Following its identification in and extraction from porcine mucosa (Foltmann *et al.*, 1978), the properties of pig chymosin have been extensively investigated (Foltmann and Axelsen, 1980; Foltmann, 1981a, b; Foltmann *et al.*, 1981; Foltmann *et al.*, 1985; Foltmann, 1986). Pig chymosin is primarily a milk-clotting enzyme with limited proteolytic activity and it has a milk-clotting activity to general proteolytic activity ratio that is more than ten times greater than pig pepsin (Table 8). Its zymogen, prochymosin, requires only hydrogen ions to initiate the formation of fully active chymosin and this can occur at pH 5.5. Although immunologically and enzymically similar to calf chymosin, pig chymosin has a much higher clotting activity against porcine milk than bovine milk, as does pig pepsin (Table 8).

Table 8. Characteristics of porcine pepsin A and chymosin

	Optimum pH	Relative proteolytic activity @ optimum pH	Relative milk-clotting activity	
			bovine milk	porcine milk
Pepsin A	2.0	100	100	72 (2.7x) ¹
Chymosin	3.5	2	58	100 (6.4x)

¹Times greater activity in porcine milk than in bovine milk (Foltmann 1981a, b; Foltmann *et al.* (1981)

In a recent study of the effect of age and chronic ACTH treatment from 3 days of age on the development of the gastric proteases in the fundic mucosa of foetal pigs from 93 days gestation and sucking-pigs from birth to 36 days of age the concentrations of the individual proteases were measured by quantitative rocket immunoelectrophoresis (Sangild *et al.*, 1989a). The results (Figure 2) confirm and extend the earlier findings of Foltmann *et al.* (1981, 1987b) and show that prochymosin is present in the fundic mucosa 22 days before birth, and that its concentration reaches a peak at birth. Although the concentration of prochymosin decreased with age there were still significant amounts present at 36 days of age. Pepsinogen was not detected in the fundic mucosa until 5-7 days of age and progastricsin not until 9-11 days of age; the concentrations of both increased with age during the course of the experiments with pepsinogen being predominant.

Chronic administration of ACTH significantly increased adrenal size and circulatory cortisol concentrations in treated pigs. However, it did not induce the precocious appearance of pepsinogen (or progastricsin) in the fundic mucosa (Figure 2) which has been observed in a number of experiments with laboratory rats and mice during the first two weeks of life (Pelletier *et al.*, 1983; Tseng and Johnson, 1986). It was not until the pigs were 5 weeks old that ACTH treatment was seen to have a significant effect on the concentration of pepsinogen and progastricsin in fundic mucosa. The pentagastrin stimulated secretion of milk-clotting activity and proteolytic enzyme activity by both treated and control pigs shows a development which is related to the concentration of enzymes in fundic tissues (Figure 3; Sangild *et al.*, 1989b).

Apart from the studies of Foltmann *et al.* (1981, 1987a, b) and Sangild *et al.* (1989a,b) investigations into the development of gastric proteases in the young pig have been confined to measuring the proteolytic activity of extracts of gastric mucosa and gastric contents (Cranwell, 1985a; Lindemann *et al.*, 1986), and of gastric secretions (Cranwell and Titchen, 1976; Cranwell and Stuart, 1983; Cranwell, 1985a,c). The results of these studies would therefore reflect the development of pepsin and gastricsin rather than chymosin. The results complement the findings of Foltmann *et al.* (1981, 1987a, b) and Sangild *et al.* (1989 a, b) referred to above and show that the proteolytic enzyme content of the gastric mucosa and gastric secretions are low in pigs up to 3-4 weeks of

age and then undergo a very rapid increase. This pattern of development is reflected by the results of Braude *et al.* (1970) and Leibholz (1981) which show that the 28-day-old pig has only limited ability to hydrolyze dietary protein (either casein or soya) in the stomach, and the later findings of Leibholz (1985) that this ability increases markedly during the next 28 days so that at 56 days of age about 53% of dietary casein and 35% of dietary soya is hydrolyzed in the stomach. The capacity of the stomach to hydrolyze dietary protein continues to increase to at least 150 days of age (Zebrowska, 1973). In addition to age, the results of earlier studies referred to, and those reported by Cranwell (1985a,c) show that access to solid food before weaning and weaning on to solid food have significant positive effects on the capacity of the stomach to secrete proteolytic enzymes. This is illustrated in Figure 4 which is taken from the study by Cranwell (1985c). In this study the pigs which had been allowed creep-feed from 12-14 days had at 18-22 days (weaning) a gastric proteolytic enzyme secretory capacity (per unit body weight) which was 300% greater than that in the pigs fed solely by the sow. This difference was still of the same order at 6 weeks of age.

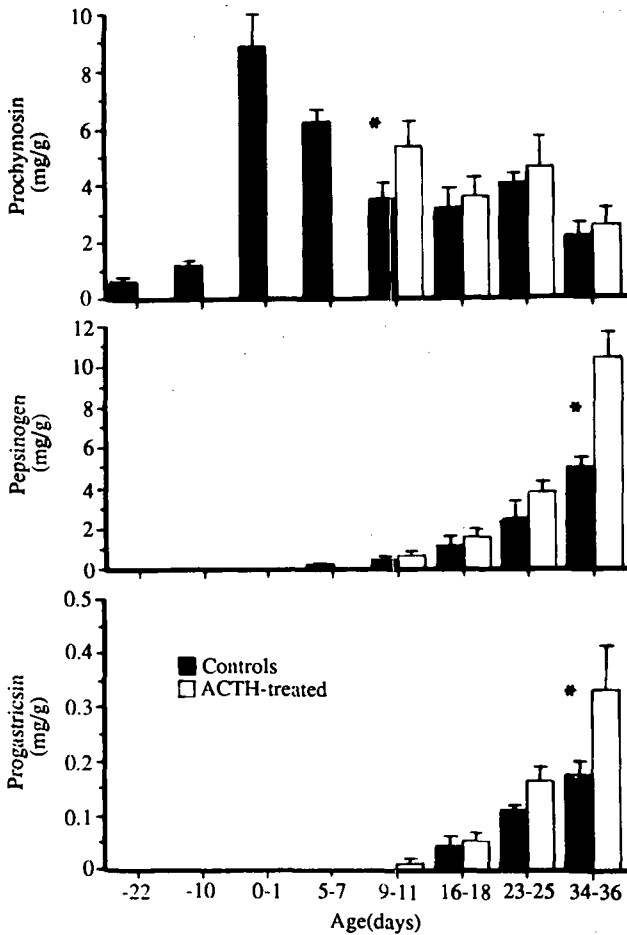


Figure 2. Concentration of zymogens in tissue from the fundic region of the stomach of 10 foetal pigs (93 days and 105 days gestation), 9 sucking pigs (0-7 days of age), and 36 sucking pigs (9-36 days of age)(18 littermate pairs from 5 litters). One pig of each pair was injected (i.m.) with 12.5 µg ACTH/kg^{0.75} twice daily from 3 days of age, the other pig was injected twice daily with physiological saline (from Sangild *et al.*, 1989a).

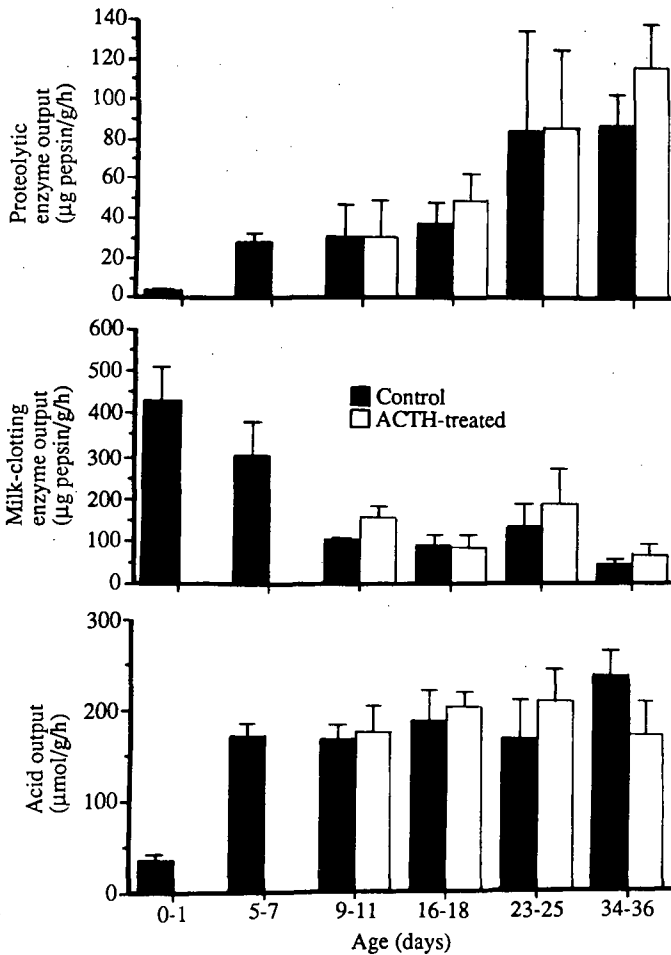


Figure 3. Maximal gastric outputs of milk-clotting activity, proteolytic activity and acid/unit stomach weight in response to intravenous pentagastrin infusion at 4 and 8 $\mu\text{g}/\text{kg}/\text{h}$ in sucking pigs (see figure 2 for details of pigs and treatments) (from Sangild *et al.* 1989b).

The Pancreas

Anatomy and histology

The anatomy of the porcine pancreas has been described by Nickel *et al.* (1973). The structural components of the exocrine pancreas are the acinar unit and the duct system. The acinar unit is comprised of acinar cells which produce and store the various pancreatic zymogens. When stimulated these cells secrete zymogens into the acinar lumen from where they are transported to the small intestine through the pancreatic duct system. The cells lining the first two components of the duct system, the intercalated and intralobular ducts, are the main sources of bicarbonate, other ions and water in pancreatic juice. A more detailed account of the exocrine pancreas can be found in the reviews by Scheele and Kern (1986) and Gorelick and Jamieson (1987).

Regulation of exocrine pancreatic secretion

As is the case for gastric secretion, pancreatic secretion is controlled by a tight interplay between endocrine, paracrine and neurocrine control mechanisms (Holst,

1985). The major components involved in the stimulation of secretion by the pancreas are the vagus nerve and the two hormones secretin and cholecystikinin (CCK). The main stimulus for bicarbonate secretion from the duct cells is secretin which is released from S-cells in the upper jejunum in response to the presence of acid in the duodenum and jejunum (Holst, 1985, 1986; Harada *et al.*, 1986). In addition it has been demonstrated that vagal stimulation brings about bicarbonate secretion which is independent of the action of secretin (Holst, 1986). It is probable that the neurotransmitter responsible is vasoactive intestinal peptide (VIP). Both the vagus nerve and CCK are important in the control of enzyme secretion by the acinar cells (Harada *et al.*, 1982; Holst, 1985; Scheele and Kern, 1986). The stimuli which evoke the enzyme secretory response include fatty acids, amino acids, peptones and acid (Hong and Magee, 1970; Harada *et al.*, 1986). Further details about the regulation of pancreatic secretion, including the potentiating effects which secretin, CCK, VIP and acetylcholine have on each other, are to be found in the reviews by Holst (1986) and Scheele and Kern (1986).

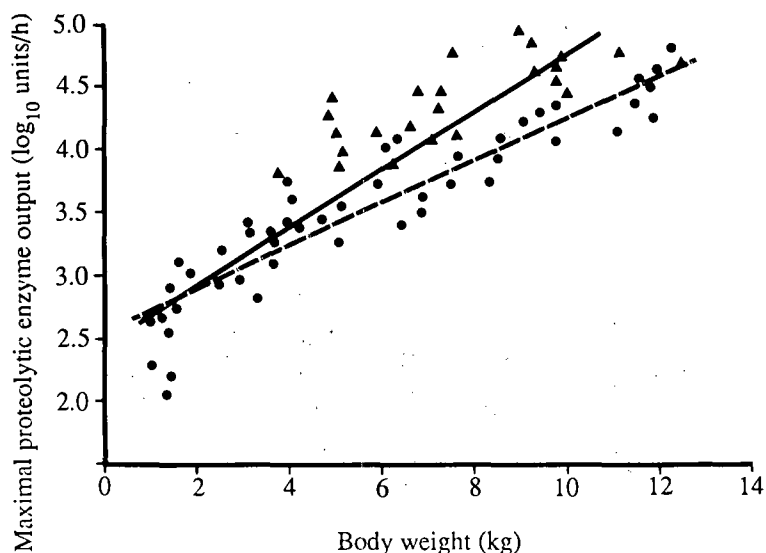


Figure 4. Linear regression of maximal gastric proteolytic enzyme output in response to intravenous histalog infusion at 3 mg/kg/h vs. body weight in 70 pigs (see figure 1 for details of pigs and treatments). The regression equations were:

$$M\text{-pigs (broken line)} Y = 0.16X + 2.58 \quad (r^2 = 0.85; P < 0.001; df = 34) \text{ and}$$

$$C\text{-pigs (solid line)} Y = 0.23X + 2.50 \quad (r^2 = 0.85; P < 0.001; df = 34),$$

where Y = Maximal proteolytic enzyme output (\log_{10} units/h) and X = body weight (kg) (unit = Unit of proteolytic activity defined as equivalent to a ΔA_{280} of 0.001/min at pH 2.0, 37°C) (from Cranwell, 1985c).

Development of exocrine pancreatic secretion

Studies on the development of pancreatic secretion, as distinct from studies on the occurrence and changes in concentration of the many different zymogens in pancreatic tissue, are few in number. It is only recently that the secretory responses of the exocrine pancreas to vagal stimulation, CCK and secretin administration, and intraduodenal infusion of HCl have been studied in pigs younger than 28 days of age (Harada *et al.*, 1988). In this study the magnitude of the response to secretin administration or HCl infusion at 3 days of age indicates that the pig at this age is capable of secreting quantities of pancreatic juice which, when expressed on a body

weight basis (1.6-2.2 ml/kg/h), are within the same range as those secreted by sucking pigs of 6-28 days of age, i.e. 0.9-2.3 ml/kg/h. Also, the rates of pancreatic secretion in the sucking pig are comparable with those recorded in older pigs in response to secretin administration; e.g. 1.5 ml/kg/h (15-20 kg pigs; Magee and White, 1965), 2.7 ml/kg/h (17-25 kg pigs; Holst *et al.*, 1979); and in response to a meal, e.g. 2.4 ml/kg/h (42 kg pig; Corring *et al.*, 1972).

When the ionic concentration of pancreatic juice secreted by 3 day old pigs in response to secretin infusion was compared with that from 8 week old pigs it was found that chloride, sodium and potassium concentrations were similar whereas bicarbonate concentrations in the 3 day old pigs were only half that found in the 8 week old pigs (55 vs. 110 mmol/l; Harada *et al.*, 1988). In older pigs, 17-25 kg body weight, Holst *et al.* (1979) reported bicarbonate concentrations in pancreatic juice following secretin infusion of 130-143 mmol/l and peak bicarbonate outputs of 0.29-0.43 mmol/kg/h. These outputs are 3-5 times greater than those calculated from the results of Harada *et al.* (1988) for 3-day-old pigs (0.09 mmol HCO₃/kg/h).

In the study by Harada *et al.* (1988) the protein content of pancreatic juice secreted by sucking pigs in response to CCK infusion underwent a 3-4 fold increase during the period from 3-14 days of age (5-17 µg/µl), the concentrations at 14, 21 and 28 days of age were all similar. The only specific enzyme measured by Harada *et al.* (1988) was amylase, which as a proportion of total protein in CCK stimulated pancreatic juice, increased gradually from 3-21 days and then underwent a 2-3 fold increase at 28 days of age.

Ontogeny of pancreatic enzymes

The four major classes of digestive enzymes produced by the pancreas are the proteases, carbohydrases, lipases and nucleases, and at least 19 different individual enzymes and cofactors have been identified (Kidder and Manners, 1978). Studies on the development of these enzymes in the pig have been confined mainly to estimations of their concentration in pancreatic tissue. Direct comparison of the results from different studies is complicated by a number of factors including differences between the breeds and strains of pig studied, the methods and units of measurement used for the estimation of the various enzymes, the design of the experiments and the methods used to analyse the results, the diets and feeding regimes used throughout the experiment and prior to slaughter, and the age of the pigs at weaning. An important consideration in studies of the development of the different enzymes is whether the observed changes are regulated solely by the age of the pig, or by the age and body weight at weaning, or the physical and chemical nature of the pre- and post-weaning diets, or by interactions between a number of these factors.

Of the more recent publications dealing with the development of the pancreas only three have provided comprehensive information on the changes in the pancreatic content of a number of enzymes throughout the period from birth to 6-8 weeks of age. Unfortunately in none of these studies was there a control group of pigs which was suckled by the sow throughout the experimental period and not given access to solid food. Also, direct comparison of the results from each of these studies is not possible because they differ in experimental design and in several other ways as discussed above. For instance, Corring *et al.* (1978) studied sucking pigs which were given access to creep feed from 10 days of age and were suckled by the sow for the entire 8 weeks. Prior to slaughter at different ages the pigs were fasted for 15 h. In the study by Owsley *et al.* (1986a) pigs were given access to creep feed at 14 days of age and were weaned at 4 weeks. Prior to slaughter the sucking pigs were suckled by the sow, whereas the weaned pigs were fasted for 15 h and then allowed access to solid food for 45 min. Lindemann *et al.* (1986) in their study also weaned pigs at 4 weeks of age but did not provide any creep feed. Prior to slaughter all pigs were fasted for 15 h.

A summary of the findings from these three studies is presented in Table 9 in which the results have been expressed as total pancreatic enzyme content/unit body weight at different ages in relation to that in unsuckled pigs at birth (taken as 1.0). In an attempt to correct for the differences in the concentration of enzymes in the pancreas between animals which had been fed prior to slaughter (Owsley *et al.*, 1986a) with those that had been fasted (Corring *et al.*, 1978; Lindemann *et al.*, 1986) the amounts of the various pancreatic enzymes in the contents of the small intestine have been added to the amounts in the pancreas as indicated in Table 9.

In all three studies it was evident that the relative amounts of the proteases, trypsin and chymotrypsin, remained fairly constant during the first 4-5 weeks of life (Table 9). In the subsequent 2-4 weeks there was a significant increase in the relative amounts of both enzymes, particularly trypsin. For chymotrypsin the increase was due mainly to the increase in the relative size of the pancreas (see earlier section) whereas for trypsin there was also a significant increase in its concentration in pancreatic tissue. In the absence of the data from a control group of animals which only received sows' milk it is not possible to determine to what degree age as distinct from dietary change was responsible for the observed increases in these two enzymes. However, the results of the study by Corring *et al.* (1978), in which sucking pigs ate a progressively greater amount of solid food in relation to their intake of sows' milk would suggest that change in the nature of the diet does have some influence on the development of these two enzymes.

Table 9. The relative amounts of four pancreatic enzymes during the first 6-8 weeks of life in the pig. The figures represent the total amount of each enzyme in the pancreas/unit body weight (enzyme units/kg) relative to that in unsuckled pigs at birth (taken as 1.0)

	Age (days)							
	0	7	14	21	27-28	31-38	42	56
Trypsin								
Corring <i>et al.</i> (1978) ^{2,3}	1.0	1.1	0.5	0.4	1.5	- ¹	4.5	11.9
Owsley <i>et al.</i> (1986a) ⁴	1.0	-	0.5	-	1.1	1.6	10.7	14.3
(Pancreas + SI) ⁵	1.0	-	1.9	-	1.7	5.6	27	29
Lindemann <i>et al.</i> (1986) ^{3,6}	1.0	1.8	1.7	1.7	2.3	1.3	4.0	-
Chymotrypsin								
Corring <i>et al.</i> (1978)	1.0	1.6	1.1	1.3	1.5	-	1.9	1.8
Owsley <i>et al.</i> (1986a)	1.0	-	1.0	-	1.5	0.5	2.3	2.3
(Pancreas + SI)	1.0	-	1.3	-	1.8	1.8	4.0	3.7
Lindemann <i>et al.</i> (1986)	1.0	2.3	2.5	2.8	3.3	1.1	3.1	-
Amylase								
Corring <i>et al.</i> (1978)	1.0	2.1	3.1	3.6	14.5	-	42.7	48.8
Owsley <i>et al.</i> (1986a)	1.0	-	28.9	-	59.7	27.8	143.8	177.4
(Pancreas + SI)	1.0	-	30.8	-	63.0	32.5	166.9	194.5
Lindemann <i>et al.</i> (1986)	1.0	23.9	57.1	83.8	101.2	25.9	106.7	-
Lipase								
Corring <i>et al.</i> (1978)	1.0	5.1	5.6	9.7	19.0	-	23.8	16.1
Lindemann <i>et al.</i> (1986)	1.0	2.1	5.1	4.5	10.3	3.3	2.6	-

¹No observations at this age; ²pigs suckled for 8 weeks and given access to creep-feed at 10 days of age; ³fasted for 15 h before slaughter; ⁴pigs weaned at 4 weeks and given access to creep-feed at 14 days of age and fed before slaughter; ⁵the sum of the enzyme in the pancreas and in the contents of the small intestine; ⁶pigs weaned at 4 weeks and not given access to creep-feed

The large size of the relative increase in pancreatic amylase early in life (Table 9) is due to the extremely small amount of amylase which is present in the pancreas

at birth. Increases in total amounts of amylase over the 6-8 weeks period of the order of 10^3 - 10^4 were recorded in all three studies. It is evident that there is a significant age component in the development of pancreatic amylase since there were significant increases during the first four weeks of life even in pigs which did not have access to creep feed. (Table 9; Lindemann *et al.*, 1986). However, in studies with weaned pigs, 4-7 weeks of age, Aumaitre (1972) found that the amylase concentration in pancreatic juice increased 2-2.5 fold 5 days following a significant increase in dietary starch. Similar results were reported by Corring and Chayvialle (1987) in older pigs; a 3 fold increase in dietary starch was followed by a 2.3 fold increase in amylase secretion.

Pancreatic lipase also undergoes large relative increases during the first 4 weeks of life (Table 9). It is likely that this is not just an effect of age since sows milk contains a high concentration of lipid (Table 1), and both the amount and the lipid content of milk increases during the first 3-4 weeks of lactation (Elsley, 1970). Also in the study by Lindemann *et al.* (1986), in which pigs were weaned at 4 weeks of age on to a predominantly carbohydrate-protein diet, lipase underwent a relative decrease at 5 and 6 weeks of age. This is in contrast to the findings in sucking pigs (Corring *et al.*, 1978) in which pancreatic lipase continued to increase up to 6 weeks of age and was still relatively high at 8 weeks. The influence of the dietary lipid concentration on lipase secretion has been demonstrated by Corring and Chayvialle (1987) in older pigs in which a 7 fold increase in dietary lipid intake resulted in a 1.8 fold increase in pancreatic lipase secretion.

The small intestine

Anatomy and histology

The small intestine of fully grown pigs is 16-21 m long, of which 4-4.5% is duodenum, 88-91% jejunum and 4-5% ileum (Nickel *et al.*, 1973). At birth, the length of the small intestine is 2-4 m long (Table 5) and the proportion identified as duodenum is similar to that in the adult (Vodovar *et al.*, 1964; Widdowson *et al.*, 1976) but the differentiation of the other two regions is not clear-cut.

The structure of the intestinal mucosa follows a similar pattern from the proximal duodenum to the distal ileum. The surface is composed of numerous, finger-shaped villi around the base of which are short, tubular glands or crypts descending to the *muscularis mucosa*. The whole epithelial structure is supported by the *lamina propria* which is comprised of connective tissue containing variable numbers of white blood cells, together with lymph and blood vessels, smooth muscle, and nerve fibres (Rowland, 1981). Stem cells located towards the base of the crypts give rise to Paneth cells, enteroendocrine, goblet (mucus) and columnar epithelial cells (enterocytes) by a process of primary differentiation (Smith, 1985). It is the goblet cells and the enterocytes which are the principal cells lining the villi. As the enterocytes migrate from the crypts towards the tips of the villi, they undergo both structural and functional maturation. The capacity of their brush border membrane to transport nutrients only begins when structural differentiation has finished. The functions of the enterocytes are twofold, digestive and absorptive (Smith, 1985).

Digestive capacity

Whereas the digestive action of the pancreatic enzymes takes place in the lumen of the intestine the actions of the various digestive enzymes found in enterocytes takes place at the cell surface or brush border (microvilli). The two major groups of brush border enzymes are the carbohydrases and the peptidases (Alpers, 1987).

Six different carbohydrases have been identified in the brush border of enterocytes in the small intestine of the pig. They are lactase, trehalase, and the four maltases isomaltase, sucrase and maltases II and III (Kidder and Manners, 1980). In a recent study on the development of four of these enzymes in the mid-jejunum of sucking pigs

up to 24 days after birth, James *et al.* (1987) found that sucrase and maltase activities (per unit mucosal protein) both remained low during the first 4 days of postnatal life. The activities of these enzymes then increased in pigs up to 10 days of age after which sucrase activity continued to increase to reach a plateau activity about twice that of maltase. Sucrase and maltase activities in 24-day-old pigs were not significantly different from values reported by Miller *et al.* (1986) for 4- and 6-week old sucking pigs.

Lactase activity was high at birth and remained so during the first 10 days of postnatal life (Widdowson *et al.*, 1976; James *et al.*, 1987). There was then, a fall in activity during the next 6 days to a level which remained constant up to 24 days of age. This reduced activity was again not significantly different from that reported previously for 4- and 6-week old sucking pigs by Miller *et al.* (1986).

In pigs which were weaned at 3 or 5 weeks of age, however, the activities of sucrase, isomaltase and lactase 5 days after weaning were significantly lower than those in sucking pigs of the same age (Miller *et al.*, 1986). Maltase II and III, on the other hand, increased significantly in the same period in pigs weaned at 5 weeks but not in those weaned at 3 weeks. A reduction in lactase and sucrase activity during the 3-8 day period following weaning at 3 weeks of age has also been reported by Hampson and Kidder (1986). By 11 days after weaning there was a partial recovery of sucrase activity to the pre-weaning levels whereas lactase activity continued to decline in both weaned and unweaned pigs during this time. In both the studies of Hampson and Kidder (1986) and Miller *et al.* (1986), the reductions in brush border enzymes in the period following weaning, expressed/unit of mucosal protein (specific activity), were large. However, they are an under-estimate of the total reduction of enzyme activity in the small intestine because, during the post-weaning period, a concomitant significant reduction in villus height of the order of 30-63% was observed in the 3-8 day period following weaning at 3 weeks of age together with a 76-180% increase in crypt depth (Hampson, 1986; Miller *et al.*, 1986). Cera *et al.* (1988) reported both a reduction in villus height (65%) in the mid-jejunum and a cessation of growth of the small intestine during the 3 days following weaning at 3 weeks of age. Effects on villus height, a 27% reduction, and small-intestinal weight observed in pigs weaned at 5 weeks of age were similar but less dramatic. In the pigs weaned at 3 weeks of age, small-intestinal weight and villus height subsequently increased from 3 days until 14 and 21 days after weaning, respectively, when they were similar to those in 5-week old sucking pigs. In those pigs weaned at 5 weeks of age the equivalent recovery in villus height took only 7 days. In addition to a reduction in villus height, Cera *et al.* (1988) showed that the length of the microvilli (brush border) was also reduced in the 3-7 day period following weaning at 3 weeks of age.

From 2 weeks after weaning onwards the specific activities of the carbohydrases, with the exception of lactase, continue to increase up to 200 days of age. After this time maltase II and III and trehalase activities plateau but sucrase and isomaltase activities continue to increase (Kidder and Manners, 1980). After reaching a peak at 1-2 weeks of age (James *et al.*, 1987), lactase activity decreases with age, however some activity has been detected in the intestine of a 7.5 year old pig (Kidder and Manners, 1980).

In contrast to the relatively small number of carbohydrases, the number of peptidases found in the enterocyte is large (Alpers, 1987). This is not surprising since the variety of peptide bonds produced by the action of pancreatic proteases is much larger than for glycosidic bonds. Two endoproteases have been identified in the brush border, enterokinase and endopeptidase 24.11, the former being responsible for the activation of trypsinogen (Alpers, 1987). Most of the other peptidases identified in the enterocyte are either aminopeptidases or dipeptidases and the location of their activity is either in the brush border or the cytoplasm (Kidder and Manners, 1987; Alpers, 1987). The final stages of protein digestion are carried out by these enterocyte

peptidases. Peptides larger than three amino acids are hydrolyzed extracellularly by brush border enzymes whereas tripeptides and dipeptides are hydrolyzed either at the brush border or intracellularly (Alpers, 1987). There is a considerable literature describing the occurrence and location of the peptidases found in enterocytes from the adult pig (Kidder and Manners, 1978; Alpers, 1987). However, apart from the data of Lindberg and Karlsson (1970) regarding dipeptidase activities in foetal, newborn, 1 week old and 6 week old pigs, little further information is available about the development of these enzymes in the young pig or the effects of weaning on their specific activities in the small intestine.

Absorptive capacity

The precise membrane transport mechanisms for the end products of digestion, i.e. hexoses, amino acids, peptides (tri- and di-), fatty acids and monoglycerides have recently been described in reviews by Alpers (1987), Hopfer (1987) and Shiau (1987). However, the development of these mechanisms in the small intestine of the pig is much less well documented, as can be seen by the paucity of references to the pig in the recent review on the ontogenic development of intestinal nutrient transporters by Buddington and Diamond (1989). Principal among the workers who has contributed to our knowledge in this area in the pig is M.W. Smith whose publications have already been referred to frequently in this paper.

In the newborn pig, the primary function of the enterocytes lining all but the most proximal regions of the small intestine is to absorb colostral antibodies by a process of endocytosis. Whereas the bulk of this process is complete within the first 1-2 days of life, macromolecular uptake can still occur in some mucosal cells for up to 2-3 weeks after birth (Smith and Jarvis, 1978). The enterocytes responsible for macromolecular uptake are present at birth (foetal-type) and are eventually totally replaced during the following 19 days with cells (adult-type) which have the capacity to digest and absorb nutrients (Smith and Jarvis, 1978; Smith and Peacock, 1980). The longer turnover time of enterocytes in newborn pigs compared with that in mature pigs (2-4 days) is due mainly to two factors. Firstly, during the first 10 days of post-natal life, the small intestine grows very rapidly both in overall length (mean growth rate 1.25 cm/h) and in diameter (mean increase 0.1 mm/day), and also in the length of the villi; by day 10 villi were 29-75% longer than they were at birth (Smith and Jarvis, 1978). Secondly, there appears to be a differential movement of cells up the villus with some adult-type cells formed after birth moving up the villus at a faster rate than those of the foetal-type (Smith and Jarvis, 1978).

The most extensive studies on the development of the absorptive capacity of the small intestine have been done with the sodium-dependent alanine transport system (James *et al.*, 1987b). Enterocytes of unsuckled newborn pigs were found to be capable of transporting lysine and alanine, the latter in the presence or absence of sodium. Following the intake of colostrum there was a reduction in absorptive capacity which, for lysine uptake and sodium-independent alanine uptake, remained more or less constant over the next 4 weeks. In contrast, sodium-dependent alanine uptake increased steadily throughout the first 4 weeks of post-natal life in suckled pigs. In 6-week-old sucking pigs though, Miller *et al.* (1986) found that sodium-dependent alanine uptake was lower than in 4-week-old sucking pigs. Finally, absorptive capacity of the small intestine appears to be adversely affected by weaning, the degree of impairment being inversely related to the age of weaning. For instance, weaning at 2 and 3 weeks of age was found to reduce sodium-dependent alanine absorption 5 days after weaning (Smith, 1984; Miller *et al.*, 1986), whereas weaning at 5 weeks had little effect when compared with absorption in unweaned littermates of the same age (Miller *et al.*, 1986). Weaning at 3 weeks of age was also found to reduce the capacity of the gut to absorb xylose (Hampson and Kidder, 1986).

Conclusions

From the foregoing it is evident that the consequences of weaning piglets from a diet of predominantly sow's milk to one of solid food are many and complex. At weaning, the piglet is removed from:

- (1) a controlled, semi-continuous source of highly digestible and available nutrients;
- (2) a supply of immunological and non-immunological protective agents;
- (3) a supply of stimulatory and regulatory factors which may be important for the completion of the development of the digestive tract and its regulatory systems;

and is provided with a post-weaning diet which at best only replaces (1) of the above. Usually though, the post-weaning diet is of a quality that is significantly inferior to that of sow's milk and it is provided in an unregulated way.

From the information we have about the gastrointestinal system of the piglet it is apparent that its development is far from complete even by four weeks of age, that it has to undergo a period of adaptation to increase its physical capacity and size, its capacity to secrete digestive enzymes, HCl, bicarbonate and other chemical secretions, and its absorptive capacity before it can satisfactorily cope with the post-weaning diet, and that the younger a piglet is at weaning, the longer the period of adaptation will be. Creep-feeding has been found to enhance the development of the stomach, but evidence about its effects on the development of the pancreas and the small intestine is inconclusive. Of all the components of the gastrointestinal system, it is the small intestine which is the most vulnerable to disruption in the immediate post-weaning period.

Solutions to the problem of how to improve post-weaning performance of the pig will only be found by a collaborative and concerted research effort to determine the relationships and interactions between the nature of the pre- and post-weaning diets, the age and weight of the piglets at weaning and the development and maturation of the gastrointestinal system. To do this, the team of researchers will have to include physiologists, nutritionists and those responsible for the development of management systems.

ALTERNATIVE FEEDING STRATEGIES FOR WEANER PIGS

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Introduction

Although there have been remarkable achievements in improving the efficiency of pig production by the application of advances in scientific knowledge, the performance and health of piglets in the period around weaning remains a particularly problematic area. Fundamental research has allowed mathematical description of the bio-energetics of growth in terms of maintenance, protein deposition, fat deposition, the influence of environmental temperature and so on. In addition, concepts have evolved to describe maximum daily lean tissue growth rate, the linear acceleration phase in response to feed intake and the deposition of excess energy as body fat. This information has made it possible to predict with reasonable accuracy the response of growing pigs to changes in nutrient inputs at the farm level. The exception to this is during the few weeks around weaning.

The difficulties of consistent prediction and manipulation of weaner performance arise from the major physiological changes which take place in the young pig, particularly in association with weaning itself. The problem has been accentuated by our desire to wean pigs increasingly early. There is an inverse relationship between the age of the pig and its sensitivity to the environment. Thus the degree to which environmental factors confound the response to nutritional treatments is increased by earlier weaning. These factors have constituted a considerable challenge to the commercial nutritionist and pig producer. This paper considers the evolution of commercial early weaning diets in the UK, variously referred to as pig creeps, starter diets or pre-starter diets, and examines the alternative approaches to formulation which have been used elsewhere.

Age at weaning

The primary factor which influences decisions concerning the feeding of weaners is their age at weaning. We talk loosely of "early weaning" but clearly this is a relative term; quite small differences in the age and probably more importantly, the weight and physiological maturity of piglets at weaning can make quite a large difference in the optimum diet specifications. From the point of view of sow productivity it is generally believed that around three weeks is the optimum lactation length (Self and Grummer, 1958; Smidt *et al.*, 1965; Moody and Speer, 1971; Aumaitre, 1973). However, the optimum is likely to vary from one herd to another because genetic and managerial factors influence the degree of reduction in litter size and the increase in weaning to service interval which are associated with decreased lactation length.

It is interesting to observe the trends in weaning age in recent years. Table 10 shows data presented by the UK Meat and Livestock Commission (MLC, 1989). In 1988, 89% of MLC recorded herds were weaning litters at 19-32 days, 10% more than in 1980. In the early 1980s there was a trend towards very early weaning, which has since declined and a move towards four week weaning is evident now, with a 3% drop from 1986 of herds weaning at 19-25 days. On the other hand, financial results in the same publication (MLC, 1989) clearly illustrate the increased returns from weaning at 16-20 days compared with 21-25 days and more. This suggests that some producers have been rather successful with earlier weaning and have stayed with it, whilst others

have reverted to weaning later, presumably because of unsatisfactory performance.

In Australia it appears that weaning age has been reduced from around 33-39 days in the 1970s and early 1980s to around 28 days at the present time. The opportunity for even earlier weaning is perhaps limited by a lack of suitable commercial starter diets.

Clearly an important factor in the success of earlier weaning is the ability of the housing, management and nutrition to meet the precise and critical needs of the piglets according to actual weaning age and weight. This paper deals in general with the feeding of pigs weaned at between 19 and 32 days, with some discussion of appropriate fine-tuning for specific ages within this range.

Table 10. Trends in weaning age in UK 1980-1988 (MLC, 1989)

Age at weaning (days)	1980	1982	1984	1986	1988
	(Percentage of herds)				
Under 19	5	11	7	3	2
19-25	41	45	57	67	64
26-32	13	18	22	21	25
33-39	32	20	10	7	7
Over 39	8	6	3	2	2
Number of herds	653	766	725	711	700

Objectives of post-weaning nutrition

The goal in developing a feeding strategy for piglets in the period immediately after weaning should be to minimise the incidence of diarrhoea and maximise growth rate. Evidence gathered in recent years suggests that growth rate of young pigs after weaning at 5-7 kg live weight is largely limited by feed intake rather than by inherent growth potential. The pig has an in-built drive to attain its maximum potential growth rate at the earliest opportunity. In experiments where high feed intakes have been achieved between 5 and 20 kg live weight, lean tissue growth rates approaching the maximum have been observed (Whittemore, 1984). The challenge to the commercial nutritionist and the pig producer is to achieve sufficiently high feed intakes to reach this potential. In the past and even today on some pig units, feed intakes in newly weaned pigs have been restricted intentionally such that very low growth rates were achieved. The objective was to avoid digestive upsets and diarrhoea, so often associated with feeding to appetite. High levels of medication have also been applied, extending further the strategy of treating the symptoms rather than the cause of the problem.

Improved understanding of the digestive physiology of piglets acquired during the last 15 years has allowed us to develop diets which can be offered *ad libitum* (except perhaps for the first day or two after weaning) and achieve high intakes and high growth rates without predisposing them to diarrhoea. This has only been achieved at considerable expense in terms of the feed ingredients required. Therefore the rationale for such a strategy, compared with the low input and low output system in which a poorer quality diet is given in restricted amounts, is open to question. The benefits of fast growth in the early stages after weaning are several. Firstly, this will accelerate physiological maturity, thus reducing the time during which the piglet is at its most susceptible to disease challenges. In addition, overall production costs may be lowered due to reduced maintenance feed requirement, reduced time in expensive weaner accommodation and by reducing the need for supplemental heating. The latter point is illustrated well by the work of Close and Stainer (1984) who showed that the lower critical temperature of piglets reduced from 27-24 as feed intake increased from the

maintenance level to twice maintenance.

The growth check which often occurs at weaning is associated with a substantial loss of body fat. The piglet which just maintains its body weight may be losing as much as 50 g of body fat/day to satisfy its energy requirement, this being matched by a gain of a similar weight of water (Whittemore, 1984). This situation may be regarded as representing an undesirable metabolic crisis. Piglets which remain on the sow between three and four weeks of age typically gain 300 g/day. Furthermore, the growth rate above which fat degradation is replaced by fat deposition is about 200 g/day. Perhaps then it is reasonable to suggest that the target growth for piglets in the first week after weaning should be between 200 and 300 g/day. Assuming suitable environmental conditions and a feed conversion ratio close to 1:1, this means that they would need to consume 200-300 g of feed/day.

Diet formulation for optimal growth

The need to achieve high feed intakes and efficient feed conversion without subjecting the piglet to the risk of diarrhoea or oedema implies the need for a diet composed of highly digestible ingredients. In essence the objective is to adapt the digestive system from a diet of sows' milk in which energy is provided predominantly as fat, together with lactose and milk proteins, to one in which the energy is provided predominantly as starch, with mixed vegetable and animal proteins. When pigs are weaned at between 3 and 4 weeks the prior intake of solid feed during the suckling period is inevitably small, as discussed later. Thus the adaptation must largely take place in the period immediately after weaning. It would therefore seem appropriate to provide a diet which is intermediate in composition between sow's milk and the type of formulation we wish subsequently to use for the major part of the growing period.

The benefits of taking account of these considerations in the formulation of a starter diet were demonstrated by English *et al.* (1978) on a large commercial unit in Scotland. The formulation of their highly digestible diet is shown in Table 11. It can be seen that this contained a high proportion of milk products, vegetable oil and cooked oat flakes. The ability of the young piglet to digest starch is limited, principally because it takes some time for amylase secretion to be induced in response to starch ingestion (Aumaitre, 1972). It is thought that flaking of cereals or other heat treatments aid enzymic hydrolysis by rupturing the starch grains. In the trials of English *et al.* (1978) the test diet was compared with two commercial diets which had similar specifications in terms of declared DE and protein content. As the commercial diets were considerably cheaper than the test diet it was assumed that they contained less of the higher value ingredients such as milk products and partially cooked cereals. A high incidence of diarrhoea was observed with the commercial diets, particularly when these were offered *ad libitum*. With the test diet there was little diarrhoea and the incidence was not increased by feeding *ad libitum*.

The principle of high density diets based on high digestibility ingredients has been widely adopted by the pig industry in the UK. Clearly the high cost of such diets is an important consideration but this should be weighed against the benefits of trouble-free weaning. Such expensive diets need not be fed for very long. Once pigs have adapted to dry feed, perhaps after about 10 days, a diet of lower ingredient specification can be introduced.

The detailed compositions of commercial pig starter diets are largely secret and have been based on unpublished in-house research. Most companies produce a range of products designed principally for differing weaning ages and production environments. On the one hand, these differ in nutrient specification, particularly with respect to DE and total lysine concentration which may range from 14-18 MJ/kg and from 13-16 g/kg, respectively. On the other hand they differ in ingredient specification, particularly with

regard to the proportions of milk products such as skimmed milk powders, whey powders and lactose and in the proportion of processed rather than "raw" cereals.

Table 11. Formulation of a highly digestible diet for pigs weaned at an average age of 19 days (English *et al.*, 1978)

	g/kg
Cooked oat flakes	340
Maize oil	100
Skim milk powder	240
Milk substitute (20% tallow, 80% skim milk powder)	250
Glucose	50
Minerals and vitamins	20
DE (MJ/kg)	19.6
Crude Protein	212

Results of commercial trials reported by Phelps (1987) have emphasised the significance of weaning weight in relation to the type of starter diet needed. Pigs which weigh less than 6 kg at weaning need a diet very rich in oils and milk products whilst those weighing more than 6 kg can perform well on less costly diets containing a higher proportion of cereals and modest amounts of milk products. This is illustrated in Table 12. Pigs which were heavier at weaning performed equally well on the medium and high cost diets such that the former was more cost-effective. The lighter weaners performed better on the high cost diet such that there was little difference in the cost of live weight gain. However, those on the lower-cost diet were 1 kg lighter at six weeks.

Feed presentation

In view of the importance of achieving high intakes of feed early in the post-weaning period attention should be paid to its physical characteristics. In our own trials (carried out by Colborn-Dawes Nutrition, UK) a standard commercial formulation was presented as a meal, crumbs, or as pellets of 2.4 or 3.2 mm diameter. The results given in Table 13 show that the best performance was achieved with the 2.4 mm pellet. Apparent feed intake was highest with the meal but wasted feed was also included in this figure. Clearly pelleting is important, if only to minimise wastage of such expensive feeds. It should be pointed out that with high levels of milk products in the formulation careful attention has to be paid to the pelleting process to avoid the production of pellets which are too hard or overheated. Flavours, flavour enhancers and sweeteners are widely used in commercial pig starter diets. These may help to improve intakes in some circumstances but the benefits are small. It is more important to ensure that starter diets are fed fresh, by ensuring regular manufacture and delivery with minimum storage.

Compensatory growth

The performance of pigs during the early post-weaning stage should not be considered in isolation but as a component of overall performance from weaning to slaughter. It is often suggested that a period of slow growth after weaning can be tolerated provided that the pigs catch up later. It is even suggested that such a regime may be beneficial in terms of overall feed cost.

Table 12. Performance of weaners according to weaning weight and diet specification (Phelps, 1987)

	Diet Category			
	High cost	Medium cost	High cost	Medium cost
Weaning weight (kg)	6.26	6.24	5.10	5.20
Six-week weight (kg)	13.16	13.08	11.93	10.93
Daily gain (g)	328	325	325	273
Feed:gain ratio	1.20	1.25	1.04	1.37
Cost/kg gain (cents)	98	77	85	84

Table 13. Effect of form of presentation of feed on performance of piglets during the 3 weeks after weaning at 21 days of age

Meal	Crumbs	2.4 mm Pellet	3.2 mm Pellet	SE
Daily weight gain (g)	280	270	300	10
Daily feed intake (g)	390	310	330	19
Feed:gain ratio	1.37	1.16	1.11	0.025

When discussing compensatory growth it is important to distinguish between growth restriction resulting from reduced feed intake and that resulting from qualitative aspects of the diet. Lightfoot (1984) observed that pigs which had poorer post-weaning performance due to a lower quality and lower cost starter diet also had poorer finishing performance, as shown in Table 14. With poorer quality starter diets although reduced feed intake may be one reason for slower growth, the health status of the piglet and the maturation of the digestive system may also be compromised. If this is the case then the likelihood of subsequent compensatory growth will be reduced. Evidence presented by Elsley (1963), Nielsen (1964) and more recently by Campbell and Dunkin (1983) indicated that compensatory growth can be achieved following a period of restricted feeding, at least under specific experimental conditions. What is open to question is whether this can be achieved under farm conditions and indeed whether it is a desirable objective.

Table 14. The influence of starter diet on subsequent performance (Lightfoot, 1984)

	Starter diet 1 (\$826/tonne; 25% milk powder)	Starter diet 2 (\$564/tonne; 10% milk powder)
Starter performance		
Initial weight (3 weeks)	5.93	5.94
Final weight (6 weeks)	10.31	9.56
Daily gain (g)	219	181
Feed:gain ratio	1.23	1.47
Feed cost/kg gain (cents)	101	83
Finisher performance (40-80 kg, both groups on same regime)		
Daily gain	689	670
Feed:gain ratio	2.59	2.71
Feed cost/kg gain (cents)	85	89

Alternative feedstuffs and feed additives for pig starter diets

For pigs weaned between three and four weeks of age the use of high density, high digestibility diets has been the most consistently successful approach. Skim milk powder and other dried milk products have been the most valuable feedstuffs for starter diets and it is perhaps the availability and price of these which have most influenced starter diet formulations around the world.

Alternatives to milk powders

Patterson (1985) has attempted to determine the relative importance of the protein and lactose components of skim milk powder in starter diets. Rates of gain fell significantly when soya or fishmeals replaced the protein component of skim milk powder but replacement of lactose with glucose or starch had little effect. In contrast Giesting (1986) found that replacement of lactose by either cornstarch or a variety of hydrolysed cornstarch products depressed performance. Clearly the response must depend upon the specific protein and energy sources substituted and the other components of the diet but it appears that the benefits of skimmed milk powder are both as a replacement for vegetable proteins and as a source of lactose. Vegetable proteins present a range of problems to the immature digestive system. In addition to "anti-nutrient factors" such as trypsin inhibitor and the possibility of antigenic factors (Newby *et al.*, 1984), plant proteins are enclosed in or intimately associated with cell wall polysaccharides which can markedly limit their digestion.

Fish meals and meat meals are rather variable products but the best samples may have digestibility values which approach that of skimmed milk. Thus Green (1989) reported ileal lysine digestibility values for single batches of meat meal, fishmeal and skimmed milk powder of 0.84, 0.94 and 0.94, respectively, in piglets (13 kg live weight). Careful sourcing and quality control of meat meals and fish meals is essential for starter diet use.

Amino acid supplementation

Supplementation with free amino acids can be particularly valuable in the formulation of pig starter diets. These can be used to minimise the protein content of the diet whilst maintaining an adequate supply of amino acids. This has the dual role of reducing the amount of protein which must be digested by the immature piglet and reducing the burden of undesirable compounds associated with protein supplements. To achieve a target lysine concentration of 15 g/kg with conventional raw materials would necessitate a crude protein concentration of 230-240 g/kg. This can be reduced to 195-200 g/kg by supplementation with lysine, methionine and perhaps threonine and tryptophan to maintain the optimum ratios of lysine to the other essential amino acids.

Acidification

The development of a sufficiently low pH in the piglet's stomach is important, both to ensure efficient digestion and to control the proliferation of potentially harmful micro-organisms. Gastric acid secretory capacity is not fully developed in the 3-4 week old animal and the rate of development is dependent upon the intake of solid feed (Cranwell, 1985c). During the last 20 years a number of reports have indicated that there may be beneficial effects of adding organic acids or their salts to piglet diets. Cole *et al.* (1968) reported that the addition of lactic acid to drinking water reduced *E. coli* counts in the duodenum and jejunum and improved growth rate and feed:gain ratio. This response perhaps gives some credence to anecdotal remedies for piglet diarrhoea involving the administration of acidic products such as cider vinegar in the UK (Stockill, 1989), lime juice in Costa Rica (Easter, 1988) or cordial solutions in Australia (Paton *et al.*, 1988).

The supplementation of weaner diets with organic acids has been comprehensively reviewed by Easter (1988). Many trials have shown improvements in performance traits including growth rate, feed intake and feed:gain ratio from the addition of 1-3% of fumaric, formic, citric, malic or propionic acids or their calcium salts. On the other hand a number of trials have shown no response. As suggested by Easter (1988) it seems likely that the response depends on the formulation of the diet. The greatest benefit seems to be when diets are formulated with cereals, supplemented with plant proteins while diets containing lactose show less response, as illustrated in Table 15. Presumably lactose, provided as such or in the form of milk products is converted to lactic acid in the stomach creating the desired reduction in pH. This raises the question concerning the acid binding capacity of feeds. Research has shown that most minerals and raw materials high in protein have a considerably higher buffering capacity than cereals (Bolduan *et al.*, 1988).

As most of the acidulants mentioned cost in excess of \$2000/tonne, a 1-2% addition will cost in the order of \$20-40/tonne of starter feed. Unfortunately, trials with the much less expensive inorganic acids have given negative results, perhaps due to electrolyte imbalances (Easter, 1988).

Probiotics

A wide range of commercial products described as probiotics have appeared on the market in recent years. Essentially they are live bacterial feed supplements containing *lactobacilli*, *bifidobacteria* and *streptococci*, all genera which occur naturally in the gastrointestinal tract. These organisms are known to provide the host with some immunity to infection from pathogenic bacteria. Although the mechanism of protection is not fully understood, competitive exclusion of pathogenic organisms, production of antibiotic substances and production of lactic acid have all been suggested. Unfortunately there is no well documented evidence indicating consistent beneficial effects of the commercial preparations. One possible problem concerns the ability of such products to survive the pelleting process. Another is the potential inhibition of the probiotic organisms by antibiotic growth promoters. For the present, at least, the conventional antibiotic products are to be preferred in view of their well documented consistent beneficial effects on the health and performance of piglets.

Table 15. The effect of fumaric acid addition on performance of piglets (initial live weight =82 kg) given diets formulated with soyabean meal or dried skimmed milk (Easter, 1988)

Protein source Fumaric acid (%)	Soyabean meal			Skimmed milk			SE
	0	2	3	0	2	3	
Performance, weeks 0-2							
Live weight gain(g/day)	133	152	171	195	223	195	23.3
Feed intake (g/day)	306	296	304	312	332	310	22.1
Feed:gain ratio	2.33	1.96	1.79	1.67	1.49	1.59	0.154
Performance, weeks 0-4							
Live weight gain(g/day)	289	320	311	327	359	350	22.3
Feed intake (g/day)	540	549	533	532	565	536	33.1
Feed:gain ratio	1.85	1.72	1.72	1.64	1.56	1.54	0.382

Fibre

It is often claimed that there are benefits to the health of piglets from adding fibrous feeds to starter diets. Thus, Bolduan (1988) suggested that the crude fibre content of the diet should be about 5% in order to activate the large intestine, this being achieved by the addition of 20-30% wheat bran, 10% lucerne meal or 5% straw. The benefits of activating the large intestine are said to be the enhanced removal of

harmful by-products of N metabolism via faecal excretion. On the other hand, as Bolduan (1988) admits, increasing the crude fibre content of the diet reduces daily gain. It seems likely that, just as restricted feeding may ameliorate the affects of a poorly digested diet, increasing its fibre content may achieve the same effect through voluntary reduction in intake. Both strategies may reduce the piglets' susceptibility to diarrhoea by reducing the quantity of suitable substrates for the growth of pathogens (undigested proteins and starch) in the lower regions of the digestive tract.

Creep feeding

The practice of providing piglets with solid feed as a supplement to sows' milk during the suckling period was universally accepted when weaning took place at six weeks of age or more. Milk yield declines after three weeks so supplemental feeding was essential to ensure satisfactory growth and weaning weights. With earlier weaning the need for creep feed is increasingly questioned and is perhaps more difficult to justify. It is evident that the growth potential of piglets during the first three weeks of life considerably exceeds what is normally achieved in practice. This has been demonstrated in comparisons of sow reared and artificially reared piglets (e.g. Campbell and Dunkin, 1983). However, it has proved difficult in practice to achieve a significant increase in weaning weight at three weeks by giving supplemental solid feed, due to the limitation of feed intake at this stage.

Another argument which has been used against the practice of creep feeding is based on the work of Professor John Bourne and colleagues at Bristol University. They have pointed out the possibility of hypersensitivity reactions leading to post-weaning diarrhoea in response to dietary antigens. The suggestion is that the intake of small quantities of certain proteins, particularly soya, before weaning sensitizes the immune system so that there is an adverse reaction to larger intakes of the same proteins after weaning. The result is intestinal damage which may itself cause only a mild diarrhoea but may leave the gut more susceptible to *E. coli* proliferation. Limited evidence for the practical consequences of this phenomenon has been presented by Newby *et al* (1984), as shown in Table 16. Possible solutions to this problem may be to wean abruptly without giving creep feed before weaning or to eliminate antigenic factors from the creep feed. However, as stated by Newby *et al.* (1984), the best solution would be to increase creep intake so as to ensure that the immune system has become tolerant to the antigens before weaning. This may be difficult to achieve with pigs weaned as early as three weeks of age.

Table 16. The incidence of diarrhoea in pigs fed varying amounts of weaner diet before weaning (Newby *et al.*, 1984)

	Creep given for 2 weeks before weaning	Creep given for 3 days before weaning	No creep before weaning
Diarrhoea	0	7	2
No diarrhoea	7	0	4

Creep feeding may be beneficial as a means of stimulating the earlier development of the mature digestive enzyme system. The ability to produce enzymes for the digestion of complex carbohydrates such as starch, non-milk sugars and proteins other than those in milk develops only slowly. There is evidence that the secretion of these enzymes which include sucrase, maltase, amylase and trypsin can be induced earlier by encouraging earlier consumption of the respective dietary substrates (Aumaitre, 1972). This suggests that creep feeding should precondition the digestive system to solid feed, which may reduce the growth check at weaning and improve post-weaning performance.

The work of Okai *et al.* (1976) illustrated in Table 17 is often quoted as indicating that there are no benefits from creep feeding in terms of post-weaning performance.

Table 17. Performance of pigs weaned at 3 weeks (and given creep from 10 days) or 5 weeks (and given creep from 14 days) and given (A) no creep, (B) creep based on raw cereal and soya bean, (C) creep with some animal protein and all cooked cereal (Okai *et al.*, 1976)

	A	B	C	D
Weaned at 3 weeks				
Daily gain 3-7 weeks (g)	320	280	310	300
Weaned at 5 weeks				
Daily gain 5-8 weeks (g)	300	290	320	320

However, these results are perhaps not surprising in view of the fact that total creep feed intake before weaning at three weeks was less than 75 g/pig, even on the most sophisticated diet. In contrast, the work of English *et al.* (1980) shows that when creep feed intakes in excess of 600 g/pig were achieved before weaning at four weeks, there were substantial benefits in post-weaning performance. This is shown in Table 18. In order to obtain such high intakes before weaning, careful attention must be paid to all aspects of creep feeding practice. This includes the provision of a highly digestible and palatable diet and ensuring that it is fed fresh, little and often, from about five days of age.

Table 18. Effects of providing a highly digestible diet as a creep feed from 7 days to weaning at 28 days (English *et al.*, 1980)

	Advantage over control %
Suckling period (to 28 days)	
Live weight gain 7-28 days	+ 7
Live weight gain 21-28 days	+ 17
29-47 days	
Feed intake	+ 6
Live weight gain	+ 15
48-69 days	
Feed intake	+ 5
Live weight gain	+ 5

Conclusion

Practical nutrition of the early weaned pig is more of an art than a precise science. The main reason for this is the difficulty of applying normal methods of scientific study to such a transient stage in the development of the animal. The response of the pig, normally measured in terms of growth rate and efficiency of feed use, is considerably modified by the variable effects of environment and disease. Thus a dietary regime which is entirely satisfactory in a one-off feeding trial at a research station may well not stand up to the rigours of commercial production. Similarly, commercial diets of apparently similar nutrient specification may give quite different results (performance and/or animal health): Responses vary from one pig unit to another and within one pig unit at different times.

Experience has shown that highly digestible, high density diets containing a significant proportion of milk products consistently support high rates of growth with

minimal digestive disturbance. Provided such diets are introduced early during the suckling period and a substantial intake is achieved before weaning at 3-4 weeks such diets can be fed virtually *ad libitum* from weaning onwards (some restriction may be needed in the first day or two after weaning). The continued use of such diets over many years by intensive pig producers in Europe is an indication of their cost-effectiveness.

In circumstances where such diets cannot be produced, for one reason or another, a number of options may be considered. The first is to delay weaning to ensure increased development of digestive secretory mechanisms, aided by greater intake of creep feed. Alternatively it must be accepted that there will be a substantial growth check during the week or so after weaning and that measures must be taken to minimise the incidence of diarrhoea. These include restriction of feed intake by rationing or by increasing dietary fibre concentration, addition of organic acids and medication.

THE NUTRITIONAL MANAGEMENT OF WEANER PIGS

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Introduction

The growth performance of pigs from weaning through to 10-11 weeks of age is critical in determining subsequent weight for age relationships and as such ultimate carcass weight. However, whilst the young pig has a high potential for growth there are numerous factors influencing the extent to which this potential is expressed particularly in the period immediately after weaning.

Apart from having higher dietary nutrient requirements, newly weaned pigs are less able to digest vegetable proteins (Owsley *et al.*, 1986b) compared to grower-finisher pigs and this constraint needs to be accounted for in the formulation of diets for the immediate post-weaning period.

Although the dietary nutrient requirements of weaner pigs are well established, commercial experience suggests that current protein (and amino acid) recommendations do not necessarily reflect the wide range of weaning weights experienced in practice and may not be appropriate for the younger/lighter piglets at weaning.

Questions have also been raised concerning the interrelationship between diet and the incidence and severity of post-weaning diarrhoea. This in turn has led to a resurgence in interest in the formulation and use of weaner diets with prophylactic functions. Although this idea is not new, there is some recent evidence suggesting further research is warranted.

The aim of this paper is to outline the factors known to influence weaner growth performance and how they interact to influence the ingredients and diet(s) needed to promote performance levels required to achieve a target weight of 28-30 kg at 10 weeks of age.

Factors affecting the performance of weaner pigs

The factors known to influence the growth performance of weaner pigs are listed in Table 19. They are not given in order of priority and because they interact to affect performance and piglet viability, they cannot be considered separately.

Table 19. Factors affecting the performance of weaner pigs

1.	Age/weight at weaning
2.	Nutrition
	(i) Nutrients
	(ii) Ingredients
	(iii) Feeding practices
	(iv) Diet form
3.	Disease status
4.	Stockmanship
5.	Environment

Environment, and in particular the thermal environment is probably the single most important factor determining the growth performance and to some extent the survival of newly weaned pigs. Poor environmental conditions can be offset to some

extent by increasing weaning age and appropriate veterinary care. On the other hand, they are difficult to combat nutritionally and almost invariably prevent target performance levels from being achieved.

Weaning weight and nutrition

Because of the strong positive relationship between weaning weight and post-weaning growth performance, there are considerable advantages associated with increasing the piglets weight at weaning. This is demonstrated in Table 20 which gives the comparative performance to 78 days of age of piglets weighing approximately 6 or 8 kg live weight at weaning at 25-29 days of age.

Table 20. Effect of weaning weight at 25-29 days of age of the performance of piglets to 78 days of age

Weight at weaning (kg)	78 day weight (kg)	Daily gain (g)
6.14 (n=1000)	30.4	454
7.95 (n=1000)	35.6	529

Pigs weighing 7 kg or more at weaning are less susceptible to post-weaning diarrhoea than their lighter counterparts and are more readily able to achieve a target weight of 30 kg at 10 weeks of age on a relatively simple wheat based diet formulated to have a minimal DE concentration of 14.5 MJ/kg and 0.75-0.8 g available lysine/MJ DE.

This however is not surprising since with current creep and sow feeding practices, an increase in weaning weight is generally associated with an increase in age at weaning. These heavier/older piglets have generally had more experience with dry feed prior to weaning, which in turn enhances gastric development, and are immunologically more competent than their lighter/younger counterparts at weaning.

For these reasons increasing minimal weaning age to 28 days of age or alternatively minimal weaning weight to 6.5 kg will generally confer marked economic advantages on piggeries with the farrowing accommodation and reproductive efficiency to permit such strategies. Unfortunately in the majority of situations management practices and constraints on farrowing accommodation imposed by reproductive inefficiencies often mean that piglets are weaned at an average age as low as 21 days and range in age from 18-24 days (4.5-6.0 kg).

These animals, but in particular the lighter/younger piglets, generally have had no experience with dry feed, are unable to efficiently digest vegetable proteins (Owsley *et al.*, 1986b), require higher environmental temperatures and tend to be more susceptible to post-weaning diarrhoea than heavier/older piglets. For these reasons younger piglets at weaning require better management and a different nutritional strategy if they are to achieve target growth rates in the post-weaning period.

There is considerable scientific (ARC, 1981; NRC, 1988) and commercial evidence suggesting that pigs weighing 4-5 kg at weaning respond positively in terms of growth rate to dietary DE concentration up to 15 MJ/kg and available lysine levels as high as 0.9-1.0 g/MJ DE. The latter is not surprising since it has long been known that the pigs capacity for protein deposition/unit energy intake is very high during early life and falls rapidly with increasing live weight up to 40 kg (Carr *et al.*, 1977).

Ingredient selection is also more critical for the younger animals and the higher lysine and more digestible animal protein supplements (milk powder, fish meal, blood meal and meat meal) should be used in preference to vegetable protein supplements such as soybean, lupins or peas. Diets formulated to meet these nutrient specifications and ingredient constraints, are necessarily relatively expensive. Nevertheless, they only need to be offered to piglets during the first three weeks following weaning after which

they can be replaced with more conventional weaner diets of the type described previously for heavier pigs. Consequently, these first-stage weaner or creep diets represent only 4-5% of total feed usage in a piggery and have a minor effect on overall feed costs. On the other hand, because they are designed to match the special requirements of newly weaned pigs they can have a marked positive effect on ensuring subsequent performance targets are achieved.

Use of nutrient dense specialised weaner diets

Considerable debate continues to surround the role of nutrient dense, highly digestible diets for weaner pigs. These feeds often termed super starter or "English"-type diets are based on cooked cereals, high levels of milk proteins and other highly digestible animal protein supplements and vegetable or animal fats. They commonly contain 17 MJ DE/kg, 0.9-1.0 g available lysine/MJ DE and can cost \$900-1000/tonne.

Commercial evaluations of diets of this type seriously question their cost effectiveness for pigs weighing 5 kg or more at weaning since they do not support faster growth compared to more conventional first-stage weaner diets (15MJ DE/kg and 0.85-0.90 g av. lysine/MJ DE) and the difference in feed:gain between the two diets tends to be proportional to the difference in DE concentration between the two diet types. On the other hand, the cost/unit DE is disproportionately higher for the "English"-type diets compared with the more conventional first-stage weaner feeds.

Nevertheless, the "English"-type diets may have an important strategic role in weaner nutrition because they tend to reduce mortality and promote more normal growth in pigs exhibiting ill-thrift at weaning (runt or reject pigs). They also tend to support improved growth performance in the very small piglets at weaning (<4.1 kg) compared with more conventional diets. Furthermore, with slight modification to ingredient restrictions the cost of these more specialized diets can be considerably reduced without altering their effectiveness for the very small and less thrifty pigs at weaning.

Based on the information outlined above, the basis of a practical nutritional strategy for piglets weaned at 18-28 days of age is given in Table 21. Depending on individual circumstances, and in particular on weaning age and the piglets environment at weaning (affected by season), it may be necessary to extend the use of the first-stage weaner diet for 7-14 days in order to achieve overall target growth rates to 10 weeks of age of 480-500 g/day and 540-600 g/day for pigs weaned at 21 and 28 days of age respectively.

Table 21. Nutritional strategy for weaner pigs

Weight at weaning	Diet and time after weaning		
	Week 1	Weeks 2-3	Weeks 4-7
<5 kg and less viable piglets	Super ¹ Starter	First stage weaner ²	Second stage weaner ³
5-7 kg		First stage weaner	Second stage weaner

¹Diet based on cooked rolled oats and animal protein supplements including 5% whey powder (16-17 MJ DE/kg and 1.0 g available lysine/MJ DE); ²diet based on wheat and animal protein supplements (15 MJ DE/kg and 0.85-0.9 g available lysine/MJ DE); ³wheat based diet and vegetable and animal protein supplements (14.5 MJ DE/kg and 0.75-0.8 g available lysine/MJ DE)

Other considerations in diet formulation and feeding for weaner pigs

Prophylactic function of the diet

Because of the newly weaned piglets' susceptibility to diarrhoea and the adverse effects of the latter on both performance and piglet survival, there is increasing interest

in the extent that diets and feeding practices can be manipulated to reduce the incidence and severity of the problem.

Bolduan *et al.* (1988) published convincing information on gastro-intestinal development of the young pig immediately after weaning and on how the selection of ingredients with low acid binding capacity (Table 22) and the use of organic acids (lactic, formic or fumaric acid) can assist in the breakdown of proteins in the stomach and in doing so reduce the amount of substrate available for bacterial proliferation in the small intestine. Although the effectiveness of organic acids is equivocal and may be limited to diets containing high levels of vegetable protein supplements, both these prophylactic measures warrant further research, particularly under situations in which post-weaning diarrhoea is a problem.

Table 22. Acid-binding capacity of feedstuffs (consumption of mmol HCl required to reach pH 4 per 100 g original matter) (from Bolduan *et al.*, 1988)

Skim milk (acid)	3.07
Skim milk (fresh)	7.12
Wheat	8.99
Barley	9.97
Yeast	30.10
Soyabean meal (extracted and toasted)	50.68
Fish meal	60.38
Skim milk (dried)	66.37
Mineral mixture	1260.50
Starter for weaner piglets	30.00

Bolduan *et al.* (1988) also suggested that raising the crude fibre level of first-stage weaner diets to 5% assists in enhancing development of hind gut function, which in turn decreases the transit time of ingesta, and as such may again reduce the availability of substrate for bacterial growth (Table 23).

Table 23. Influence of straw meal on gut characteristics of weaner piglets (Bolduan *et al.*, 1988)

	Feed	
	Starter alone	Starter + 5% straw
Crude fibre of diet (%)	2.8	4.9
Transit time (hours)	130	107
Proportion of digesta in the hindgut (%)	15	33
Days with diarrhoea (%)	6.0	3.5
Metabolites of microbes (mmol^l)		
Stomach	LA	2.8
	VFA	4.0
Colon	NH ₃	3.2
	VFA	23.9
	NH ₃	6.2

¹In the whole part of the gut; LA= lactic acid; VFA= volatile fatty acids.

Although the crude fibre level of diets currently used in Australia for pigs during the first 21 days after weaning is not generally restricted, the high nutrient specifications and ingredient restrictions employed in these formulations often result in low crude fibre levels (<3%). The potential value of diets with higher crude fibre levels can be readily evaluated under the appropriate conditions and this is an aspect of weaner nutrition which warrants further investigation.

Restricted feeding for weaner pigs

It has long been recognized that restricting the piglets feed intake during the first 7-10 days after weaning can reduce the incidence and severity of post-weaning diarrhoea, probably by increasing digestive efficiency and reducing substrate availability for bacterial proliferation. However, whilst such a practice might be conceivable for smaller units it is almost totally impractical for larger piggeries. On the other hand, because the piglets' voluntary feed consumption declines linearly with dietary energy concentration above 15 MJ DE/kg the use of high energy diets can serve a similar function as feed restriction. It is possibly for this reason that the high energy "English"-type weaner diets discussed previously seem so effective in improving the survivability and performance of the very light and less viable piglets at weaning. The extent that dietary energy concentration influences the voluntary feed intake of weaners is shown in Table 24.

Antigenicity of ingredients

A report on the potential of particular dietary ingredients to produce hypersensitivity responses in early weaned pigs by Newby *et al.* (1984) generated considerable interest in this factor as a possible cause of poor growth and even diarrhoea in the immediate post-weaning period. However, whilst nominal "antigenic" ratings for some ingredients have been published recently in the farming press this concept is not proven and there appears to be some confounding of the digestibility of different ingredients by newly weaned pigs and their so called antigenicity. In the longer term our efforts will be better rewarded if we concentrate on determining the dry matter and protein digestibility of ingredients for young pigs and use these values to formulate diets with accurate digestibility co-efficients for weaner pigs. At present most digestibility co-efficients for dietary ingredients are based on grower-finisher pigs and similar information is urgently required for weaner pigs.

Table 24. Effect of dietary energy concentration on the performance of piglets during the first 20 days after weaning

	Dietary DE (MJ/kg) ¹	
	14.4	17.4
Weight at weaning (kg)	6.12	5.70
Weight at 20 days post-weaning (kg)	12.20	10.90
Daily gain (g)	304	260
Feed intake (g/day)	600	360
Feed:gain	1.97	1.39
DE/kg gain (MJ)	28.3	24.5
Cost/kg gain (\$)	0.64	0.88

¹Lower and higher energy diets based on wheat and cooked rolled oats respectively.

Other factors

The strategic use of antibiotics remains a major weapon against post-weaning diarrhoea. However, again the most appropriate antibiotic(s) and the time period after weaning they might have to be included in the diet will depend on individual circumstances, and expert veterinary advice is generally required.

Australian scientists have tended to lead the world in the development of vaccines for controlling post-weaning diarrhoea and this technology has proved very successful in many commercial units in improving post-weaning performance by reducing the incidence and severity of diarrhoea.

Diets presented as a meal or in the form of small pellets (2.5 mm) also promote increased nutrient digestibility and better growth performance in the period immediately

after weaning compared to the same diets offered as 4.0 or 5.0 mm pellets (Campbell and Mackenzie, unpublished data).

Summary and conclusion

Debating the relative merits of nutrients and ingredients as they impact on weaner performance is essentially an exercise in futility. Both factors play equally important roles in successful weaner nutrition. The levels of dietary nutrients and choice of ingredients need to be based on the piglets' capacity for muscle growth and developing digestive function, which are affected by age and weight at weaning, and should be altered as these factors change with time after weaning.

The various techniques discussed in this paper for imparting prophylactic functions to weaner diets are not new and have tended to be dismissed in the past. However, the potential for selecting ingredients on the basis of their acid binding capacity and the use of higher crude fibre levels for first-stage weaner diets warrants further investigation. These techniques however, might not be required if the piglets gastric and intestinal development could be enhanced prior to weaning and/or its weight at weaning increased. In these respects further research is required on creep feeding practices and the nutritional management of the lactating sow and her litter.

SYMPOSIUM CONCLUSION

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The change from liquid milk to solid food has many consequences for the weaned baby pig. Liquid milk is more than a controlled regular supply of highly digestible and available nutrients. It also provides immunological and non-immunological protective agents and stimulatory and regulatory factors which may be important for the development of the digestive tract and its regulatory systems. At best, the post-weaning diet only replaces the nutrients and, even when provided in a regular and controlled way, it is consequently inferior to milk. The gastrointestinal system is far from completely developed even by four weeks of age.

To be able to efficiently utilize the post-weaning diet, the digestive system has to undergo a period of adaptation to increase its physical size and capacity, its secretory capacity of digestive enzymes and other chemical secretions and its absorptive capacity. Of all the components of the gastrointestinal system, the small intestine is the most vulnerable to disruption in the post-weaning period and the younger a pig is at weaning, the longer will be the period of adaptation and recovery. While creep feeding has been observed to enhance the development of the stomach, the effects it has on the small intestine and pancreas have not been conclusively demonstrated.

Highly digestible, high density diets containing a significant proportion of milk products consistently support high rates of growth with minimal digestive disturbance in the smaller weaned pigs less than four weeks old. These diets can be fed virtually *ad libitum* from weaning onwards provided substantial intake has been achieved prior to weaning. High fibre diets may be beneficial for later weaned pigs if creep feed intake has been insufficient or when piglets are of greater live weight and eating well. The levels of dietary nutrients and choice of ingredients need to be based on the capacity for muscle growth and development of the digestive system of baby pigs. These are affected by age and weight at weaning. Diets containing high levels of good quality animal proteins with some milk products seem to give better results for smaller piglets. Cooking of cereals, and added fat with raised energy levels are also preferred for these little pigs for the first 7-14 days after weaning.

If such diets are not available, the first option to consider is to raise the weaning weight of the litter to increase development of the digestive system. This may be achieved by increasing the quantity and/or quality of sow milk consumed. This is not easy in practice, particularly for gilts, and is more easily accomplished by weaning at a greater age. In this context weaning at four weeks would appear to have distinct advantages over weaning at three weeks.

In conclusion a sound nutritional strategy for weaner pigs would appear to be based on time of weaning as a guide, but with the time factor overridden by live weight. For example, in a three week weaning situation, most pigs will respond favourably to a diet containing cooked cereals, animal proteins and some processed milk products and containing a minimum of 16 MJ DE/kg and 1 g available lysine/MJ DE. This should be fed during the week following weaning which is then followed by two weeks on a minimum of 15 MJ DE/kg diet with 0.85 g available lysine/MJ DE predominantly containing wheat and animal proteins. With four week weaning most piglets will weigh more than 5 kg and a 15 MJ DE/kg diet would be adequate for them. Any unthrifty piglets or animals less than 5 kg live weight will benefit from one or two weeks on the 16 MJ diet. Small pelleted diets result in less waste than meals with no difference in piglet performance.

Once piglets are established on dry feed and are older than six weeks of age, a wheat based diet with vegetable and animal proteins can be used successfully with a reduced energy content of 14.5 MJ DE/kg and a reduced available lysine content of 0.75 g/MJ DE. This would be fed to around 10 weeks of age or 30 kg live weight before changing to a standard grower/finisher ration.

Diets of this type based on good quality ingredients when fed fresh in good environmental conditions in piggeries with good health status should result in minimal incidence of diarrhoea, reduced requirement for medication and achievement of target growth rates suggested, giving 3 month old pigs every chance to reach slaughter weights at the optimum time and maximize returns to the pig producer.

Future research effort should be of a multidisciplinary nature involving physiologists, nutritionists and veterinarians working together with piggery management personnel. Some topics requiring further investigation are the digestibility of ingredients for baby pigs, means of increasing growth performance prior to weaning which may also include enhancing digestive development, and specifically research into protein utilization to elucidate interactions between age and weight at weaning and dietary amino acid levels in relation to development of protein digestion and absorption systems in the gastrointestinal tract of baby pigs.

References

- ABIN, J., CORNELIUS, S.G., EL KANDELGY, S.M., MOSER, R.L. and PETTIGREW, J.E. (1983). Pentagastrin stimulated gastric acid secretion in the weanling pig. *Journal of Animal Science*. **57**(Supplement 1):235.
- AGRICULTURAL RESEARCH COUNCIL. (1981). "The Nutrient Requirements of Pigs" (Commonwealth Agricultural Bureaux: Slough, UK).
- ALPERS, D.H. (1987). Digestion and absorption of carbohydrates and proteins. In "Physiology of the Gastrointestinal Tract" Vol. II, second edition, pp. 1469-1487, ed. L.R. Johnson (Raven Press: New York).
- AUMAITRE, A. (1972). Development of enzyme activity in the digestive tract of the suckling pig: Nutrition significance and implications for weaning. *World Review of Animal Production*. **8**:54-68.
- AUMAITRE, A. (1973). The influence of weaning methods on the productivity of sows. *World Review of Animal Production*. **9**:56-63.
- BARBEZAT, G.O., WATERWORTH, M.W., DANIEL, M., BANK, S. and TERBLANCHE, J. (1974). Effect of burimamide on histamine- and pentagastrin-stimulated acid and pepsin secretion in the pig. *South African Medical Journal*. **48**:1985-1990.
- BOLDUAN, G., JUNG, H., SCHNABEL, E and SCHNEIDER, R. (1988). Recent advances in the nutrition of weaner piglets. *Pig News and Information*. **9**:381-385.
- BRAUDE, R. (1981). Symposium on the function of the gastrointestinal tract in health and disease: Introduction. *Progress in Clinical and Biological Research*. **77**:841-846.
- BRAUDE, R. and NEWPORT, M.J. (1973). Artificial rearing of pigs. 4. The replacement of butterfat in a whole-milk diet by either beef tallow, coconut oil or soyabean oil. *British Journal of Nutrition*. **29**: 447-455.
- BRAUDE, R., NEWPORT, M.J. and PORTER, J.W.G. (1970). Artificial rearing pigs. 2. The time course of milk protein digestion and proteolytic enzyme secretion in the 28-day-old pig. *British Journal of Nutrition*. **24**:827-842.
- BRENT, G., HOVELL, D., RIDGEON, R.F. and SMITH, W.J. (1975). "Early Weaning of Pigs" (Farming Press: Ipswich, UK).
- BUDDINGTON, R.K. and DIAMOND, J.M. (1989). Ontogenic development of intestinal nutrient transporters. *Annual Review of Physiology*. **51**:601-619.
- BUESCHER, E.S. and PICKERING, K.J. (1986). Polymorphonuclear leukocytes in human colostrum and milk. In "Human Milk in Infant Nutrition and Health" pp. 160-173, eds. R.R. Howell, F.H. Morriss and L.J. Pickering (C.C. Thomas: Illinois).
- CAMPBELL, R.G. (1989). The nutritional management of weaner pigs. In "Manipulating Pig Production II", pp. 138-183, eds. J.L. Barnett and D.P. Hennessy (Australasian Pig Science Association: Werribee, Victoria, Australia).
- CAMPBELL, R.G. and DUNKIN, A.C. (1983). The influence of nutrition in early life on growth and development of the pig. 2. Effects of rearing method and feeding level on growth and development to 75 kg. *Animal Production*. **36**:425-434.
- CARR, J.R., BOORMAN, K.N. and COLE, D.J.A. (1977). Nitrogen retention in the pig. *British Journal of Nutrition*. **37**:143-155.

- CERA, K.R., MAHAN, D.C., CROSS, R.F., REINHART, G.A. and WHITMOYER, R.E. (1988). Effect of age, weaning and postweaning diet on small intestinal growth and jejunal morphology in young swine. *Journal of Animal Science*. **66**:574-584.
- CHANDAN, R.C., PARRY, R.M. and SHAHANI, K.M. (1968). Lysozyme, lipase and ribonuclease in milk of various species. *Journal of Dairy Science*. **51**:606-607.
- CLOSE, W.H. and STAINER, M.W. (1984). Effects of plane of nutrition and environmental temperature on the growth and development of the early weaned pig. 2. Energy metabolism. *Animal Production*. **38**:221-231.
- COLE, D.J.A., BEAL, R.M. and LUSCOME, J.R. (1968). The effect on performance and bacterial flora of lactic acid, propionic acid, calcium propionate and calcium acrylate in the drinking water of weaned pigs. *Veterinary Record*. **83**:459-464.
- CORRING, T. (1980). The adaptation of digestive enzymes to the diet. Its physiological significance. *Reproduction, Nutrition and Development*. **20**:1217-1235.
- CORRING, T., AUMAITRE, A. and DURAND, G. (1978). Development of digestive enzymes in the piglet from birth to 8 weeks. I. Pancreas and pancreatic enzymes. *Nutrition and Metabolism*. **22**:231-243.
- CORRING, T., AUMAITRE, A. and RERAT, A. (1972). Fistulation permanente du pancreas exocrine chez le porc application: reponse de la secretion pancreatique au repas. *Annales de Biologie Animale, Biochimie, Biophysique*. **12**:109-124.
- CORRING, T. and CHAYVIALLE, J.A. (1987). Diet composition and the plasma levels of some peptides regulating pancreatic secretion in the pigs. *Reproduction, Nutrition and Development*. **27**:967-977.
- CRANWELL, P.D. (1985a). The development of acid and pepsin (EC 3.4.23.1) secretory capacity in the pig: The effects of age and weaning. 1. Studies in anaesthetized pigs. *British Journal of Nutrition*. **54**:305-320.
- CRANWELL, P.D. (1985b). The development of the stomach in the pig: The effect of age and weaning. I Stomach size, muscle and zones of mucosa. In "Proceedings of the 3rd International Seminar on Digestive Physiology in the Pig" pp. 112-115, eds. A. Just, H. Jørgensen and J.A. Fernandez (Beretning Statens Husdyrbrugsforsøg Number 580: Copenhagen).
- CRANWELL, P.D. (1985c). The development of the stomach in the pig: The effect of age and weaning. II. Acid and proteolytic enzyme secretory capacity. In "Proceedings of the 3rd International Seminar on Digestive Physiology in the Pig" pp. 116-119, eds. A. Just, H. Jørgensen and J.A. Fernandez (Beretning Statens Husdyrbrugsforsøg Number 580: Copenhagen).
- CRANWELL, P.D., FOLTMANN, B., NEWPORT, M.J. and HOWARTH, G.L. (1987). Basal and pentagastrin-induced gastric acid and milk-clotting enzyme secretion in pigs from birth to 4 weeks of age. *Proceedings of the Nutrition Society*. **46**:25A.
- CRANWELL, P.D., NOAKES, D.E. and HILL, K.J. (1976). Gastric secretion and fermentation in the suckling pig. *British Journal of Nutrition*. **36**:71-86.
- CRANWELL, P.D. and STUART, S.J. (1983). The development of gastric secretion of pepsin and milk-clotting enzymes in the conscious young pig. *Proceedings of the Nutrition Society of Australia*. **8**:134.
- CRANWELL, P.D. and TITCHEN, D.A. (1974). Gastric acid secretion in newly born piglets. *Research in Veterinary Science*. **16**:105-107.
- CRANWELL, P.D. and XU, R.-J., (1986). Development of gastric acid secretion in pigs: The response to pentagastrin. *Proceedings of the Australian Physiological and Pharmacological Society*. **17**:187P.
- DREW, M.D. and OWEN, B.D. (1988). The provision of passive immunity to colostrum-deprived piglets by bovine or porcine serum immunoglobulins. *Canadian Journal of Animal Science*. **68**:1277-1284.
- DUKE, M. and HEADON, D.R. (1988). Lactoferrin: Its functions and its future. In "Biotechnology in the Feed Industry" pp. 189-197, ed. T.P. Lyons (Alltech Technical Publications: Kentucky).
- EASTER, R.A. (1988). Acidification of diets for pigs. In "Recent Advances in Animal Nutrition 1988" pp. 61-71, eds. W. Haresign and D.J.A. Cole (Butterworths: London).
- EFIRD, R.C., ARMSTRONG, W.D. and HERMAN, D.L. (1982a). The development of digestive capacity in young pigs: Effects of weaning regimen and dietary treatment. *Journal of Animal Science*. **55**:1370-1379.
- EFIRD, R.C., ARMSTRONG, W.D. and HERMAN, D.L. (1982b). The development of digestive capacity in young pigs: Effects of age and weaning system. *Journal of Animal Science*. **55**:1380-1387.
- ELLIOT, J.I., SENFT, B., ERHARDT, G. and FRASER, D. (1984). Isolation of lactoferrin and its concentration in sow's colostrum and milk during a 21-day lactation. *Journal of Animal Science*. **59**:1080-1084.
- ELSLEY, F.W.H. (1963). Studies of growth and development in the young pig. *Journal of Agricultural Science, Cambridge*. **61**:243-251.
- ELSLEY, F.W.H. (1970). Nutrition and lactation in the sow. In "Lactation" pp. 393-411, ed. I.R. Falconer (Butterworths: London).

- ENGLISH, P.R., DELIGEORGIS, S.G., DAVIDSON, F.M., DIAS, M.F.M., SMITH, W.J. and FOWLER, V.R. (1978). Evaluation of alternative diets and feeding systems for early-weaned piglets. *Animal Production*. **26**:398.
- ENGLISH, P.R., ROBB, C.M. and DIAS, M.F.M. (1980). Evaluation of creep feeding using a highly-digestible diet for litters weaned at 4 weeks of age. *Animal Production*. **30**:496.
- FLEMSTROM, G. (1987). Gastric and duodenal mucosal bicarbonate secretion. In "Physiology of the Gastrointestinal Tract" Vol. II, second edition, pp. 1011-1029, ed. L.R. Johnson (Raven Press: New York).
- FOLTMANN, B. (1981a). Gastric proteinases - structure, function, evolution and mechanism of action. *Essays in Biochemistry*. **17**:52-84.
- FOLTMANN, B. (1981b). Mammalian milk-clotting proteases: Structure, function, evolution and development. *Netherlands Milk and Dairy Journal*. **35**:223-231.
- FOLTMANN, B. (1986). Pepsin, chymosin and their zymogens. In "Molecular and Cellular Basis of Digestion" pp. 491-505, eds. P. Desnuelle, H. Sjostrom and O. Noren (Elsevier: Amsterdam).
- FOLTMANN, B. and AXELSEN, N.H. (1980). Gastric proteinases and their zymogens. Phylogenetic and developmental aspects. *Federation European Biochemical Societies Proceedings*. **60**:271-280.
- FOLTMANN, B., CRANWELL, P.D., NEWPORT, M.J. and HOWARTH, G.L. (1987a). Ontogeny of the pig gastric proteases: Chymosin (EC 3.4.23.4), Pepsin (EC 3.4.23.1) and Gastricsin (EC 3.4.23.3) (Proceedings of the 4th Animal Science Congress of the Asian-Australian Association of Animal Production Societies, p. 463).
- FOLTMANN, B., CRANWELL, P.D., NEWPORT, M.J. and HOWARTH, G.L. (1987b). Ontogeny of pig gastric proteases; observations on fetal, stillborn, unsuckled newborn, suckled and growing pigs. *Proceedings of the Nutrition Society*. **46**:26A.
- FOLTMANN, B., JENSEN, A.L., LONBLAD, P., SMIDT, E. and AXELSEN, N.H. (1981). A developmental analysis of the production of chymosin and pepsin in pigs. *Comparative Biochemistry and Physiology*. **68B**:9-13.
- FOLTMANN, B., LONBLAD, P. and AXELSEN, N.H. (1978). Demonstration of chymosin (EC 3.4.23.4) in the stomach of newborn pig. *Biochemical Journal*. **169**:425-427.
- FOLTMANN, B., SZECSEI, P.B. and TARASOVA, N.I. (1985). Detection of proteases by clotting of casein after gel electrophoresis. *Analytical Biochemistry*. **146**:353-360.
- FORTE, J.G., FORTE, T.M. and MACHEN, T.E. (1975). Histamine-stimulated hydrogen ion secretion by *in vitro* piglet gastric mucosa. *Journal of Physiology, London*. **244**:15-31.
- FREED, L.M., YORK, C.M., HAMOSH, M., MEHTA, N.R., STURMAN, J.A., OFTEDAL, O.T. and HAMOSH, P. (1986). Bile salt stimulated lipase: The enzyme is present in non-primate milk. In "Human Lactation. 2. Maternal and Environmental Factors" pp. 595-601, eds: M. Hamosh and A.S. Goldman (Plenum Press: New York).
- FRIEND, B.A., SHAHANI, K.M. and MATHUR, B.N. (1983). Newer advances in human milk substitutes for infant feeding. *Journal of Applied Nutrition*. **35**:88-115.
- FRIEND, D.W., GORRILL, A.D.L. and MACINTYRE, T.M. (1970). Performance and proteolytic enzyme activity of the suckling piglet creep-fed at one or three weeks of age. *Canadian Journal of Animal Science*. **50**:349-354.
- FUJITA, S., KOKUE, E., KUREBAYASHI, Y. and HAYAMA, T. (1980). Secretary kinetics of electrolytes in porcine gastric juice from Heidenhain pouch. *Japanese Journal of Veterinary Science*. **42**:401-406.
- GIESTING, D.W. (1986). "Utilization of Soy Protein by the Young Pig" (Doctor of Philosophy Thesis, University of Illinois: Urbana).
- GOLDMAN, A.S. and GOLDBLUM, R.M. (1980). The anti-infective properties of human milk. *Pediatrics Update*. **1**:359-367.
- GORELICK, F.S. and JAMIESON, J.D. (1987). Structure-function relationship of the pancreas. In "Physiology of the Gastrointestinal Tract" Vol. II, second edition, pp. 1089-1108, ed. L.R. Johnson (Raven Press: New York).
- GREEN, S. (1989). A note on the digestibilities of nitrogen and amino acids in meat, skimmed-milk and fish meals in young pigs. *Animal Production*. **48**:237-240.
- GYORGY, P., KUHN, R., ROSE, C.S. and ZILLIKEN, F. (1954). Bifidus factor. II. Its occurrence in milk from different species and its other natural products. *Archives of Biochemistry*. **48**:202-207.
- HAFEZ, E.S.E. (1974). "Reproduction in Farm Animals" third edition (Lea and Febiger: Philadelphia).
- HAMOSH, M. (1986). Enzymes in human milk. In "Human Milk in Infant Nutrition and Health" pp. 66-77, eds. R.R. Howell, F.H. Morriss and L.K. Pickering (C.C. Thomas: Illinois).
- HAMPSON, D.J. (1986). Alterations in piglet small intestinal structure at weaning. *Research in Veterinary Science*. **40**:32-40.
- HAMPSON, D.J. (1987). Dietary influences on porcine postweaning diarrhoea. In "Manipulating Pig Production" pp. 202-214, eds. APSA Committee (Australasian Pig Science Association: Werribee, Victoria, Australia).

- HAMPSON, D.J. and KIDDER, D.E. (1986). Influence of creep feeding and weaning on brush border enzyme activities in the piglet small intestine. *Research in Veterinary Science*. 40:24-31.
- HARADA, E., KIRIYAMA, H., KOBAYASHI, E. and TSUCHITA, H. (1988). Postnatal development of biliary and pancreatic exocrine secretion in piglets. *Comparative Biochemistry and Physiology*. 91A:43-51.
- HARADA, E., NAKAGAWA, K. and KATO, S. (1982). Characteristic secretory response of the exocrine pancreas in various mammalian and avian species. *Comparative Biochemistry and Physiology*. 73A:447-453.
- HARADA, E., NIYAMA, M. and SYUTO, B. (1986). Comparison of pancreatic exocrine secretion via endogenous secretin by intestinal infusion of hydrochloric acid and monocarboxylic acid in anaesthetized piglets. *Japanese Journal of Physiology*. 36:843-856.
- 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., McCAULEY, I., GOONERATNE, A.D. and WHITELY, J.L. (1984). Inadequacies of sow lactation: survival of the fittest. In "Physiological Strategies in Lactation" pp. 301-326, eds. M. Peaker, R.G. Vernon and C.H. Knight (Academic Press: London).
- HOLST, J.J. (1985). The neuro-endocrine control of the digestive processes. In "Proceedings of the 3rd International Seminar on Digestive Physiology of the Pig" pp. 17-34, eds. A. Just, H. Jørgensen and J.A. Fernandez (Beretning Statens Husdyrbrugsforsøg Number 580: Copenhagen).
- HOLST, J.J. (1986). Hormonal regulation of digestive secretions. In "Molecular and Cellular Basis of Digestion" pp. 79-112, eds. P. Desnuelle, H. Sjostrom and O. Noren (Elsevier: Amsterdam).
- HOLST, J.J., SCHÄFFALITZKY DE MUCKADELL, O.B. and FAHRENKRUG, J. (1979). Nervous control of pancreatic exocrine secretion in pigs. *Acta Physiologica Scandinavica*. 105:33-51.
- HONG, S.S. and MAGEE, D.F. (1970). Pharmacological studies on the regulation of pancreatic secretion in pigs. *Annals of Surgery*. 172:41-48.
- HOPFER, V. (1987). Membrane transport mechanisms for hexoses and amino acids in the small intestine. In "Physiology of the Gastrointestinal Tract" Vol. II, second edition, pp. 1499-1526, ed. L.R. Johnson (Raven Press: New York).
- INOUE, T. (1981). Possible factors influencing immunoglobulin A concentration in swine colostrum. *American Journal of Veterinary Research*. 42:533-536.
- ITO, S. (1987). Functional gastric morphology. In "Physiology of the Gastrointestinal Tract" Vol. I, second edition, pp. 817-851, ed. L.R. Johnson (Raven Press: New York).
- JAEGER, L.A., LAMAR, C.H., BOTTOMS, G.D. and CLINE, T.R. (1987). Growth-stimulating substances in porcine milk. *American Journal of Veterinary Research*. 48:1531-1533.
- JAMES, P.S., SMITH, M.W., TIVEY, D.R. and WILSON, T.J.G. (1987a). Epidermal growth factor selectively increases maltase and sucrase activities in neonatal piglet intestine. *Journal of Physiology*. 393:583-594.
- JAMES, P.S., SMITH, M.W., TIVEY, D.R. and WILSON, T.J.G. (1987b). Dexamethasone selectively increases sodium-dependent alanine transport across neonatal piglet intestine. *Journal of Physiology*. 393:569-582.
- JENNESS, R. (1986). Inter-species comparison of milk proteins. In "Developments in Dairy Chemistry" Vol. I, pp. 87-114, ed. P.F. Fox (Applied Science: London).
- KIDDER, D.E. and MANNERS, M.J. (1978). "Digestion in the Pig" (Scientifica: Bristol).
- KIDDER, D.E. and MANNERS, M.J. (1980). The level and distribution of carbohydrases in the small intestine mucosa of pigs from 3 weeks of age to maturity. *British Journal of Nutrition*. 43:141-153.
- KNUHTSEN, S., HOLST, J.J., KNIGGE, U., OLESEN, M. and NIELSEN, O.V. (1984). Radio-immunoassay, pharmacokinetics, and neuronal release of gastrin-releasing peptide in anaesthetized pigs. *Gastroenterology*. 87:372-378.
- KOONG, L.-J., NIENABER, J.A., PEKAS, J.C. and YEN, J.-T. (1982). Effects of plane of nutrition on organ size and fasting heat production in pigs. *Journal of Nutrition*. 112:1638-1642.
- LEIBHOLZ, J. (1981). Digestion in the pig between 7 and 35 d of age. 6. The digestion of hydrolyzed milk and soya-bean proteins. *British Journal of Nutrition*. 46:59-69.
- LEIBHOLZ, J. (1985). The digestion of protein in young pigs and the utilization of dietary methionine. *British Journal of Nutrition*. 53:137-147.
- LICHTENBERGER, L.M. (1982). Importance of food in the regulation of gastrin release and formation. *American Journal of Physiology*. 243:G429-G441.
- LIGHTFOOT, A.L. (1984). Diets for early weaned pigs. In "Recent Advances in Animal Nutrition" pp. 45-48, eds. W. Haresign and D.J.A. Cole (Butterworths: London).
- LINDBERG, T. and KARLSSON, B.W. (1970). Changes in intestinal dipeptidase activities during fetal and neonatal development of the pig as related to the ultrastructure of mucosal cells. *Gastroenterology*. 59:247-256.
- LINDEMANN, M.D., CORNELIUS, S.G., EL KANDELGY, S.M., MOSER, R.L. and PETTIGREW, J.E. (1986). Effect of age, weaning and diet on digestive enzyme levels in the piglet. *Journal of Animal Science*. 62:1298-1307.

- LUCAS, A. (1986). Breastfeeding and gut hormones. In "The Breastfed Infant - a Model for Performance" pp. 73-83, eds. L.J. Filer and S.J. Fomon (Ross Laboratories: Ohio).
- LONNROTH, I., MARTINSSON, K. and LANGE, S. (1988). Evidence of protection against diarrhoea in suckling piglets by a hormone-like protein in the sow's milk. *Journal of Veterinary Medicine*. 35B:628-635.
- LUCAS, I.A.M. and LODGE, G.A. (1961). "The Nutrition of the Young Pig" (Commonwealth Agricultural Bureaux: Farnham Royal, UK).
- MAGEE, D.F. and WHITE, T.T. (1965). Influence of vagal stimulation on secretion of pancreatic juice in pigs. *Annals of Surgery*. 161:605-607.
- MANDYLA, H. and XANTHOU, M. (1986). Function of leukocytes in human milk. In "Human Lactation. 2. Maternal and Environmental Factors" pp. 533-540, eds. M. Hamosh and A.S. Goldman (Plenum Press: New York).
- MARRABLE, A.W. (1971). "The Embryonic Pig. A Chronological Account" (Pitman: London).
- MAY, J.T. (1988). Microbial contaminants and antimicrobial properties of human milk. *Microbiological Sciences*. 5:42-46.
- MILLER, B.G., JAMES, P.S., SMITH, M.W. and BOURNE, F.J. (1986). Effect of weaning on the capacity of pig intestinal villi to digest and absorb nutrients. *Journal of Agricultural Science*. 107:579-589.
- MITSUOKA, T. (1982). Recent trends in research on intestinal flora. *Bifidobacteria Microflora*. 1:3-24.
- MLC. (1989). "Pig Yearbook 1989" (UK Meat and Livestock Commission: Milton Keynes).
- MOODY, N.W. and SPEER, V.C. (1971). Factors affecting sow farrowing interval. *Journal of Animal Science*. 32:510-514.
- MORRIS, F.H. (1986). Growth factors in milk. In "Human Milk in Infant Nutrition and Health" pp. 98-114, eds. R.R. Howell, F.H. Morriss and L.J. Pickering (C.C. Thomas: Illinois).
- MOUGHAN, P.J., CRANWELL, P.D. and SMITH, W.C. (1989b). An evaluation with piglets of bovine milk, hydrolysed bovine milk and isolated soyabean proteins for inclusion in infant milk formula. II. Stomach emptying rate and the postprandial change in gastric pH and milk-clotting enzyme activity. *Journal of Pediatric Gastroenterology and Nutrition*. In press.
- MOUGHAN, P.J., PEDRAZA, M., SMITH, W.C., WILLIAMS, M. and WILSON, M.N. (1989c). An evaluation with piglets of bovine milk, hydrolysed bovine milk and isolated soyabean proteins for inclusion in infant milk formula. I. Effect on organ development, digestive enzyme activities and amino acid digestibility. *Journal of Pediatric Gastroenterology and Nutrition*. In press.
- MOUGHAN, P.J., WILSON, M.N., SMITH, C.H.M. and SMITH, W.C. (1989a). An evaluation of skim milk powders subjected to different heating conditions during processing as dietary protein sources for the young pig. *Animal Feed Science and Technology*. 22:203-215.
- MUGGENBURG, B.A., REIMANN, E.M., KOWALCZYK, T. and HOEKSTRA, W.G. (1967). Effect of reserpine and histamine in mineral oil-beeswax vehicle on gastric secretion in swine. *American Journal of Veterinary Research*. 28:1427-1435.
- NEWBY, T.J., MILLER, B., STOKES, C.R., HAMPSON, D. and BOURNE, F.J. (1984). Local hypersensitivity response to dietary antigens in early weaned pigs. In "Recent Advances in Animal Nutrition" pp. 49-59, eds. W. Haresign and D.J.A. Cole (Butterworths: London).
- NEWBY, T.J., MILLER, B., STOKES, C.R., HAMPSON, D. and BOURNE, F.J. (1985). Local hypersensitivity response to dietary antigens in early weaned pigs. In "Recent Developments in Pig Nutrition", pp. 211-221, eds. D.J.A. Cole and W. Haresign (Butterworths: London).
- NICKEL, R., SCHUMMER, A. and SEIFERLE, E. (1973). "The Viscera of the Domestic Mammals" (Verlag Paul Parey: Berlin).
- NIELSEN, H.E. (1964). Effects in bacon pigs of differing levels of nutrition to 20 kg body weight. *Animal Production*. 6:301-308.
- NOAKES, D.E. (1971). "Gastric Function in the Young Pig" (Doctor of Philosophy Thesis, University of London: London, UK).
- NRC. (1988). "Nutrient Requirements of Swine", ninth revised edition (National Research Council: Washington, DC).
- OKAI, D.B., AHERNE, F.X., HARDIN, R.T. (1976). Effect of creep and starter composition on feed intake and performance of young pigs. *Canadian Journal of Animal Science*. 56:573-586.
- OWSLEY, W.F., ORR, D.E. and TRIBBLE, L.R. (1986a). Effects of age and diet on the development of the pancreas and the synthesis and secretion of pancreatic enzymes in the young pig. *Journal of Animal Science*. 63:497-504.
- OWSLEY, W.F., ORR, D.E. and TRIBBLE, L.F. (1986b). Effects of nitrogen and energy source on nutrient digestibility in the young pigs. *Journal of Animal Science*. 63:492-496.
- PATON, M.W., MERCY, A.R. and WALLACE, J.F. (1988). An evaluation of the efficacy of cordial for preventing post-weaning diarrhoea in pigs. *Australian Veterinary Journal*. 65:46-49.
- PATTERSON, D.C. (1985). The response of pigs weaned at 16 or 21 days to skimmed milk powder or its components in the starter diet. *Animal Production*. 40:535.

- PELLETIER, G., LANOE, J. FILION, M. and DUNNIGAN, J. (1983). Effect of age and glucocorticoid administration on the proteolytic activity of gastric mucosa: A comparative study in the young rat, calf and piglet. *Journal of Animal Science*. **57**:74-81.
- PHELPS, A. (1987). Weaning weight determines quality of pig starter feed. *Feedstuffs*. **59**:11.
- PICKERING, L.K. and KUHL, S. (1986). Human milk humoral immunity and infant defence mechanisms. In "Human Milk in Infant Nutrition and Health" pp. 123-140, eds. R.R. Howell, F.H. Morriss and L.K. Pickering (C.C. Thomas: Illinois).
- PLUSKE, J.R. and WILLIAMS, I.H. (1988). Split weaning increases the growth of small pigs. *Proceedings of the Australian Society of Animal Production*. **17**:453.
- POND, W.G. and HOUP, K.A. (1978). "The Biology of the Pig" (Comstock Publishing: Ithaca).
- REITER, B. (1985). The biological significance and exploitation of the non-immunoglobulin protective proteins in milk: Lysozyme, lactoferrin, lactoperoxidase, xanthine oxidase. *International Dairy Federation Bulletin*. **191**:1-35.
- ROWLAND, A.C. (1981). Intestinal pathology. *Pig Veterinary Society Proceedings*. **7**:27-28.
- SANDERS, M.J. and SOLL, A.H. (1986). Characterization of receptors regulating secretory function in the fundic mucosa. *Annual Review of Physiology*. **48**:89-101.
- SANGILD, P.T., CRANWELL, P.D., XU, R.-J., and HENNESSY, D.P. (1989b). Gastric acid and enzyme secretion in young pigs following chronic administration of adrenocorticotrophin (ACTH). *Proceedings of the Australian Physiological and Pharmacological Society*. **20**:51P.
- SANGILD, P.T., FOLTMANN, B. and CRANWELL, P.D. (1989a). Development of the porcine gastric proteases. The effect of age, ACTH-treatment and early weaning. *Acta Veterinaria Scandinavica*. In press.
- SCHEELE, G. and KERN, H. (1986). The exocrine pancreas. In "Molecular and Cellular Basis of Digestion", pp. 173-194. eds. P. Desnuelle, H. Sjostrom and O. Noren (Elsevier: Amsterdam).
- SCHULZE, F. and MULLER, G. (1980). Lysozyme in sow's milk and its importance for bacterial colonisation of the gastrointestinal tract of the unweaned piglet. *Archiv fur Experimentelle Veterinarmedizin*. **34**:317-324.
- SELF, H.L. AND GRUMMER, R.H. (1958). The rate and economy of pig gains and the reproductive behaviour in sows when litters are weaned at 10 days, 21 days or 56 days of age. *Journal of Animal Science*. **17**:862-869.
- SHIAU, Y.-F. (1987). Lipid digestion and absorption. In "Physiology of the Gastrointestinal Tract" Vol. II, second edition, pp. 1527-1556, ed. L.R. Johnson (Raven Press: New York).
- SHIELDS, R.G., EKSTROM, K.E. and MAHAN, D.C. (1980). Effect of weaning age and feeding method on digestive enzyme development in swine from birth to ten weeks. *Journal of Animal Science*. **50**:257-265.
- SMIDT, D., SCHEVEN, B. and STEINBACH, J. (1965). The influence of lactation on reproductive function in sows. *Zuchtungskunde*. **37**:23-27.
- SMITH, M.W. (1984). Effect of postnatal development and weaning upon the capacity of pig intestinal villi to transport alanine. *Journal of Agricultural Science*. **102**:625-633.
- SMITH, M.W. (1985). Expression of digestive and absorptive function in differentiating enterocytes. *Annual Reviews of Physiology*. **47**:247-260.
- SMITH, M.W. and JARVIS, L.G. (1978). Growth and cell replacement in the new-born pig intestine. *Proceedings of the Royal Society of London*. **203B**:69-89.
- SMITH, M.W. and PEACOCK, M.A. (1980). Anomalous replacement of foetal enterocytes in the neonatal pig. *Proceedings of the Royal Society of London*. **206B**:411-420.
- STOCKILL, P. (1989). Acids: The next breakthrough in piglet nutrition? *The Feed Compounder*. **9**:56-60.
- TERROINE, E.F. and SPINDLER, H. (1925). De l'influence des divers procedes de pasteurisation par chauffage sur la digestibilite des constituants albuminoïdes et minéraux du lait. *Le Lait*. **5**:241-256.
- TRUGO, N.M.F. and NEWPORT, M.H. (1983). Resistance of vitamin B₁₂-binding protein in sow's milk to proteolysis *in vivo*. In "Proceedings of the 6th International Congress of Food Science and Technology" pp. 77-78, eds. J.V. McLoughlin and B.M. McKenna (Boole Press: Dublin).
- TSENG, C.-C. and JOHNSON, L.R. (1986). Does cortisone affect gastric mucosal cell growth during development? *American Journal of Physiology*. **250**:G633-G638.
- TUDOR, E.McI. (1983). "Studies on the Gastric Mucosa of Young Pigs" (Doctor of Philosophy Thesis, Monash University: Clayton, Victoria).
- VODOVAR, N., FLANZY, J. and FRANCOIS, A.C. (1964). Intestin grêle du porc. 1. Dimensions en fonction de l'âge et du poids, étude de la jonction du canal cholédoque et du canal pancréatique a celui-ci. *Annales de Biologie Animale, Biochimie, Biophysique*. **4**:27-34.
- WALSH, J.H. (1984). Gastric secretion. In "The Role of the Gastrointestinal Tract in Nutrient Delivery" pp. 107-118, eds. M. Green and H.L. Greene (Academic Press: New York).
- WATSON, R.G.K., VON HOORN HICKMAN, R. and TERBLANCHE, J. (1985). The "divided stomach" - A model for separate acid and alkali collection in the pig. *Journal of Surgical Research*. **38**:7-12.

- WIDDOWSON, E.M., COLOMBO, V.E. and ARTAVANIS, P.A. (1976). Changes in the organs of pigs in response to feeding for the first 24 h after birth. II. The digestive tract. *Biology of the Neonate*. **28**:272-281.
- WIDDOWSON, E.M. and CRABB, D.E. (1976). Changes in the organs of pigs in response to feeding for the first 24 h after birth. I. The internal organs and muscles. *Biology of the Neonate*. **28**:261-271.
- WILLIAMS, I.H. (1976). "Nutrition of the Young Pig in Relation to Body Composition" (Doctor of Philosophy Thesis, University of Melbourne: Melbourne, Victoria).
- WHITTEMORE, C.T. (1984). Nutrition of the sow and weaner (Proceedings of the Colborn-Dawes Nutrition Conference: UK).
- XU, R-J. (1989). "Studies on the Physiology of Gastrin in the Young Pig" (Doctor of Philosophy Thesis, La Trobe University: Bundoora, Victoria).
- ZEBROWSKA, T. (1973). Influence of dietary protein source on the rate of digestion in the small intestine of pigs. Part 1. Amount and composition of digesta. *Roczniki Nauk Rolniczych Seria B Zootechniczna*. **95**:115-131.

MONOSODIUM GLUTAMATE AS A FLAVOUR ENHANCER IN CREEP-WEANER DIETS FOR PIGLETS

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Monosodium glutamate (MSG) can stimulate feed intake and improve growth rate of young pigs particularly in those of low weaning weight (Gatel *et al.*, 1988). The aim of this experiment was to determine if MSG supplementation would improve the feed intake of piglets given a conventional wheat-based creep-weaner diet.

The control diet containing wheat, meat and bone meal, fish meal, soyabean meal, L-lysine and tallow was formulated to 15 MJ digestible energy (DE)/kg and 0.67 g available lysine/MJ DE. The MSG diet was produced by supplementation of the control diet with 5 g MSG/kg. Ten litters were allocated to each diet with attempts made to match litters of comparable size on each diet. Fresh feed was provided daily during the creep period (10-28 days) and via self feeders during the weaner period (29-49 days).

Table 1. Performance of piglets given monosodium glutamate (MSG) as a flavouring agent in creep-weaner diets

	Control	+MSG	SEM
Live weight (kg/piglet)			
10 days	3.24	2.97	0.141
28 days	7.13	6.96	0.224
49 days	13.03	13.13	0.369
Feed intake (g/piglet/day)			
10-28 days	24	18	4.9
29-49 days	429	433	13.7
10-49 days	242	242	8.2
Feed conversion ratio			
29-49 days	1.52	1.51	0.07

Feed intake was variable and only half that reported by Campbell (1976) for pigs over a three-week post-weaning period. There was no significant effect of MSG supplementation on any parameter during either the creep or weaner period. These results indicate no benefit of MSG as a flavour enhancer in a conventional wheat-based creep-weaner diet, despite feed intake being low, especially prior to weaning.

References

- CAMPBELL, R.G. (1976). *Animal Production*. 23:417-419.
 GATEL, F., BURON, G., GUION, P. and FEKETE, J. (1988). *Journées de la Recherche Porcine en France*. 20:375-379.

PROTEASE INHIBITORS IN CEREALS FOR PIGS

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Inhibitors of proteases (trypsin and chymotrypsin) are widely distributed in plant seeds, particularly grain legumes. Excessive ingestion of these inhibitors may depress animal growth due to an interference with the digestion of protein. Generally these inhibitors are heat labile and their levels are considerably reduced in meals which undergo heat treatment during processing. There is little information on the distribution of protease inhibitors in cereals and their presence is usually ignored. The aim of this investigation was to determine the trypsin and chymotrypsin inhibitor levels in a range of cereal grains used in the pig industry. Samples of the main varieties for six cereals were collected from grain producing areas in NSW. Trypsin and chymotrypsin inhibitors were readily extractable in tris-HCl buffer (pH 8.0, 0.1 M containing 10 mM CaCl₂) and assayed using chromogenic substrates (Saini, 1989).

Table 1. Trypsin and chymotrypsin inhibitor concentrations in cereals

	Number of samples	Trypsin inhibitor ¹		Chymotrypsin inhibitor ²	
		mean	range	mean	range
Barley	4	0.68	0.50-0.90	0.78	0.59-0.92
Maize	5	0.18	0.13-0.24	0.02	0.00-0.12
Oats	7	0.05	0.03-0.07	0.17	0.06-0.27
Rye	5	0.81	0.54-1.07	1.08	0.80-1.27
Triticale	6	0.35	0.27-0.47	0.64	0.40-1.14
Wheat	5	0.15	0.10-0.23	0.22	0.14-0.45

¹Activity expressed as mg inhibitor/g sample, on the basis of 62% concentration of active trypsin in the commercial sample used as standard; ²activity expressed as mg inhibitor/g sample, assuming the concentration of active chymotrypsin in the commercial sample was 100%

There was considerable variation in the concentration of inhibitors between cereals, and chymotrypsin inhibitor concentrations were higher than trypsin inhibitor levels in the majority of cereals. The actual levels of trypsin inhibitors in cereals were considerably lower than those found in some grain legumes (field peas, 0.6-1.9; chick and pigeon pea, 2.1-5.2) (unpublished data).

Information on the pigs' tolerance to trypsin inhibitor level is lacking. Nevertheless, if grain legumes rich in trypsin inhibitors are being used, the concentrations in the cereals need to be considered. In this regard more grain legumes could be used with wheat or oat-based diets than with barley and rye.

References

SAINI, H.S. (1989). *Food Chemistry*. 32:59-67.

UTILIZATION OF ILEAL DIGESTIBLE LYSINE FROM DIFFERENT PROTEIN SOURCES BY GROWING PIGS

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There is uncertainty in the literature to the extent that amino acids absorbed in the ileum are available to the pig. If amino acids are fully available, then the ileal digestibility assay could be used to assess amino acid availability, and would be useful in determining amino acid requirements and formulating diets. This experiment was conducted to determine if values for ileal digestible lysine from different protein sources were, (1) suitable for formulating diets, and (2) to determine the retention of ileal digestible lysine by growing pigs.

Three sugar-based diets containing 350 g/kg cottonseed meal (CSM), 280 g/kg meat and bone meal (MBM) or 240 g/kg soyabean meal (SM) as the only source of protein were formulated to 0.36 g ileal digestible lysine/MJ digestible energy (DE). Other essential amino acids were added to ensure a 28% surplus relative to lysine. The pigs were fed frequently, at a feeding scale of three times maintenance, over the 20-45 kg growth phase. The pigs were then slaughtered and the lysine content in the empty bodies determined.

Table 1. Performance of pigs given diets formulated to 0.36 g ileal digestible lysine/MJ DE over the 20-45 kg growth phase

	Protein source			SEM
	CSM	MBM	SBM	
Gain (g/day)	377 ^c	492 ^b	541 ^a	11.5
Feed conversion ratio	3.5 ^c	2.6 ^b	2.3 ^a	0.07
Lysine retention:ileal digestible lysine intake	0.36 ^c	0.60 ^b	0.75 ^a	0.013

^{a,b,c}differ at $P < 0.01$

Growth rate, feed conversion ratio and retention of ileal digestible lysine were all significantly different ($P < 0.001$) for the pigs given the three protein sources.

These results indicate that lysine is absorbed in the ileum in a form(s) that is(are) not fully utilized. As such, values for ileal digestible lysine are not effective in diet formulations as they do not reflect the amount of lysine that is available to the pig.

CRITERIA FOR ASSESSING THE REQUIREMENTS AND AVAILABILITY OF PHOSPHORUS IN GROWING PIGS

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The large variation in estimates of phosphorus (P) requirement for pigs may be due to different criteria (growth or bone mineralization) being used to assess response (NRC, 1988). The aim of this study was to assess the usefulness of growth or bone development as criteria for P requirements and availability in growing pigs.

The experiment was a 4x4 factorial design involving four levels of calculated available P (1, 2, 3, and 4 g/kg) and four calcium:available P ratios (1.7, 2.1, 2.5 and 2.9). Diets were formulated by substituting the required amounts of limestone and sodium tripolyphosphate for sugar in soyabean meal and sugar based diets. The diets were offered *ad libitum* to 96 pigs over the 20-50 kg growth phase. The pigs were then slaughtered and the metatarsal bone (M) collected for bone bending moment and the radius/ulna bone (RU) for other parameters.

Table 1. Response of pigs to level of available P

Response criteria	Available P (g/kg)				Statistics			
	1	2	3	4	%Var ¹	Lin	Quad	SEM
1. Growth performance								
Feed intake (g/day)	2052	2106	2118	2031	0	NS	NS	35
Growth rate (g/day)	915	946	955	916	0	NS	NS	15
FCR	2.2	2.2	2.2	2.2	0	NS	NS	0.03
2. Bones								
RU fresh weight (g)	81	84	87	88	11	**	NS	2.00
RU dry weight (g)	44	51	53	55	40	**	NS	1.23
RU dry matter (g/kg)	550	600	610	630	45	**	**	8.00
RU ash (g/kg)	490	530	540	540	66	**	**	3.90
RU ash weight (g)	17	22	23	25	70	**	**	0.46
M bending moment (kg/cm)	24	34	38	43	58	**	NS	1.28

¹% variation due to linear effect of available P excluding other variables

The main effects of the response to available P are shown in Table 1. Growth performance was not influenced by available P. The results indicate that bone ash was the most sensitive parameter for assessing P requirement as it had the highest proportion of per cent variation explained by the linear regression equations. However for P availability, bone bending moment was preferable as it had the highest per cent variation of those criteria which responded linearly to the increase of the available P. Recommendations for available P requirements would be ≤ 1 g/kg if based on growth, ≤ 3 g/kg if based on bone ash and ≥ 4 g/kg if based on bone bending moment.

References

NATIONAL RESEARCH COUNCIL. (1988). "Nutrient Requirements of Swine" ninth revised edition (National Academy Press: Washington DC).

RETENTION OF ILEAL DIGESTIBLE LYSINE BY GROWING PIGS

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Leibholz (1985) reported that the retention of ileal digestible lysine from five diets ranged from 0.86 to 0.94 and suggested that the ileal digestibility assay could be used to assess lysine availability for pig diets. These estimates of lysine retention are, however, higher than retentions recorded at our Institute (0.53-0.63; Batterham *et al.*, unpublished data). It is possible that the efficiency of lysine retention could be affected by the lysine status of the diet and this may account for the difference in the above estimates. Accordingly, an experiment was conducted to determine the effect of dietary lysine concentration on lysine retention.

Eight sugar-based diets were formulated to contain 0.09-0.72 g ileal digestible lysine/MJ digestible energy (DE) with soyabean meal as the sole source of lysine in the diets. Additional essential amino acids were added to maintain a minimum 20% surplus of other essential amino acids relative to lysine. Eight pigs were allocated at 20 kg live weight to each diet and were fed frequently at a feeding rate of three times maintenance. They were slaughtered at 45 kg live weight, and the lysine content determined in the empty bodies.

Table 1. Response of pigs given diets formulated from 0.09-0.72 g ileal digestible lysine/MJ DE over the 20-45 kg growth phase

	0.09	0.18	0.27	0.36	0.45	0.54	0.63	0.72	SEM
Gain (g/day)	176	392	481	561	658	688	730	727	12.5
Feed conversion ratio	5.7	3.2	2.7	2.3	2.0	1.9	1.8	1.8	0.09
Lysine retained:ileal digestible lysine intake	0.18	0.55	0.61	0.71	0.71	0.70	0.55	0.52	0.022

These were significant ($P < 0.001$) linear and quadratic responses in gain/d and feed conversion ratio to lysine concentration. Lysine retention responded in a linear and quadratic manner ($P < 0.001$) up to a estimated maximum of 0.73 at 0.45 g ileal digestible lysine/MJ DE.

These results indicate that, (1) lysine retention was affected by dietary lysine concentration and, (2) maximum retention was only 0.73. As such, a measure of retention of ileal digestible lysine cannot necessarily be used to estimate availability.

References

LEIBHOLZ, J. (1985). *British Journal of Nutrition*. 53:615-624.

EFFECTS OF CIMATEROL ON PIG GROWTH AND NUTRIENT DIGESTIBILITY

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Cimaterol (CIM) is an orally active β -adrenergic agonist that depresses appetite and increases body leanness (Moser *et al.*, 1986). It is not clear if this carcass effect is due to altered nutrient utilization or to reduced energy intake *per se*. This paper reports growth and metabolic responses of pigs to CIM when appetite effects were nullified by pair-feeding; meat quality effects have been presented by Thornton *et al.* (1989).

In the growth study, 20 entire male and 20 female pigs were used to test five dietary treatments: T1, control diet given *ad libitum*; T2, CIM at 0.5 mg/kg given *ad libitum*; T3, control diet pair-fed to T2; T4, CIM at 1.0 mg/kg given *ad libitum*; and T5, control diet pair-fed to T4. The barley-based diet contained (g/kg): Ca, 6.5; total P, 6.2; total Lys, 9.5, M+C, 5.8; and Thr, 5.8. Pigs were trialed from 53.4 kg (SD \pm 1.92) until slaughtered at 92.1 kg (SD \pm 2.54). Results are shown in Table 1.

Table 1. Growth responses of pigs given diets with or without CIM

	T1	T2	T3	T4	T5	LSD ¹
Feed intake (kg)	116.3 ^a	110.1 ^{bc}	110.7 ^{ab}	105.0 ^c	105.4 ^{bc}	5.75
Daily gain (kg)	0.96	1.05	1.06	1.02	0.98	0.061
FCR (kg:kg)	2.89	2.77	2.74	2.71	2.85	0.188

¹LSD (P=0.05); ^{a,b,c}differ at P<0.05

The apparent digestibility and N retention of diets with CIM at either 0, 0.5, 1.0 or 2.0 mg/kg were determined in crated pigs using a replicated 4x4 latin square design. Diet formulation was identical to that of the growth assay. Pigs were given a daily food allowance equal to 8% of the pig's live weight^{0.75}. Results are given in Table 2.

Table 2. Effect of CIM on apparent digestibilities and N retention

	Dietary CIM (mg/kg)				LSD (P=0.05)
	0	0.5	1.0	2.0	
Dry matter (%)	81.2 ^b	82.2 ^a	81.7 ^{ab}	82.3 ^a	0.81
Dig. energy (MJ:kg)	12.98 ^b	13.18 ^a	13.07 ^{ab}	13.17 ^a	0.174
N retained (%)	40.3 ^b	46.3 ^a	45.6 ^a	46.8 ^a	4.72

^{a,b}differ at P<0.05

CIM improved N retention, indicating a positive effect on protein metabolism. As food conversion was not significantly improved, CIM may also have increased metabolic rate. Both effects would favour a shift in the lean to fat ratio towards a leaner carcass.

References

- MOSER, R.L., DALRYMPLE, R.H., CORNELIUS, S.G., PETTIGREW, J.E. and ALLEN, C.E. (1986). *Journal of Animal Science*. 62:21-26.
 THORNTON, R.F., ADAMSON, D., HARRIS, P.V., SHORTHORSE, W.R. and WILLIAMS, K.C. (1989). In "Manipulating Pig Production II" p. 71, eds. J.L. Barnett and D.P. Hennessy (Australasian Pig Science Association: Werribee, Victoria, Australia).

AN EVALUATION OF MICROWAVE - TREATED SOYBEANS USING LABORATORY RATS

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Microwave processing of soybeans for pig diets may be less expensive than extrusion. The experiment described here used microwave-treated soybeans in rat diets as a model for pigs. Treatment was in an industrial, high-power conveyor microwave oven at an output of 14.1 kW for 2.5 or 5.5 min giving end point temperatures of 78 and 120 °C, respectively. The treated beans were assessed using standard criteria and methods.

Table 1. Effects of microwave heating of whole soybeans on trypsin inhibitor (TI) and urease activity, cresol red absorption (CRA) and available lysine (Av Lys), with extruded full fat soybeans (FFS) and soybean meal (SBM) as controls

Treatment	TI (mg/g DM)	Urease (Δ pH)	CRA (mg/g DM)	Av Lys (g/16 gN)
Raw	28.1 ^a	2.27 ^a	1.64 ^a	6.77 ^a
2.5 min	23.2 ^b	2.24 ^b	1.90 ^b	6.60 ^b
5.5 min	5.3 ^c	0.68 ^c	3.66 ^c	5.70 ^c
Extruded	5.2 ^c	0.37 ^d	3.90 ^d	4.91 ^d
SBM	4.9 ^d	0.13 ^e	4.14 ^e	4.68 ^d

^{a,b,c,d,e}differ at $P < 0.05$

Protein denaturation, as measured by CRA resulting from 5.5 min treatment, reduced both trypsin and urease activity (Table 1). Other proteins were made more susceptible to digestion despite a reduction in chemically-determined available lysine. Results of rats grown from 28 to 45 days on five dietary treatments, each of 10 rats, and given balanced diets containing 21% soybeans are shown in Table 2. Performance on the diet with 5.5 min treated soybeans was similar to that on diets with extruded soybeans and a control (commercial) diet.

Table 2. Mean (and overall SD in parentheses) of feed intake, growth rate, feed efficiency (FCR) and protein efficiency ratio (PER) of rats

Treatment	Feed intake (g/day)	Growth rate (g/day)	PER	FCR
Raw	13.6 ^a	4.3 ^a	1.42 ^a	3.56 ^b
2.5 min	13.8 ^{ab}	4.5 ^a	1.44 ^a	3.50 ^a
5.5 min	14.6 ^{ab}	5.5 ^b	1.65 ^b	2.98 ^b
Extruded	15.1 ^b	5.5 ^b	1.60 ^b	3.05 ^b
Commercial	16.3 ^c (0.19)	5.7 ^b (0.10)	1.56 ^b (0.021)	3.06 ^b (0.047)

^{a,b,c}differ at $P < 0.05$

Microwave treatment of soybeans is a potentially viable and inexpensive method of processing full-fat soybeans for inclusion in pig diets if confirmed with pigs.

DESIGNING THE PIG PEN

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Introduction

There are many published designs of pig pens (Sainsbury, 1970; Baxter, 1984; Brent, 1986) and an even wider range in practice. Many of these pen types have evolved over a period of years, gradually accommodating the latest technology and often the idiosyncrasies of the individual producer. It is easy to assume that because they exist, the pens must have been designed, but how were they designed? There are very few reported studies of pig pens having been designed from first principles. Baxter (1984) has made an attempt to draw together the basic principles of pig production and to use these to develop housing systems. This paper aims to consolidate and extend this, and later studies (Baxter, 1987, 1988). However, it would be unwise to believe that the design of a pig pen, or a complete building for that matter starts with a technical decision and one which depends only on the past and has no regard for the future. Although the issue is complex, the system oriented, iterative nature of design justifies a short statement of the wider issues involved.

The hierarchy of decision making

Technical decisions concerned with pig buildings are only part of a set of other decisions which deal with biological, agricultural, managerial, social, economic and political issues (Baxter, 1985a). Most of these major decisions emanate from a nested hierarchy of systems in which the effect of a decision passes and repasses from one system to the next in the form of a continuous dynamic interplay of forces, growing and decaying. Biological systems are at the very core of pig production, but in the commercial world they remain subservient to management. The managed biological systems are in turn subsumed by economic systems resulting from the industrial model of economic production. Management manipulates animals in response to economic factors. Finally, all these systems are embraced by the political system in which economics may be manipulated for ideological reasons and often in apparently irrational ways. This model of decision making is, of course, simple and perhaps a little unreal, but it is enough to demonstrate that it is almost impossible to develop a set of basic requirements for the design of pig accommodation which would remain inviolate across space, time and thought. In the face of irrational political decisions or crude economics, few technical principles can be sustained in practice.

Nevertheless, it is clearly important to be aware of the dynamic interplay of these systems in order to be able to evolve a strategy(s) for the present design of future structures. It is impossible to handle all the combinations of decisions which might arise but it is equally impossible to design for total uncertainty. Where the latter appears to exist then for design to be possible some certainties must be assumed. One way is to develop a preferred model or scenario of the future and then to examine its implication for the design of buildings and facilities. Many scenarios are possible and the following example will be used to provide the background for the remainder of this paper.

Change and the future of pig production

There is no guarantee that any proposed scenario will be correct and the further ahead one forecasts the less likely it will be that the prediction will be correct. Nevertheless, we have to look ahead; buildings erected now will be in use for the next 10-20 years. The following model of change is again rather simple and likely to be unreal but it will help to provide the context for choosing criteria and designing buildings. It is also likely to provoke debate and promote thinking.

There are many forces which bring about change. In the last 25 years or so, two forces have been primarily responsible for the direction in which pig production buildings have developed. Both are responses to economic pressures. The first major force has resulted in the decline of labour on farms. The trend has been to reduce unproductive jobs like littering and muck removal first, then to move to mechanical feeding systems and perhaps ultimately to management by machine. As investment has been transferred from labour to buildings and equipment, a second force has sought to reduce the ever-growing burden of capital investment. This has usually been manifested in less space/pig or more pigs in the same space. To respond to the combination of these two forces, new and innovative ideas have allowed building solutions to progress in a more or less predictable fashion. For example, when in the group housing of pigs on straw, the combined response to both forces results in the reduction of space/pig and in the amount of litter used to bed the pigs, a point is reached when no further progress can be made. At such a point any further reduction in space accelerates the consumption of litter and the system regresses. At this point a major shift in thinking is needed. For example, it has been observed that only some activities require litter e.g. resting, and others, feeding and excreting, are the unproductive consumers of litter. When this is realised then systems distinguishing between feeding/excreting and resting are developed. Developments then progress as before until again the system-in-use reaches a critical point when a major innovation is required. To date this line of progress has resulted in partially and then fully slatted floors for growers and crates and stalls for sows. Can this trend continue?

Even if it were possible to give pigs less space with no litter and still effect economic production, will the forces that have given rise to this change continue? The answer is probably "yes", in some places, in some economic circumstances and in some socio-political regimes, but "no" in others. Where this trend continues, the system will ultimately reach the lowest space allowance/pig possible i.e. bodyspace in the most constrained posture. Where the trend changes, the way forward is less clear unless we can identify the new predominant forces for change. Some signs of change are already obvious in many European countries. The crude economic model is now a more refined ecological model and factors such as pollution, conservation, health and welfare are exerting a more significant role in bringing about change. If such a change is sustainable, then it is hard to see how pig production systems can continue to lose people, i.e. increase the number of animals managed/unit of labour and intensify built space i.e. provide less space/unit of animal accommodated. In the face of such dramatic change, it seems inevitable that our technical decision making regarding the bounded space we call buildings or pens will also change. We now need to look at the place of pen design in the spatial hierarchy of the built environment.

The spatial hierarchy

Pens are part of a hierarchy of spaces incorporated into the housing systems of pig production units (Baxter, 1987). As part of the hierarchy they are influenced by the larger system of which they are a part (the building) and the smaller systems which they contain (the cells). These three parts of the hierarchy have been described by Baxter (1987) as follows:

- (1) *The cell* (examples; sow stall, farrowing crate): This is the smallest spatial unit and usually contains or refers to only the single animal. Where the cell is bounded as in a farrowing crate, then its definition is clear. On the other hand, unbounded space associated with the single animal, such as the notion of "personal space" (McBride *et al.*, 1963) or the space to perform individual activities is less clear. Precise performance specifications are not yet available for most cells but it is now clear that such detail is essential if the pig's welfare is to be fully accommodated (Baxter and Baxter, 1984). Although the cellular space of the farrowing crate has been studied in some detail (Schwaller, 1981; Baxter and Schwaller, 1983; Clough, 1985), the main activities they recorded, standing up and lying down are appropriate to all cellular behaviours. The amount of space provided in a cell, bounded or otherwise, will be determined largely by the size of the pig's body and the behaviour to be accommodated. Where cells are bounded and designed to eliminate those behaviours requiring a gross change in position, then it is imperative that body space and dynamic space are adequately defined. Cells influence the internal design of pens.
- (2) *The room or pen* (examples; pens for groups of pigs, cages for piglets): As pens are aggregates of cells, then similar requirements are necessary to specify the design of space in them but they are further complicated by the need to consider the interacting behaviours of groups of pigs. In group situations, the amount of behavioural space is more difficult to define; it must accommodate social as well as individual behaviours. Many behaviour patterns are predictable and observable, others are changeable and highly responsive to environmental change. Most behaviours are influenced by the amount and quality of the space provided. Adaptation or maladaptation may result from novel or barren environments. Pens to a greater extent than cells may additionally be conceived as the aggregation of places, feeding, drinking, resting etc. In the past, an over concentration of studies of space to the exclusion of place has produced oversimplified design standards. The amount of space in a cell or pen is dominated by animal requirements and the interface with people is important but minimal in spatial terms. Pens influence the layout of buildings.
- (3) *The building*: As most buildings are the aggregation of cells, pens and ancillary spaces, their main feature is the pattern of spatial relationships and the dynamic interlinking of spaces and places to accommodate efficient management routines. For example, a building containing several pens each holding a number of pigs may also contain a space (passageway) for moving the animals, a pen for collecting the animals together and a cell for weighing individual pigs. The pattern and layout of these space is now determined by man/animal activities. Spaces like passageways are determined more by the requirements of stockpersons and feeder wagons than by the pigs. The location of one space or place in relation to another is the result of a predetermined work routine (like weighing pigs) and the economics of aggregating space into the smallest, most cost-effective building shape. However, the more a building is divided into pens and cells especially with fixed boundaries, the greater will be the residual space, the less adaptable the total space will be, and the more costly the resultant building. Changing the quality of the spatial boundaries will also change the cost of the building.

Clearly, then, to design a pen it is necessary to be aware of the function of the pen space and to understand the components of the cells and behaviours it must contain and also to be aware how, in aggregate the building layout will affect the shape and orientation of the individual pen.

Function and the performance concept

On any pig production unit there will be many pens of several different types. Pen types are often classified by function. Although function may be described by the principle activities and events which take place within the pen more often than not they are simply defined in production terms like, growing, finishing, gestating or in behavioural terms like mating farrowing or suckling. None of these descriptors are adequate in terms of a design brief for the pen.

All functional specifications however need to be preceded by a measurable set of aims. These aims will be dependent on who formulated them or indeed who they were formulated for, the producer, the stockperson, the accountant, the public or the pigs!

Baxter (1988) has suggested that the conventional, producer-oriented view of building function can be described in relation to the following aspects of productivity:

- (1) *Animal productivity*: Buildings modify climate, behaviour and resources, and by controlling predators and parasites, pigs are healthier; they grow faster on less food; morbidity and mortality are lower and reproductive efficiency is higher.
- (2) *Human productivity*: By modifying climate, behaviour and resources, personnel work more efficiently, they look after more animals in less time; they concentrate on the production aspects of pig husbandry and use machines for routine chores; work routines are undisturbed by inclement weather.

This view does not deny that animal welfare and environmental concerns are not part of the system, merely that their consideration has been subordinate to production. If the scenario described earlier is correct then this emphasis will change.

Since the mid-1960s (Harrison, 1964), there has been a revival of interest (Turner, 1980) in animal welfare directed particularly at practices in intensive animal production. The movement is vociferous and articulate; it has a militant wing and political power and in twenty years it has forced changes in legislation in some countries, united several European countries in intention (Council of Europe, 1976) and forced the publication of national Codes of Recommendations (MAFF, 1983; Agriculture Canada, 1984). In addition, and some would argue the ultimate in animal welfare, there has been a regrowth in interest in animal rights and a burgeoning philosophical debate (Singer, 1976; Clark, 1977; Regan, 1983).

Animal rights apart, there is now no doubt that a concern for animal welfare must be upheld by the designers of pig pens and buildings. Sufficient popular and political pressure has been exerted to demonstrate that some existing practices can be banned and new, welfare oriented systems will be encouraged. There is still much doubt on what is meant by animal welfare but here the view is taken that animal welfare is welfare as perceived by the pig (Baxter, 1983a). How do we know what the pig wants or needs from its environment? There is, as yet, no well-established criteria to answer this question although the application of the performance concept has been suggested as a means of linking biological criteria to design criteria (Baxter, 1983b). Performance requirements are intended to describe elements of welfare as the animal perceives them and to provide a foundation for animal-centred analysis of building and pen designs.

Performance requirements have been identified for sows as follows (Baxter and Baxter, 1984) and can, in most cases, be extrapolated to all pigs:

Respiration; hunger; thermoregulation; sleep; health; living space; sex; maternal requirements; neophilia/recreation; predictability and control; thirst; skin comfort; sociality.

When all of the requirements have been identified, then performance criteria and finally performance specifications can be developed. The former are concerned with the physical expressions of the performance requirement and so describe the dimension or dimensions on which the welfare-need can be quantified. The performance specification contains the quantitative description of the welfare need and identifies the boundaries of welfare acceptability. It is from the performance specification that design criteria for acceptable pig environments are derived for application to the creation of new pen systems or the evaluation of existing facilities. Though much of the detailed information needed to compile such performance specifications is not yet available there is no reason why the framework for animal-centred analysis should not be applied qualitatively now, in addition to the conventional measures of productivity.

Similar arguments can be extended to promote the notion of environmental concern and here too, pig production systems to be acceptable in future, will need to be evaluated for their effect on the surrounding environment. Any future system of pig production must now meet and satisfy three measures of evaluation (Baxter, 1988):

- (1) production;
- (2) welfare;
- (3) environment.

At present, welfare and environmental concerns are growing at the expense of production. The producers' problem is not a lack of awareness of these factors, but of getting some idea of the priority order of the factors and an idea of how to assimilate the appropriate data for the design of facilities.

From the range of performance requirements already outlined this paper will now concentrate on a detailed analysis of living space and thermoregulation.

Living space and pen design

Space and productivity

The effects of space allocation on the productivity of pigs has been reviewed (Syme and Syme, 1969; Petherick, 1983) and although the tendency is to suggest that live weight gain deteriorates as the area/pig decreases, this is an oversimplification and lacks complete agreement. The review by Petherick (1983) however suggests that pig productivity is only consistently depressed by space *per se* below a minimum value and that effects above this value are more likely to be attributed to inadequacies of place rather than space. There is a strong possibility that this threshold value for space lies between $0.019W^{0.67}$ and $0.024W^{0.67}$, where W = live weight of pigs in kg and the area is in m^2 . These values would appear to equate with "packing" density at sternum lying and full recumbency respectively. In cool environments, Bruce and Boon (1984) have suggested an alternative hypothesis to explain the reduced areas occupied when pigs huddle together or even lie on top of one another. Their suggestion indicates that pigs may reduce their occupied space until a mechanical stress of approximately 200 kg/m^2 exists. In hot environments thermal stress will induce pigs to lie apart and fully recumbent and where this in itself is insufficient, to wallow in excreta and reduce feed intake. Table 1 summarises the various concepts and provides and estimates of space allowances under different environmental conditions for pigs weighing 5-100 kg.

Space and welfare

The relationship between space and welfare is even less clearly identified although spatial recommendations are quoted in various codes of welfare (MAFF, 1983; Agriculture Canada, 1984). In the UK, the relationship of space to welfare has been officially described three times in the last 20 years. The technical committee formed to enquire into the welfare of animals kept under intensive livestock husbandry systems gave its recommendations in 1965 (Brambell, 1965). This was followed in 1971 by the first of the Codes of Recommendations for the welfare of livestock (MAFF, 1971) and then in 1983 by a revised Code of Recommendations (MAFF, 1983). Those aspects of these three sets of recommendations which concern space are summarised in Table 2. The recommendations clearly differ, but is there a recognisable trend or any evidence on which to base a comparison in order to explain the differences?

Baxter (1988) compared data from Wight and Clark (1984) and from Peet (1984) and concluded that from a range of six housing layouts only one scheme met all the welfare requirements for space, yet all schemes were presented as possible economic solutions. The comparison also showed that unless the criteria presented to buyers of pig houses are normalized then erroneous conclusions can be derived from apparently suitable data. It seems clear then that if the dimensioning of space is a determinant of pig pen design and space allowances are an important requirement for welfare then the two factors should be related.

The components of space

The total amount of space included in a pen probably consists of several component parts. Baxter (1987) has suggested the following descriptors of space.

Bodyspace: Bodyspace is the most obvious component of an occupied space and it can refer to animate or inanimate objects. It is the smallest amount of space which can be provided for an animal and its geometry is a function of the animal's body posture at any specific moment in time. It is not unreasonable to expect that the main linear dimensions of pigs should be proportional to $W^{1/3}$ and that surface area should be proportional to $W^{2/3}$ where W = live weight (kg). As early as 1879 apparently, Meek (1879) aimed to predict surface area from body weight using the general formula $A_s = C \cdot W^{2/3}$ where C is a constant of proportionality having a particular value for each species. Brody *et al.* (1928) and then later Deighton (1932) found that the equation $A_s = 0.097W^{0.633}$, where A_s = surface area (m^2) and W = live weight (kg) fitted their data best. Kelley *et al.* (1973) then found that the expression $0.0734W^{0.656}$ produced a better prediction for modern "meat-type" female swine. Grommers *et al.* (1970) described four different lying postures for pigs and then expressed the floor contact areas as a percentage of the total surface area calculated from the Brody equation. The constant of proportionality varied from 0.016 with the pig in the most relaxed recumbent posture to 0.006 when the pig supported itself on all four legs when resting. Hsia (undated) has also shown that these postures are related to environmental temperature with pigs adopting full recumbency in hot environments and sternal lying in cold environments.

Petherick and Baxter (1981) and Petherick (1982, 1983) have confirmed that the principal linear dimensions of pigs may be adequately expressed in the form $L = KW^{1/3}$ and then they predicted that the floor area occupied by an individual pig may be expressed in the form $A_f = CW^{2/3}$ where $C = 0.019$ for sternal recumbency and $C = 0.024$ for full recumbency. It is to be expected that these values should be greater than the floor contact areas expressed by Grommers *et al.* (1970). For the individual pig, it seems reasonable to assume that the area it occupies when resting will be proportional

Table 1. Space allowances for pigs from 5 to 100 kg live weight

Live weight	Threshold of productivity m ² /pig (thermoneutrality)		Threshold of mechanical stress m ² /pig (cool environments)	Minimum resting space m ² /pig (thermoneutrality)	Occupied space m ² /pig (hot environments)	Welfare codes m ² /pig (thermoneutrality)	
	$0.019W^{0.67}$	$0.024W^{0.67}$				1965	1971 1983
5	0.06	0.07	0.03	0.08	0.10	-	-
10	0.09	0.11	0.05	0.13	0.16	-	-
20	0.14	0.18	0.10	0.20	0.25	-	0.15
40	0.23	0.28	0.20	0.32	0.40	-	0.25
60	0.30	0.37	0.30	0.42	0.53	-	0.49 0.35
80	0.36	0.45	0.40	0.51	0.64	0.75	0.66 0.45
100	0.42	0.53	0.50	0.59	0.74	0.93	0.82 0.50

Table 2. Trends in welfare recommendations for space allocation in swine accommodation

Class of pig	Recommendations by	
	Technical committee on welfare of animals in intensive livestock husbandry systems (Brambell, 1965)	Code number 2, Pigs (MAFF, 1971)
Fatteners	68-96 kg = min 0.75 m ² /pig > 96 kg = min 0.93 m ² /pig	(Including maiden gilts) Adequate for sleeping and feeding and of such size that soiling of lying area may be avoided. > 57 kg = min 1 m ² /122 kg Proportionally more space for smaller pigs especially in straw yards.
Pregnant sows	Daily exercise; space to turn around; no tethering	Total floor space should be adequate for sleeping, feeding and exercising. Sleeping areas must accommodate all pigs lying on their sides. 20 kg = 0.15 m ² 40 kg = 0.25 m ² 60 kg = 0.35 m ² 80 kg = 0.45 m ² 100 kg = 0.50 m ²
Boars	No recommendation	Stalls and tethers not recommended
		Individual adult. Living space ≥ 7.5 m ² Living and servicing space ≥ 10 m ² Shortest dimension of pen ≥ 2.5 m
		Feed and lie down normally.
		Individual adult. Living space ≥ 7 m ² Living and servicing space ≥ 9.3 m ² Shortest dimension of pen ≥ 2.1 m
		The welfare of livestock - Pigs (MAFF, 1983)

to $W^{2/3}$, modified by a coefficient indicative of resting posture. For groups of pigs, the occupied space could be estimated from the expression $S = N \sum K_i B_i$; where S is the total occupied space, N is the number of pigs performing the i^{th} behaviour at any one time and B_i the static space associated with the i^{th} behaviours. Baxter (1984) has suggested that in a thermally neutral environment, groups of pigs will occupy space predicted from the equation $N \times 0.034W^{0.67}$ where N is the number of pigs in the group and W is the average live weight (kg). Later, Baxter and Zappavigna (1984) suggested that the minimum resting space could be estimated from the expression $0.027W^{0.66}$. Petherick (1982) concluded from her studies that a space allocation calculated from $0.030W^{2/3}$ would be adequate for pigs housed on fully perforated floors. The commonly expressed value of $0.5 \text{ m}^2/85 \text{ kg pig}$ (Peet, 1984) is equivalent to $0.027W^{0.66}$.

Dynamic Space: Dynamic space is the space occupied across an interval of time, where the body moves from one posture to another without a major change in location. The total space used by the behaviour is often referred to as the space envelope or kinetographic space. Standing up or lying down are typical examples of the use of dynamic space and this behaviour has been studied for sows in farrowing crates with interesting results (Schwaller, 1981; Baxter and Schwaller, 1983; Clough, 1985). The movement of only part of the animal's anatomy may also be used to define dynamic space and so facilitate the design of feeders for example (Baxter, 1986).

Behavioural Space: Behavioural space, which includes dynamic space also accounts for the space which is required when the body changes orientation or location. The spatial requirements of sows when turning round have also been studied (Mathieson *et al.*, 1983) and the results have been related, with some success to body dimensions. However, other than for specific activities, behavioural space is difficult to describe in detail or to predict in practice. Animals tend to use the space available to them for behavioural activities, focusing many of these activities on places defined by the location of artefacts such as feeders, drinkers etc. The difficulties of precise description are compounded when several animals engage in related behaviours. In such a situation, part of behavioural space may be considered as "social space", the space required to meet the psycho-social needs of one animal in the presence of others. It has been suggested that though pigs clearly require space to reflect aspects of sociality they do not appear to have definable social space (Baxter, 1985a). The space required is dependent upon what they are doing and in particular where they are in possession of a resource which they want to defend or retain. This suggests that in a space containing several animals, "place" (a resource) may be more significant than "space". Two other components of total space may be important.

Residual Space: Residual space is that component of space which cannot be used for behaviour. It is dependent on the size of the animal, its behavioural repertoire and the shape and location of boundaries. It is often a small component of total space and is sometimes utilized by management for alternative purpose like the storage of small objects. In some cases it may provide behavioural space for other animals like rats, mice and snakes.

System space: System space is another component of space which will be used for behavioural purposes because it is there, but its provision is a function of the management system rather than of the animal or its behaviour. The best example is to be seen in a comparison of groups of pigs kept on litter and those on fully perforated floors. In the former system, twice as much space will be provided. The extra space would appear to be determined by the system.

All spaces in a pig pen will be composed of some or all of these components. The significance of each component to the design of the total space will depend on the type and size of the space to be created.

The significance of places

When a group of pigs is given an amount of total space greater than some minimum "threshold of productivity", the variation in productivity appears to be related more to the provision of "places" than of space *per se*. There is the "feeding place" and "drinking place" usually located where the designer has installed the feeder and drinker. The "resting place" and the "excreting place", are sometimes defined by the designer's arrangements of slatted floors or separate dung passages but are always determined by the pig whatever the designers intentions! Within a general excreting area, pigs may also have individual excreting places (Baxter, 1977). There may also be other places chosen by the pigs themselves but not readily identified by designers or stockpersons. For example there may also be a "grooming place", "a play place", a "safe place" and even a "fighting place". There may also be more than one of each of these places. Some places may be mutually exclusive and others, because of the distribution of activities in time may be inclusive. For example, Baxter (1984) has suggested that the difference in space with *ad libitum* feeding may be due to the inclusive nature of feeding and resting space in restricted feeding systems and the exclusive nature of at least some space for *ad libitum* feeding. In the latter systems some pigs may seek access to the feeders at any time. Most designers try to ensure that the drinking place is included in the excreting area and both are excluded from the resting area. The exception occurs in pens with fully slatted floors. Even here however some space should be available around the drinking place which does not intrude on the resting space. Pigs may also continue to differentiate between resting and excreting places, even in a fully slatted pen.

Where activities are performed by only some pigs whilst the others perform different activities, the places may again be exclusive. Resting pigs will generally avoid areas of activity such as grooming, excreting, feeding and so on. Regarding safe places, McGlone and Curtis (1981) have shown that the provision of "hides" can reduce aggression and also reduce the amount of wounding on pigs. A more complete understanding of the space/time patterns of the activities of pigs in different environments is clearly required.

Places need to be described by at least three criteria:

- (1) "location", the relationship with other places in a total spatial pattern;
- (2) "focal object", or artefact around which the activity of place is performed;
- (3) "the space" required to perform the activity of place.

Designers will need to make an effort to describe places in these terms and to quantify the estimates of space before they proceed to develop new housing systems. There is no substantial body of data on the design of all places in a pig pen and only a limited effort has been made to quantify the most obvious of these places as follows.

Feeding Place

Baxter (1984) using data from Petherick (1982) has tried to demonstrate how two patterns of feeding, linear and point can be described in relation to the body size or weight of the pig. Using the following relationship for Large White x Landrace pigs (Petherick and Baxter, 1981):

Breadth at shoulder (mm)	$60W^{0.33}$
Length from tail to scapula (mm)	$175W^{0.35}$
Length from scapula to snout (mm)	$122W^{0.30}$

Baxter has suggested that the minimum occupied area at a linear feed trough with pigs closely packed in was $N \times 0.019W^{0.67}$ where N = number of pigs and W = average live weight (kg). With loosely packed pigs the expression was $N \times 0.023W^{0.67}$. In both cases, the area was measured over the feed trough to the point reached by the pig's snout. In addition, Baxter has suggested that there should be a clear distance behind the feeding pigs of $300W^{0.33}$ (mm) or $200W^{0.33}$ (mm) with close or loosely packed pigs respectively to allow animals to move out from their original feeding position and choose a new location.

The possibility of substituting quality (by design) for quantity of space as it applies to the feeding place has been studied by Baxter (1986). Using a trough space allocation of 1.1 x shoulder width as a baseline treatment, he looked at the effects of increased space, i.e. 2.2 x shoulder width and 4.4 x shoulder width and also at three qualitative design changes in the form of trough divisions as follows: Nose barriers, head barriers and head and shoulder barriers. Time spent feeding was not effected by treatment, but aggression was significantly reduced by head divisions ($P < 0.05$) and virtually eliminated by head and shoulder divisions ($P < 0.001$). Food wasted from the various treatments was found to increase dramatically across all treatments ($P < 0.001$) with least wastage being found with head divisions (109 g/feeding period) and most with the greatest trough space of 4.4 x shoulder width (1187 g/feeding period). The substitution of quality for quantity of space needs more study.

Drinking place

Using similar data to that applied to the design of feeding places, Baxter (1984) has suggested that at drinkers of the nipple or bowl type mounted on a wall, the area occupied by a drinking pig will also be about $0.019W^{0.67}$ (m^2). However, depending on the design and operation of the drinker the pig may be able to take up a position anywhere in a semi circle having a radius of $300W^{0.33}$ (mm). This amount of space should be clear for pigs to drink without intruding on the rest area in particular. However as with Baxter's work on feed place design, the design of drinkers could be altered to reduce water spillage and/or limit the amount of space occupied by pig(s) whilst drinking.

Excreting place

Many pens have "designer" type excreting places; partly slatted areas or dung passages but these do not guarantee use by the pigs for excreting purposes. Although some useful work has been carried out to try to understand and explain the excretory behaviour of pigs (Baxter, 1977), there is, as yet, little data on which to base spatial calculations. The occupied area of an excreting pig could be estimated from the expression $0.019W^{0.67}$ (m^2) or in terms of the area commanded, from the area of a circle, the diameter of which is equal to the length of the pig. However, the most important data is probably the number of pigs which are likely to excrete at about the same time and the pattern which develops during this vulnerable activity (Baxter, 1977). The tendency of pigs to adopt an excretory posture close to walls may have some influence on the shape of the excreting area or the design of its boundaries.

Fighting place

In an unstable group of pigs attempting to establish a dominance hierarchy, fighting will ensue. Using data from Jensen (1982) on the activity of inverse parallel pressing in aggressive encounters, Baxter (1985a) has suggested that the space required for this type of behaviour, approximately circular in shape, can be calculated from the expression $0.11W^{0.66}$ (m^2). Estimating the space for submissive retreat is however much more difficult. At present there appears to be no information on which to estimate how far an animal must retreat before submission is effective. The "hides" studied by

McGlone and Curtis (1981) may reduce the requirement for "retreating" space by accommodating alternative submissive behaviour. This may be a further example of the substitution of design for quantity of space.

Resting place

This may be the most significant space in the pen and its location may influence all uses of the remaining space for other behaviours. It is also the most readily calculated for size and can be predictably estimated for changes in environmental conditions as well as size of pig. The data suggested earlier for the calculation of body space of a number of pigs in different postures can be used to calculate the area of the resting place. The minimum amount of resting space, such as that occupied by pigs in a cold environment is likely to be around $0.019W^{0.67}$ (m²)/pig. With warmer temperatures the space occupied will increase to $0.024W^{0.67}$ where pigs are lying recumbent but in close proximity. The amount of space recommended by the welfare codes (MAFF, 1983; Agriculture Canada, 1986) generally exceeds these values. There is therefore now little reason to believe that pigs allocated space in accordance with the welfare standards will suffer depressed performance through lack of resting space. Two problems still require vigilance however:

- (1) if environmental temperatures in the pen rise towards the pig's upper critical temperature (UCT) then more resting space will be required or the quality of space will need to be changed by cooling, sprinkling etc. Where space only is provided, the resting space allocation may have to increase to $>0.03W^{0.67}$ (m²) or appetite may be depressed. Pigs may also resort to lying in wet, soiled areas of the pen;
- (2) even where total space in the pen may be adequate, the articulation of space(s) and the identification of places may not be compatible with the pig's use of the space.

A combination of effects from (1) and (2) may result in the problem of "dirty pens". Much has been said of the space and the places in a pen but nothing so far on how the boundaries of the space might influence the space itself or its use.

The boundaries of spaces

A principal feature of the spaces we have in buildings is that they are bounded, they have some sort of enclosing perimeter, a brick wall, a tubular steel rail or a mesh screen. Boundaries around spaces should be identifiable but they are not necessarily physical. The concept of personal space includes the idea of a non-physical but psycho-social boundary.

Boundaries influence the transfer of three commodities, matter, energy and information, from inside the pen to the adjacent spaces and vice versa. Doorways for example are intended to facilitate the transfer of matter (pigs) but they also enhance the transfer of energy (in air currents for example). Tubular divisions may obstruct the transfer of one type of matter (pigs) but allow the transfer of another (urine or faeces). Solid walls inhibit the transfer of some information between pigs on either side (visual communication) but have little effect on other systems of information communication (sound).

For a boundary to be effective, it should prohibit or enhance the transfer of the appropriate commodities at the least cost, and occupy the least space. The space influenced by a boundary is not just the space it occupies but also the space it influences within the pen. The space close to a solid wall can not be used for walking space but it can be used for resting. Open barriers may occupy less space, but by making the space next to them less secure for adjacent animals (Barnett *et al.*, 1987;

Holmes *et al.*, 1987) then they may have a deleterious effect on the use of that space, e.g. pigs may not choose to rest against barriers through which they can be terrorised. Conversely, by allowing information transfer through boundaries, some behaviours may be encouraged, e.g. wet areas on one side of an open barrier may encourage excretory behaviour on the other side.

Boundaries may also influence energy transfer by altering the flow of air currents. This in turn may influence the location of some behavioural activities, e.g. resting and also the amount of space and the shape of the space occupied by the behaviour.

Pen design

The design of a pig pen is no simple matter. The fact that many pens work may be attributed more to the amenable behaviour of the pigs than the ability of the designer. Few if any designers can predict the outcome of the behaviour of pigs in the pens they design, yet what is it they have designed the pen for but to accommodate behaviour?

Designing is a process and although this paper has tried to provide a rational basis for decision making and it has supplied design data in a animal-centred format one thing is still missing, a set of guiding rules or procedures, an algorithm for design decisions.

The conventional method of designing a pen for groups of pigs is simple. The total area is usually calculated by multiplying the number of pigs by some space allocation/pig. Where restricted feeding is practiced then the length of feed trough is calculated to give one dimension of the pen then divided into the area to give the second dimension. With *ad libitum* feeding, some arbitrary ratio of pen length to width is selected. Where part slatted floors are incorporated a minimum dimension of about 1.2 m is chosen and the slatted area extends the length or width of the pen. The drinker(s) are usually located over the slatted floor. This method makes no concessions or considerations to pigs and their behaviours and can therefore hardly be defended in welfare terms.

The method of zoocentric design has been demonstrated by Baxter (1984) yet no practical validation of the method is ever studied in new designs. This is unfortunate for the technique does allow hypotheses to be set and tested. The method does however require the designer to consider the behaviour of the pig and to predict a set of initial conditions. To ensure that the dynamics of pen-use are also considered, the designer must constantly check by mental simulation, the changing conditions of growth, feeding patterns, management, etc. The now ubiquitous computer jargon of "what if" needs to be continually invoked during the design process.

Baxter (1988) has suggested the following rules as examples of what a designer needs to establish when developing a pen from a pig's point of view:

- (1) The pig will choose a dry, warm draft free area in which to rest. These conditions can be specified in relation to the size of the pig, group size, feed intake, air temperature, air velocity and thermal properties of the floor (Bruce and Clark, 1979). Groups of resting pigs in a thermally neutral environment will occupy a space whose area is best approximated by the expression $0.024W^{0.67}$.
- (2) The area occupied by resting pigs will be influenced by the environmental temperature and the diurnal cycle. At temperatures below the lower critical temperature, occupied space will reduce to about $0.019W^{0.67}$, and at temperatures approaching UCT occupied space may exceed $0.03W^{0.67}$. Pigs resting at night may occupy similar spaces but at lower temperatures.

- (3) Pigs will choose and progressively occupy the resting area by first lying against walls and other resting pigs. Pigs may rest next to a feed trough which is used at specific times by all the pigs simultaneously (restricted feeding).
- (4) Pigs will not choose to rest in areas subject to commotion and disturbance such as around *ad libitum* feeders or drinkers or grooming points.
- (5) Pigs will rarely excrete in the area that has been chosen for resting but they will excrete in any space which is left after the resting area has been established.
- (6) Pigs may however choose to lie in wet, excretory areas if environmental temperatures rise to near their UCT.
- (7) In the excretory space, pigs will tend to defecate next to walls or corners where they can adopt a protected posture. Subordinates may be displaced from an overcrowded excretory area and may then eliminate anywhere. This may give rise to a new focus for excretion.
- (8) Where pigs are to be fed simultaneously at a trough, the trough should be fitted with dividers and the space should be based on 1.1 x shoulder width of the pigs. Extra space at the feeder with no dividers could lead to increased aggression and feed wastage.
- (9) Where self-feeders (*ad libitum*) are provided in the resting area, then the total resting space may have to be increased to accommodate feeding space whilst some pigs remain resting.
- (10) Drinkers should be located such that the space occupied by drinking pigs does not encroach on the resting area. Drinkers should be located where spillage and wastage can be readily drained away. A minimum of two drinkers should be provided in every pen with groups of pigs.

It is worth noting, that in behavioural terms these rules should be applicable to all pens whether totally slatted, partially slatted, littered or unlittered. Although the fully slatted floor caters for the indiscriminate pig, pigs do not necessarily change their excretory behaviour just because the floor has holes in it. The same is true of the fully bedded pen; just because all areas might make comfortable resting spaces does not mean that the pig will stop defecating.

It is not sufficient to derive a set of rules from an accumulation of evidence. Designing results in a unique integration of evidence and the result of applying the rules needs to be monitored, reviewed and, if necessary, recast in a revised format. Such reviews and results should be published for the benefit of others.

Conclusion

Designing a pig pen is not a simple activity. Technical decisions will be influenced by managerial, economic or even political circumstances pertaining at the time of designing and during the future life of the design. The future of new designs will be significantly influenced by considerations of human, animal and environmental welfare. The shape, location and articulation of spaces and places in a pig pen will be influenced by the building layout and the function of the pen and the latter should be expressed in performance terms.

The space in a pen is the combination of several spatial components and these should be considered from an animal as well as a managerial point of view (zoocentric design). Design data should be expressed in zoocentric terms (i.e. as function of the pig or its behaviour). Designing requires rules or algorithms as well as data and designers should apply these in an iterative fashion each iteration being an attempted mental simulation of the activities likely to be adopted in the pen. Pens should be

designed, then monitored and evaluated. Any corrections which are made should also result in corrections to the design model, to the facts or to the rules. Designs and evaluation studies should be published.

References

- AGRICULTURE CANADA. (1984). "Recommended Code of Practice for Care and Handling of Pigs" reprinted 1986 (Agriculture Canada: Ottawa).
- BARNETT, J.L., HEMSWORTH, P.H. and WINFIELD, C.G. (1987). The effects of design of individual stalls on the social behaviour and physiological responses related to the welfare of pregnant pigs. *Applied Animal Behaviour Science*. **18**:133-142.
- BAXTER, M. (1977). Analysis of some of the environmental determinants of excretory and lying areas of the domestic pig (*Sus scrofa*). (Internal Report Scottish Farm Buildings Investigations Unit: Aberdeen, Scotland).
- BAXTER, M.R. (1983a). Ethology in environmental design for animal production. *Applied Animal Ethology*. **9**:207-220.
- BAXTER, M. (1985a). In "Social Space for Domestic Animals" pp.116-127, ed. R. Zayan (Martinus Nijhoff: Boston).
- BAXTER, M. (1986). "The Design of the Feeding Environment for the Pig" (Doctor of Philosophy Thesis, University of Aberdeen: Scotland).
- BAXTER, M.R. and BAXTER, S.H. (1984). In "Welfare of Confined Sows" pp.281-286, eds. A. Aumaitre and R. Dantzer (Annales de Recherches Veterinaires 15: Paris).
- BAXTER, M.R. and SCHWALLER, C.E. (1983). In "Farm Animal Housing and Welfare" pp.181-195, eds. S.H. Baxter, M.R. Baxter and J.A.C. MacCormack (Martinus Nijhoff Publishers: The Hague).
- BAXTER, M.R. and ZAPPAVIGNA, P. (1984). A note on space use and spatial preferences in pigs (cited in Baxter, M., 1985a). In "Social Space for Domestic Animals" p. 126, ed. R. Zayan (Martinus Nijhoff: Boston).
- BAXTER, S.H. (1983b). In "Farm Animal Housing and Welfare" pp.xi-xvi, eds. S.H. Baxter, M.R. Baxter and J.A.C. MacCormack (Martinus Nijhoff Publishers: The Hague).
- BAXTER, S.H. (1984). "Intensive Pig Production" (Granada Publishing Ltd.: London).
- BAXTER, S.H. (1985b). In "Milk Production in Developing Countries" pp.368-381, ed. A.J. Smith (University of Edinburgh: Edinburgh).
- BAXTER, S.H. (1987). In "Alberta Pork Seminar - Disease Ventilation and Management" pp.1-41 (University of Alberta: Edmonton).
- BAXTER, S.H. (1988). In "Proceedings of the Banff Pork Seminar - Health, Housing, Management and Meat" pp.12-27, ed. M. Deacon (University of Alberta: Edmonton).
- BRAMBELL, F.W.R. (Chairman) (1965). "Report of the Technical Committee to enquire into the Welfare of Animals kept under Intensive Livestock Husbandry Systems" Cmdnd 2836 (HMSO: London).
- BRENT, G. (1986). "Housing the Pig" (Farming Press Ltd.: Ipswich).
- BRODY, S., COMFORT, J.E. and MATTHEWS, J.S. (1928). Further investigations on surface area with special reference to its significance in energy metabolism. *Missouri Agricultural Experimental Station Bulletin*. **115**.
- BRUCE, J.M. and BOON, C.R. (1984). A note on the relationship between induced mechanical stress and thermal stress in recumbent pigs. *Animal Production*. **38**:309-311.
- BRUCE, J.M. and CLARK, J.J. (1979). Models of heat production and critical temperature for growing pigs. *Animal Production*. **28**:353-369.
- CLARK, S.R.L. (1977). "The Moral Status of Animals" (Clarendon Press: Oxford).
- CLOUGH, C.E. (1985). "Environmental Design for Piglet Protection" (Master of Science, Thesis, University of Aberdeen: Scotland).
- COUNCIL OF EUROPE. (1976). "European Convention for the Protection of Animals Kept for Farming Purposes" (Council of Europe: Strasbourg).
- DEIGHTON, T. (1932). The determination of the surface area of swine and other animals. *Journal of Agricultural Science*. **22**:418.
- GROMMERS, F.J., CURTIS, S.E., ANTONISSE, H.W. and CHRISTISON, G.I. (1970). Floor contact area as a function of body weight and posture. *Journal of Animal Science*. **31**:1232-1234.
- HARRISON, R. (1964). "Animal Machines: (V. Stuart Ltd.: London).
- HOLMES, L.N., SONG, G.K. and PRICER, E.O. (1987). Head partitions facilitate feeding by subordinate horses in the presence of dominant pen-mates. *Applied Animal Behaviour Science*. **19**:179-182.
- HSIA, L.C. (Undated). Observations on the recumbent postural behaviour of pigs. (Unpublished report, Scottish Farm Buildings Investigation Unit: Aberdeen, Scotland).
- JENSEN, P. (1982). An analysis of agonistic interaction patterns in group-housed dry sows - aggression regulation through an "avoidance order". *Applied Animal Ethology*. **9**:47-61.

- KELLEY, K.W., CURTIS, S.E., MARZAN, G.T., KARARA, H.M. and ANDERSON, C.R. (1973). Body surface area of female swine. *Journal of Animal Science*. **36**:927-930.
- McBRIDE, G., JAMES, J.W. and SHOFNER, R.N. (1963). Social forces determining spacing and head orientation in a flock of domestic hens. *Nature*. **197**:1272-1274.
- McGLONE, J.J. and CURTIS, S.E. (1981). A behaviour/performance study to evaluate an alternative nursery pen design for swine. *Journal of Animal Science*. **51**:129-130.
- MATHIESON, F., BAXTER, M.R. and CLOUGH, C.E. (1983). Space for turning round by sows. (Report of UFAW Scholarship, Scottish Farm Buildings Investigations Unit: Aberdeen). Referred to by approval of the authors.
- MEEH, K. (1879). Oberflächenmessungen des menschligen. *Kopers Z. Biol.* **15**:425 (Cited by Kelley *et al.*, 1973).
- MAFF. (1971). "Codes of Recommendations for the Welfare of Livestock Code No.2 - Pigs" Leaflet 702 (MAFF: London).
- MAFF. (1983). "Codes of Recommendations for the Welfare of Livestock - Pigs" (MAFF: London).
- PEET, B. (1984). Buying fully slatted finishing houses. *Pig Farming*. **32**:32-33 and 35.
- PETHERICK, J.C. (1982). "A Biological Basis for the Design of Space in Pig Housing" (Master of Science Thesis, University of Aberdeen: Scotland).
- PETHERICK, J.C. (1983). In "Farm Animal Housing and Welfare" pp.103-120, eds. S.H. Baxter, M.R. Baxter and J.A.C. MacCormack (Martinus Nijhoff Publishers: The Hague).
- PETHERICK, J.C. and BAXTER, S.H. (1981). In "Modelling, Design and Evaluation of Agricultural Buildings" pp.75-82, ed. J.A.D. MacCormack (Scottish Farm Buildings Investigations Unit: Aberdeen).
- REGAN, T. (1983). "The Case for Animal Rights" (Routledge and Kegan Paul: London).
- SAINSBURY, D. (1970). "Pig Housing" (Farming Press Ltd.: Ipswich).
- SCHWALLER, C.E. (1981). "Space Utilization by Sows Standing up and Lying Down in Confinement" (Bachelor of Science {Honours} Thesis, Trent Polytechnic: England).
- SINGER, P. (1976). "Animal Liberation" (Jonathan Cape: London).
- SYME, G.J. and SYME, L.A. (1969). "Social Structure in Farm Animals" (Elsevier: Amsterdam).
- TURNER, J. (1980). "Reckoning with the Beast" (J. Hopkins Press Ltd.: London).
- WIGHT, H.J. and CLARK, J.J. (1984). "Farm Building Cost Guide, 1984." (Scottish Farm Buildings Investigation Unit: Aberdeen).

ENERGY EXPENDITURE IN PIGS: A NEW TECHNIQUE

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Conventional methods for the measurement of heat production in growing pigs rely on the direct measurement of oxygen consumption using either respiration chambers or the short-term measurement of gas exchange with ventilated hoods (head boxes) or face masks. An alternative approach is to use the Fick principle to calculate oxygen consumption as the product of cardiac output and the arteriovenous (AV) difference in blood oxygen concentration across the lungs. The continuous measurement of cardiac output is now possible using transit-time ultrasound to measure the volume rate of blood flow (Transonic Systems Inc., Cornell, USA).

Two female pigs were housed in individual metabolism crates at 22°C, 85% relative humidity, with still air (0.15 m/sec) and continuous lighting. Each pig was fed *ad libitum* a commercial pelleted diet estimated to contain 14 MJ digestible energy/kg, air-dry basis. Water was available from a nipple drinker. At 60 kg live weight, a 24 mm diameter blood flow probe was surgically placed around the pulmonary artery of each pig. Polyvinyl catheters were inserted in the right atrium and the femoral artery. After a two week recovery period, average cardiac output was monitored over 24 h and recorded at 5 min intervals with a data logger (Tain Electronics, Melbourne). Simultaneous venous and arterial blood samples were taken at hourly intervals for the measurement of blood oxygen concentration (Haemoximeter: Radiometer, Denmark). The pattern of blood flow and AV difference in blood oxygen concentration was similar in both pigs and the values for one pig are presented in the figure below. Voluntary food intake was 3387 g over the 24 h period.

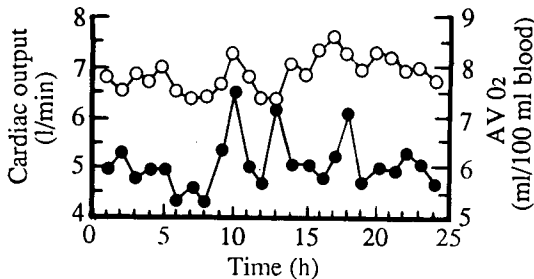


Figure 1. Hourly changes in cardiac output (open circles) and arteriovenous (AV) difference in blood oxygen (O_2) concentration (solid circles) for an 82 kg pig.

The 24 h mean and standard deviation of cardiac output and AV oxygen (O_2) concentration from Figure 1 was 6.8 ± 0.35 l/min and 6.1 ± 0.54 ml/100 ml blood respectively ($n=24$). The fluctuation in AV O_2 coincided with feeding activity. Mean oxygen consumption was 415 ± 47.4 ml/min which compared with 428 ml/min recorded with a head box on the same pig at 95 kg live weight and 366 ml/min recorded by Mount (1968) for 60 kg pigs housed in a respiration chamber. The technique offers an accurate continuous method of calculating oxygen consumption with individually-housed finisher pigs.

References

MOUNT, L.E. (1968). "The Climatic Physiology of the Pig" (Edward Arnold: London).

ENERGY EXPENDITURE OF PIGS EXPOSED TO HIGH TEMPERATURE

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Holmes and Close (1977) calculated that finisher pigs (60 kg live weight with a dry skin, fed three times maintenance energy intake) would show little change in rectal temperature or heat production over the temperature range 20-29°C. Because of the lack of experimental data and the practical significance of the responses of pigs to high ambient temperature, the objective was to measure the oxygen consumption of finisher pigs at 22°C and 30°C in the absence of skin wetness.

Two female pigs were housed in individual crates at 22°C and fed *ad libitum* a commercial pelleted diet estimated to contain 14 MJ digestible energy per kg, air-dry basis. Water was provided from a nipple drinker situated alongside each crate to exclude wetting of the skin surface. At 60 kg live weight each pig was surgically fitted with an ultrasonic blood flow probe (Transonic Systems Inc., Cornell, New York) around the pulmonary artery. Polyvinyl catheters were inserted in the right atrium and femoral artery. After a two week recovery period, both pigs were housed in a thermo-neutral environment (22°C; 85% relative humidity; still air speed 0.15 m/sec; continuous lighting) for 24 h before exposure to a high temperature environment (30°C; 75% relative humidity; still air; continuous lighting) for a further 24 h. Oxygen (O₂) consumption was calculated at 22°C and 30°C as the product of cardiac output and the arteriovenous (AV) difference in blood O₂ concentration (Table 1) as described by Giles *et al.* in this proceedings.

Table 1. Mean 24 h experimental data for two female pigs, mean live weight of 83.5 kg, housed at 22°C and 30°C

	Fig 1		Fig 2		SEM
Room temperature (°C)	22	30	22	30	
Voluntary food intake (g)	3748	1700	3387	1681	546
Voluntary water intake (l)	7.0	3.5	8.5	5	1.08
Cardiac output (l/min)	9.1	8.2	6.8	6.3	0.64
AV O ₂ (ml/100 ml blood)	5.2	4.4	6.1	5.0	0.35
O ₂ consumption (ml/min)	473	353	415	315	34.7
Rectal temperature (°C)	38.7	39.3	38.8	39.6	0.21
Respiration rate/min	25	107	24	90	21.6
Pulse rate/min	118	102	116	110	3.6

The results indicated that when first exposed to a 30°C-dry environment, an 83.5 kg pig will halve voluntary food intake and reduce oxygen consumption by 25% in order to maintain body temperature. It appears that a fall in energy expenditure is an important mechanism in the thermoregulation of finisher pigs at high temperature.

References

- HOLMES, C.W. and CLOSE, W.H. (1977). In "Nutrition and the Climatic Environment" pp. 51-73, eds. W. Haresign, H. Swan and D. Lewis. (Butterworths: London).

RECTAL TEMPERATURE OF PIGS EXPOSED TO HIGH TEMPERATURE

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Growing pigs are poorly adapted to high temperature in the absence of external water sources. Pigs are unable to sweat in response to heat, and respiratory evaporation is inadequate to maintain body temperature when ambient temperature rises above 30°C (Robinson and Lee, 1941). Much reported data on rectal temperature response to heat are confounded by inadequate definition of climatic variables and animal status, especially skin wetness and acclimatization. The objective was to measure the rectal temperature of finisher pigs exposed to high temperature in the absence of skin wetness.

Six male pigs were fed *ad libitum* a protein-adequate diet containing an estimated 14.5 MJ digestible energy/kg from 20-60 kg live weight and housed in individual metabolism crates under thermoneutral conditions with a summer light pattern (14 h light:10 h dark). Three pigs were exposed to 32.5°C, 50% relative humidity and still air (0.15 m/sec) for up to 12 h. The remaining three pigs were held in a thermoneutral environment (22.5°C; 70% relative humidity; air velocity 0.2 m/sec). Rectal temperature was measured approximately hourly and pigs were removed from the 32.5°C treatment once rectal temperature exceeded 40.5°C. Each pig had free access to a nipple drinker which was situated alongside each metabolism crate to exclude wetting of the skin surface. The two groups of pigs were interchanged and the experiment repeated after the pigs were housed in thermoneutral conditions for two days.

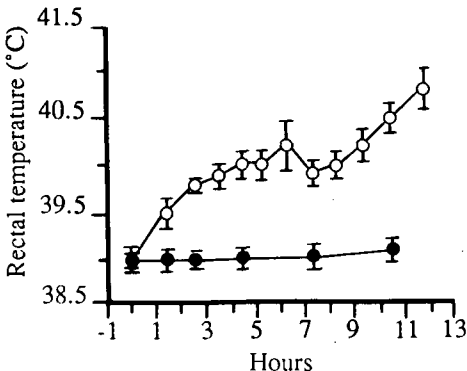


Figure 1. Relationship between rectal temperature and hours of exposure to either 32.5°C (open circles) or 22.5°C (solid circles) environmental temperature. Mean values with their standard errors. Six male pigs per temperature treatment weighing 69.6 kg live weight.

Rectal temperatures of pigs exposed to 32.5°C exceeded 40.5°C after 10.4 h exposure whereas pigs held at 22.5°C maintained rectal temperature at 39.0°C. The experiment indicated that an environmental temperature of 32.5°C exceeded the upper critical temperature for 69.6 kg male pigs which were unacclimatized to the heat and housed in a dry environment. These data infer that skin wetness from nipple drinkers, urine, faeces or water sprays is critical to the thermoregulation of growing pigs under commercial conditions where summer piggery temperatures exceed 32.5°C.

References

ROBINSON, K. and LEE, D.H.K. (1941). *Proceedings Royal Society, Queensland*. 53:145-158.

LYING - DOWN BEHAVIOUR OF LOOSE - HOUSED SOWS DURING DAY 1 AND DAY 8 AFTER PARTURITION

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Crushing of piglets by the sow after birth accounts for about 18% of pre-weaning deaths (English *et al.*, 1977). Many modern crate designs include rails to prevent the sow from flopping directly onto her side from a standing position and trapping piglets. It has been suggested by English *et al.* (1977) that sows with more space for movement are likely to increase the incidence of crushing. However, Signoret *et al.* (1975) claimed that sows lie down very cautiously and only after ploughing through the bedding with the snout. There is no detailed study on how sows lie down, or the behaviour and position of the piglets as the sow lies down after parturition. The aim was to collect data on this aspect.

Sows (Danish Landrace x Yorkshire) farrowed and lactated in a loose system (262 x 233 cm) with straw. On days 1 and 8 after parturition, sows (12 and 7, respectively) and litters (average 9) were videotaped for 24 h. Records included sow and piglet behaviours and sow positions just before lying, and how the sow lay down (Table 1).

This study supported the statement of Signoret *et al.* (1975). On 97% of occasions on day 1 sows either root bedding or move around prior to lying down, and on 75% of occasions sows lie down continuously with or without use of the rail.

Table 1. The frequencies and percentages of behaviours of sows immediately before lying down, the methods of lying down and the position and behaviour of the piglets on days 1 and 8

Behaviour	Frequency (%)	
	Day 1 (12 sows) (observations=114)	Day 8 (7 sows) (observations=104)
Sow before lying down - root	90 (79)	73 (70)
- move around	20 (18)	11 (11)
- stand	4 (4)	20 (19)
Method of lying down		
carefully - on front	43 (38)	55 (53)
- on side	24 (21)	36 (35)
rail used to slide on side	14 (12)	3 (3)
flop - on front - turn to side	11 (10)	3 (3)
- on front	10 (9)	3 (3)
- on side	8 (7)	2 (2)
rail used to slide on front	4 (4)	2 (2)

References

- ENGLISH, P.R., SMITH, W.J. and MACLEAN, A. (1977). "The Sow - Improving Her Efficiency" (Farming Press Ltd: Suffolk).
- SIGNORET, J.P., BALDWIN, B.A., FRASER, D. and HAFEZ, E.S.E. (1975). In "The Behaviour of Domestic Animals" pp. 295-329, ed. E.S.E. Hafez (Balliere Tindall: London).

THE PHYSIOLOGICAL AND BEHAVIOURAL RESPONSES OF PIGS TO DIFFERING DESIGNS OF INDIVIDUAL ACCOMMODATION

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The design of tether stall accommodation for pigs has significant effects on the behavioural and physiological responses related to their welfare (see Barnett and Hutson, 1987). In contrast, applying similar design features (vertical bars with or without mesh to manipulate aggressive interactions) to cage stalls had no effects on physiological responses compared to group housed pigs (Barnett *et al.*, 1989). The reasons for the different responses of pigs in cage stalls and tether stalls is unknown, although we have speculated (Barnett *et al.*, 1989) that the increased opportunity for movement in cage stalls affects aggressive behaviour.

This abstract reports on part of an experiment that compared the responses of pigs in two designs of cage stall. Sixteen pigs were mated over a six week period and eight were assigned to each of two treatments: (1) Vertical Stalls "VS" - individual cage stalls (0.6 x 2.0) m with the front 1.1 m of the stall division comprised of seven vertical bars 18.5 cm apart. (2) Horizontal Stalls "HS" - individual stalls of similar measurements to "VS" treatment except the side division was of five horizontal bars 18.5 cm apart. After 30 and 57 days in the treatments, a venous cannula was implanted and blood samples were collected four to five days later at 1-h intervals between 0800 and 1700 h to obtain a "daytime average" of cortisol concentrations; the mean values for the two periods were averaged for each pig. Observations of aggressive behaviours were made after 14 days in the treatments, from 60 min/pig of video records commencing 10 min after the start of feeding.

Mean free cortisol concentrations were significantly higher in the "HS" treatment (mean values \pm SE for "VS" and "HS" treatments were 4.1 ± 0.28 and 6.9 ± 0.74 nmol/l, respectively, $P < 0.01$). Behaviour recordings showed that pigs in the "HS" treatment spent less time in head to head contact with their neighbours (mean/pig of four focal pigs/treatment was 75 and 238 sec for the "HS" and "VS" treatments, $P < 0.01$) and fewer in the "HS" treatment were involved in aggressive interactions (2 and 8, chi-square test = 9.24, $P < 0.01$). The reduced contact in the "HS" treatment was also reflected in the time that banks of four neighbouring pigs spent concurrently within 31 cm of the front of the stall (51 and 440 sec for "HS" and "VS" treatments, respectively).

The data from this experiment indicate the importance of the design of individual accommodation and suggest reduced welfare in the "HS" treatment, based on the magnitude of the chronic stress response. Pigs in this treatment appear to be actively avoiding each other, although the cause(s) and function(s) of this avoidance are unknown.

References

- BARNETT, J.L., HEMSWORTH, P.H., NEWMAN, E.A., McCALLUM, T.H. and WINFIELD, C.G. (1989). *Applied Animal Behaviour Science*. In press.
- BARNETT, J.L. and HUTSON, G.D. (1987). In "Manipulating Pig Production" pp. 1-22, eds. APSA Committee (Australasian Pig Science Association: Werribee, Victoria, Australia).

PHYSIOLOGICAL RESPONSE TO EXERCISE IN PIGS

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Based on their adrenal response to a standard dose of adrenocorticotrophin (ACTH), consistently high responding or low responding pigs can be identified within a population of animals (Hennessy *et al.*, 1988). The present experiment examines the effects of exercise stress on some physiological responses in pigs with either a high (HR) or a low (LR) adrenal responsiveness to ACTH.

Pigs used were four HR and four LR, eight week old, male, Large White x Landrace. Plasma cortisol concentrations 1 h after injection with 6.25 I.U. ACTH (at two weeks of age) were HR, 781 ± 22 (mean \pm SE) nmol/l; LR, 303 ± 19 nmol/l. All pigs had a catheter placed in a femoral vein and were trained to walk on a treadmill before the experiment commenced. On the experiment day the pigs were removed from their pens and placed on the stationary treadmill for 1 h. They were then subjected to 40 min of walking at 2.16 km/h in the horizontal plane, followed by a 1 h recovery period on the stationary treadmill.

Transferring pigs to the treadmill resulted in an increase ($P < 0.01$) in oxyhaemoglobin saturation (SaO_2) and oxygen concentration (O_2), and an increase ($P < 0.05$) in plasma cortisol concentration. After 40 min exercise there were increases ($P < 0.001$) in body temperature, respiration and heart rate, and as shown in Table 1 significant rises in ACTH and cortisol. Blood SaO_2 and O_2 decreased ($P < 0.01$) during exercise. There were no significant changes in packed cell volume, haemoglobin or blood glucose throughout the experiment. One hour after the cessation of exercise, body temperature and respiration rate remained elevated, blood SaO_2 and O_2 increased ($P < 0.01$), while heart rate, plasma cortisol and ACTH decreased ($P < 0.001$).

Table 1. Plasma ACTH and cortisol concentrations in pigs before, during and after exercise (data are mean values \pm SE)

	Pigs	Rest		Exercise	Recovery
		Pen	Treadmill 1 h	After 40 min	After 1 h
ACTH (ng/ml)	HR	72 ± 15^a	83 ± 17^a	161 ± 14^b	80 ± 12^a
	LR	44 ± 5^a	64 ± 12^a	133 ± 15^b	57 ± 10^a
Cortisol (nmol/l)	HR	48 ± 12^a	99 ± 32^b	171 ± 38^c	97 ± 31^b
	LR	19 ± 5^d	33 ± 11^{ac}	91 ± 13^{bf}	31 ± 9^{ac}

For ACTH and cortisol different letters within rows and columns indicate differences at $P < 0.05$

Of all the observations and measurements made during these experiments differences between the HR and LR pigs were only apparent for plasma ACTH and plasma cortisol concentrations and only for the latter were the differences significant ($P < 0.05$). Thus differences in the pigs response to stress may be restricted to differences in the response of the pituitary adrenal axis.

References

HENNESSY, D.P., STELMASIAK, T., JOHNSTON, N.E., JACKSON, P.N. and OUTCH, K.H. (1988). *American Journal of Veterinary Research*. 49:1276-1283.

OPERANT RESPONDING BY SOWS ON RESTRICTED RATIONS FOR ADDITIONAL FOOD

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Lawrence *et al.* (1988) have demonstrated that boars maintained on a ration equivalent to 60% of their *ad libitum* intake will perform an operant response at a high rate for additional food. In contrast, boars fed *ad libitum* virtually stopped responding. A 60% ration is equivalent to about 1.3 times the ARC requirement for maintenance and low weight gain in boars. Thus, it appears that boars fed at maintenance levels are subject to a high and constant hunger. Clearly, this finding has important implications for the welfare of pigs.

An alternative, and perhaps more sensitive method of measuring hunger in the pig, is to manipulate the operant schedule rather than the daily food allowance. In this experiment hunger in the pig has been measured using a progressive fixed ratio (FR) schedule. Performance on progressive ratio schedules correlates well with variations in reward and deprivation parameters (Hodos, 1961). When the ratio of responses to reinforcements is too high the pig will stop responding and that point (extinction) can be used as a measure of the strength of feeding motivation.

Six sows (day 56-74 of gestation) were tested separately with operant conditioning apparatus in a laboratory at 21°C. Each sow was fed its normal restricted ration (2.3 kg) of commercial pellets (12.5 MJ DE/kg) at 0830 h. The sow was allowed access to a lever and additional feed trough from 0900-1700 h. Lifts on the lever on the FR schedule produced on average 2.68 g pellets as a reinforcement. Sows were trained to lift the lever on an initial schedule of 10 lifts/reinforcement. After 1 h of continuous responding (no pause greater than 5 min) the ratio was increased by 20 lifts. Thus the final schedule was 10, 30, 50, 70, responses/reinforcement. Extinction was deemed to occur if the sow did not respond for a 2 h period.

The six sows reached extinction ratios of 70, 130, 210, 210, 230 and 430. Reinforcement intakes prior to extinction were 15, 22, 17, 10, 11 and 3 reinforcement per h. On the assumption that the energy expended by a sow performing a repetitive operant response is equivalent to that expended by a sow performing stereotyped behaviour, it is possible to estimate the energy expenditure of the sows using the data of Cronin *et al.* (1986). Heat production by sows performing stereotyped behaviour at a high level was 11 kJ/kg $W^{0.75}$ /h. Net energy deficits prior to extinction were calculated to be 242, 45, 271, 75, 271 and 441 kJ/h. Clearly, four of the six sows were in energy deficit, and if they continued to respond at this rate they would have literally worked themselves to death.

In conclusion, these results suggest that sows on normal restricted rations are still extremely hungry, to the extent that they are prepared to sustain an energy deficit to gain additional food. The welfare implications of maintaining sows under conditions of feed deprivation need to be addressed.

References

- CRONIN, G.M., Van TARTWIJK, J.M.F.M., Van DER HEL, W. and VERSTEGEN, M.W.A. (1986). *Animal Production*. 42:257-268.
HODOS, W. (1961). *Science*. 134:943-944.
LAWRENCE, A.B., APPLEBY, M.C. and MACLEOD, H.A. (1988). *Animal Production*. 47:131-137.

TESTING MATERNAL BEHAVIOUR IN THE PIG: RESPONSES TO VISUAL AND TACTILE STIMULI FROM A MODEL PIGLET

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Pre-weaning piglet mortality has been estimated to range from 12-30%. Over 50% of deaths occur during the first three days after birth, with starvation and crushing accounting for 70-80% of the deaths (English and Wilkinson, 1982). The contribution of inadequate maternal behaviour to this mortality is unknown.

We are developing tests of maternal behaviour in sows with the goal of assessing whether sows are adapting to the intensive environment. We will measure variation in maternal responsiveness and general environmental responsiveness in a large number of sows and estimate the genetic component of this variation. Correlations with production parameters will enable us to predict whether these traits are changing in response to artificial and natural selection. Ultimately, we aim to determine the rate of adaptation to intensification, which may be the key to the problem of whether the welfare of confined pigs can be assumed to be guaranteed, as has been suggested by Beilharz (1982).

Maternal responsiveness was measured by testing the reaction of recently farrowed sows in the Mount Derrimut Piggery to a model piglet inserted underneath the udder as the sow knelt prior to lying down. The control model piglet was a 10x30 cm canvas cylinder filled with polyester fibre. Treatments tested which modified the tactile stimuli associated with the model were hardness (model filled with sponge, rubber, sand or concrete), texture (rough or smooth surface, and wet or dry) and temperature (hot or cold). Treatments which modified visual stimuli associated with the model were shape (small, large or lifelike) and test position (front, middle or rear of the sow). Sows were normally tested at 0830, 1030 and 1600 h on day 2 and 0830 h on day 3 post-partum, following normal feeding. Tests were done as replicated 4x4 Latin square designs (n=8 sows). Variables measured included attention to the model (scored according to the number of looks, nudges and grunts at the model), duration of kneeling and lying, and response to model removal (scored according to the positional changes made by the sow).

Model position was the only treatment which affected the response of the sow towards the model prior to lying. Sows were more attentive to models inserted at the front than the middle or rear ($P < 0.01$). None of the treatments had a significant effect on kneeling or lying duration, although there was wide variation between sows. None of the treatments affected sow response to the model on removal.

There was significant variation between sows in attentiveness to the models for the hardness and texture treatments and in kneeling duration for the shape treatments.

In conclusion, the lack of response to the models suggests that maternal responsiveness may have been modified by intensification, and the wide variation between sows suggests that there may be genetic variation in this trait.

References

- BEILHARZ, R.G. (1982). *International Journal for the Study of Animal Problems*. 3:117-124.
ENGLISH, P.R. and WILKINSON, V. (1982). In "Control of Pig Reproduction" pp. 479-506, eds. D.J.A. Cole and G.R. Foxcroft (Butterworth: London).

THE PRÉ-FARROWING BEHAVIOUR OF SOWS AND GILTS WITH ACCESS TO SPACE

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In intensive pig-farming systems it is common practice to confine sows in farrowing crates about a week before they are due to farrow. One or two days before farrowing the sows become very restless (Jones, 1966) and as parturition becomes imminent they may become destructive and bite and paw at the crate. This restless period corresponds to the time in which the free-ranging domestic sow leaves the herd and walks up to 6.5 km to find a suitable nest-site, construct a nest and farrow in it (Jensen, 1986). Concern has been voiced as to the welfare of the sow confined in the farrowing crate throughout this restless period. Indeed, confinement of animals at any time is one of the prime concerns of the welfare lobby and one of their major complaints about intensive systems. The issue of sows in farrowing crates needs to be assessed objectively. Is the sow suffering because she has been denied the space for walking and substrates for nest-building? To begin to answer this question it must be determined whether or not the pre-farrowing restlessness observed in sows in farrowing crates is manifested as increased locomotion as it is in free-ranging domestic sows. If so, we can then ask if there is any difference between primiparous and multiparous sows in the amount of locomotion shown due to prior experience of farrowing crates.

Pre-farrowing behaviour was observed in a 7x7 m test arena with a 2x2 m pen in one corner as the home pen. The sow was allowed access to the test arena each day for an 8 h observation period, from four days prior to the expected farrowing date until farrowing actually occurred. During the observation period a record was made at 1 min intervals of the behaviour performed by the pig and her position in the pen using a grid marked on the floor. Six multiparous sows and six gilts from the Mount Derrimut Pig Centre were used.

There was a significant increase in the distance travelled by both sows and gilts in an 8 h period prior to farrowing ($P < 0.001$) and no significant difference between any of the previous days. This 8 h locomotion period was separated from farrowing by a period of nest-building 6-8 h long. Parity did not significantly affect the distance travelled. The number of grid squares entered in both the home pen and test arena increased prior to farrowing ($P < 0.01$), with no effect of parity. All of the sows farrowed in the home pen, but three of the six gilts farrowed outside it. It appears that the increased restlessness shown by sows and gilts in farrowing crates prior to parturition is manifested as an increase in walking. Because there was no difference in the amount of locomotion shown by sows and gilts, it is possible that this behaviour is under innate control and may not be modifiable by experience. Now that we have established that intensively housed sows show an increase in locomotion prior to farrowing, we plan to determine the value that sows place on having access to a large space for locomotion at this time by imposing an operant requirement on entry to the test arena.

References

- JONES, J.E.T. (1966). *British Veterinary Journal*. **122**:420-426.
JENSEN, P. (1986). *Applied Animal Behaviour Science*. **16**:131-142.

A SYMPOSIUM - GENETIC SELECTION: WHICH WAY FORWARD?

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Introduction

When choosing boars and sows to breed the next generation, the pig farmer should select those that will produce the most profitable offspring. The performance of an animal's offspring define his or her breeding value, so the pig farmers' objective can be stated as "select the boars and sows with the highest breeding value for profit".

Profit is a property of the whole enterprise so the first step in carrying out this objective is to determine how profit is related to individual animal traits. The most important traits in determining profit are growth rate, food conversion efficiency, fatness and fertility.

Without a progeny test based on an infinite number of offspring we cannot know an animal's true breeding value. However, we can estimate or predict his breeding value from available information such as his own performance and that of his brothers and sisters. Thus, in practice, the pig farmer's objective becomes to estimate breeding values for important traits and use these to select boars and sows with the highest estimated breeding value for profit.

Estimated breeding values are thus central to selection decisions. Breeding values can be estimated in many ways, for instance by traditional visual appraisal of the animal or by measuring his own performance. The most accurate statistical method for estimating breeding values from a particular set of data is BLUP (best linear unbiased prediction). This method uses all available information to give a single "best" estimate of breeding value. In recent years it has been widely used in dairy and beef industries (Goddard *et al.*, 1988). In the first paper in this symposium the advantages and disadvantages of using BLUP in the pig industry are discussed. In the second paper the practical experience of a pig farmer in using a BLUP program in his piggery is discussed.

Regardless of the statistical method used for data analysis, an important issue in designing a breeding program is the choice of what data to collect. For instance although food conversion efficiency is important in determining profit, it may not be worthwhile to measure efficiency if breeding values for it can be predicted from growth rate and backfat data. As well as which traits to measure, one must also consider how to make the measurement (e.g. type of back fat probe) and under what conditions (e.g. restricted or *ad libitum* feeding). The criteria for making these choices (provided selection intensity and generation length are unaffected) should be to maximize the accuracy of estimated breeding values for profit and minimize costs of measurements and data analysis. For instance, if the heritability of growth rate is higher under restricted feeding than under *ad libitum* feeding, then data from restricted feeding allow more accurate estimation of breeding values. In the third paper the effect of selection for growth and efficiency under restricted or *ad libitum* feeding will be considered.

Estimating breeding values for litter size presents difficulties if you are restricted to using each animal's own performance as data; the heritability is low, boars produce no data and sows generate data only after they have been selected for breeding. In the fourth paper, two other sources of information/data on relatives and on traits correlated with litter size will be considered.

Symposium continued on next page

USE OF BLUP IN SELECTION FOR GROWTH RATE AND LITTER SIZE

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Introduction

Genetic improvement programs for pigs, in the past, have relied on comparing the performance of contemporary animals in a common environment or have made the assumption that the environment remained constant over time, when it was desired to compare records from animals from different test periods. It is clear, though, that a pig operation is a dynamic unit, where changes in phenotypic performance can occur due to changes in age of animals, season, feed source, feed composition, health status, housing, location within housing or personnel, and may not be due to changes in genetic merit. Biases in genetic evaluations arise when comparisons are made between animals from different generations or between animals raised in different time periods or management regimes, and these differences are not taken into account correctly. Comparisons of this type are critical in making selection and culling decisions for the genetic improvement of a pig herd or industry. Best linear unbiased prediction (BLUP) can provide a solution to many of these problems.

The BLUP method was originally developed in the USA by C.R. Henderson for the genetic evaluation of sires in the dairy industry to account for effects due to differences in management regimes and to selection on daughters' records. Currently, BLUP is being used routinely by the dairy, beef, horse, fish and poultry industries in a number of countries to enhance genetic improvement. A number of countries (Canada, US, UK and W. Germany) have developed or are developing systems that utilize BLUP in the genetic improvement of their respective pig industries. Why have so many industries adopted this newer method of predicting genetic merit?

Potential advantages

There are five advantages from using BLUP:

- (1) BLUP is a procedure which uses the information from all the relatives of an individual, thereby, providing a more accurate prediction of the genetic merit of that animal than other prediction methods available. This is especially helpful for lowly heritable traits, such as litter size;
- (2) As alluded to earlier, BLUP facilitates comparisons between animals producing records in different management regimes or over different periods of time, thereby increasing the potential for selection. Accounting for management groups or selection biases are very important advantages of using BLUP and are ones that other methods, such as classical selection index, tend to ignore. An animal raised in a hot, summer environment could have higher genetic merit for growth rate, for example, than one raised in the autumn, but its performance could be depressed such that its genetic superiority wasn't recognized, and it would be sent to slaughter;
- (3) BLUP facilitates comparisons of the genetic merit of animals with differing amounts of information, such as a sow with three litters versus a gilt that has yet to produce a litter. By accounting for parity, these comparisons can

result in selection and culling decisions that can advance genetic gain in a herd;

- (4) BLUP allows comparisons to be made among animals that have undergone different amounts of prior selection. This is important for the evaluation of males and females for reproductive performance;
- (5) BLUP partitions genetic and non-genetic effects on performance into their respective components, such that breeders can assess causes of change in mean herd performance over time. Suppose for example, during a 10-year period on a breeding farm, new farrowing crates were installed, the gestation ration was changed, a change in farrowing personnel had occurred, selection for litter size at birth had been practised and an improvement of +1.5 pigs born alive/sow was found in comparing year 1 to year 10. In assessing the selection program, how much of that change was due to the selection pressure put on litter size and how much was due to changes in the management of the sow herd? By using BLUP, genetic and environmental trends could be determined, and this is essential in evaluating a selection program and the investment in recording and breeding. No other evaluation system enables this to be done.

Several researchers have attempted to quantify the superiority of BLUP over other selection methods in expected genetic gain. Belonsky and Kennedy (1988) did a computer simulation of a closed 100-sow herd over a 10 year period to compare selection on phenotype versus selection on BLUP for traits with different heritabilities (h^2). For the simulation, values chosen were 10, 30 and 60%. They found that the relative advantages in using BLUP for selection over selection on phenotype were 55% for $h^2=0.10$, 25% for $h^2=0.30$ and 10% for $h^2=0.60$. This indicates that expected genetic gain from selection on BLUP was greater than expected genetic gain from selection on phenotype, but that the advantage decreased as heritability increased. They also found that, when estimated breeding values (EBVs) were used in both culling and selection decisions (culling when a replacement with a better EBV was available), genetic progress increased an additional 34-57%.

Long and Johnson (1988) used pig data from a Nebraska research herd to compare expected genetic gain in average daily gain (ADG) or litter size (LS) from selection on BLUP versus selection on phenotype. In this study, where estimates of h^2 were: 0.13 for ADG and 0.18 for LS, they found that BLUP resulted in an advantage in genetic gain for LS of 22% over selection on phenotype. The benefits of BLUP were even greater for ADG where BLUP exceeded selection on phenotype by 31%. Although the h^2 estimate for ADG in this study was lower than estimates found in other pig populations (Klassen *et al.* (1988), using Australian pig field data, found estimates ranging from 25-52%), this study by Long and Johnson does demonstrate the superiority of BLUP when dealing with traits having low heritabilities.

Klassen (1988) estimated what the increased genetic gain from using BLUP would mean in economic terms. Using Belonsky and Kennedy's data, he found that selection on BLUP EBVs would increase profits by \$16-27/sow/year over individual phenotypic selection. When BLUP EBVs were also used as the basis to cull parents when a better replacement was available, an additional profit of \$32-54/sow/year was found over selection on phenotype. These studies have demonstrated some of the potential advantages in genetic progress that could be obtained by using BLUP in a selection program.

There also are potential advantages to using BLUP that are somewhat tangent to increasing expected genetic gain, however, experience shows they are real. Since BLUP

requires information from all relatives to calculate EBVs, accurate pedigrees are required to be kept on the herd. This allows a breeder to better manage his rate of inbreeding than if he's only avoiding full and half-sib matings and has no record of ancestry over three generations or more. Also, since BLUP does require this additional information, many breeders when starting to use this mixed model technology (e.g. the BREEDPLAN system in the Australian beef industry and the STAGES program in the US swine industry), did a re-evaluation of how their record keeping system worked and were able to come up with more cost effective methods of recording on their farms.

Finally, because BLUP partitions herd performance into genetic and non-genetic effects, the environmental trend can be estimated. This can be very useful in assessing management decisions, predicting future production levels and budgeting the operation accordingly.

These are some of the possibilities that BLUP could provide but, since one might say that no bed of roses is without its thorns, what are some of the potential disadvantages of using BLUP in a selection program?

Potential disadvantages

The potential disadvantages of BLUP stem from the fact that relationships among all animals are used in the more powerful BLUP methods. The first potential disadvantage of BLUP involves the rate of accumulation of inbreeding in a closed herd breeding scheme. Belonsky and Kennedy (1988), in their simulation study, found that at year 10 average inbreeding in the BLUP selected populations ranged from 27-38%, while average inbreeding in the phenotypically selected populations ranged from 17-22%. They also found that inbreeding tended to increase with increasing h^2 for selection on phenotype but tended to decline, with increasing h^2 , for selection on BLUP. This is expected since with low heritabilities, using BLUP puts more weight on relatives' records and less on individual phenotype, causing related animals to have similar estimated breeding values and increasing the probability that relatives will be selected. This simulation study, however, did not address mate selection. Males and females were mated at random except that full-sib and parent-offspring matings were avoided. Also, only four boars were used for this 100-sow herd. This is a smaller effective population size than would normally be the case in a herd of this size, thereby increasing the rates of inbreeding for both phenotypic selection and BLUP. For either method of selection, rates of inbreeding can be kept at manageable levels, in the short term, through appropriate mating strategies and maintaining more than a minimum number of boars. In the longer term inbreeding can be reduced by strategic introduction of unrelated germplasm via artificial insemination (AI). Although the above study does indicate that BLUP has the potential for a higher rate of inbreeding than selection on phenotype, by managing the level of inbreeding, the advantages in genetic gain of using BLUP can be realised.

A second potential disadvantage with BLUP relates to the accuracy of the pedigrees used in the analysis. Since BLUP uses relationships among all animals, what happens when those pedigrees are wrong, as can happen due to errors in recording mating or cross-fostering events, or errors in transferring data into the computer? Long and Johnson (1988) addressed this issue by comparing expected genetic gain in ADG or LS from selection on phenotype versus selection on BLUP when parents were either correctly or incorrectly assigned to animals. Errors in pedigree do not affect selection on phenotype since parentage doesn't affect the selection criterion, but when sire and dam were incorrect for 20% of the pigs each generation, genetic gain using BLUP for selection was reduced by 9.3% for LS and 12.4% for ADG. However, these genetic gains using BLUP were still 10.5% for LS and 14.6% for ADG better than selection on phenotype. If pig pedigrees in a herd are less accurate than the 20% error level

investigated above, there might be a point where using BLUP on incorrect data would be inferior to selection on phenotype. It must be stressed that accuracy in recording pedigrees is essential to reap maximum benefit from an EBV method such as BLUP. Recording procedures and checks can be implemented which achieve this.

The last potential disadvantage of using BLUP is that it is computationally more demanding than other selection methods, but this problem has been addressed by animal breeders at The Animal Genetics and Breeding Unit, Armidale, and software (PIGBLUP), is now available to take care of this problem (Graser and Klassen, 1988).

User mistakes

Having discussed some of the potential advantages and disadvantages of using BLUP in a breeding program, what are some of the mistakes that users of BLUP might make in applying this methodology? The first mistake was mentioned earlier. If a user makes his selections using BLUP and does not have a very good mate selection program (e.g. only avoids parent-offspring or full-sib matings), that user could experience an increase in the rate of inbreeding in his herd. This mistake can be avoided by adopting mating structures designed to minimize the rate of inbreeding. Another common mistake has to do with selection of data going into the BLUP analysis. At times, breeders will throw away information on poor performing animals, thinking it was of little use since those animals did poorly or that it was making the rest of the herd "look bad". If a user deletes poor performing animals prior to analysis, he can reduce the potential genetic gain he could have made if all the data were included because, with the use of BLUP, some of the information on the remaining animals has been deleted. Long and Johnson (1988) evaluated the effects of deletion of data on expected genetic gain using BLUP and found that genetic gain/generation using BLUP was reduced by 5.4% for LS and 7.5% for ADG when 20% of the poorest performing animals were deleted prior to analysis. With this deletion, BLUP still out-performed selection on phenotype by 15.3% for LS and 21% for ADG, but throwing out data did reduce expected genetic gain using BLUP.

Another potential mistake for users of BLUP has to do with the replacement of animals. Occasionally, due to genetic drift, a boar is produced with a breeding value far above any other animal in the herd. To enhance short term gains, a breeder might retain this boar longer than normal to obtain as many progeny from him as possible. In the long term, however, this can lead to higher levels of inbreeding in the herd and potential loss of this genetic gain due to the effects of inbreeding depression. This can be managed by judicious use of this type of animal, when they arise, as well as a good mate selection strategy.

Future issues

Given that the preceding discussion suggests that BLUP is the method of choice for a pig selection program, what are some of the emerging issues for the future use of BLUP for the Australian pig industry? The first issue to be addressed is to finalize what further production traits should be included in the BLUP analysis? Currently, PIGBLUP analyses growth rate, backfat and litter size, and combines these into a breeder customised economic index. Are breeders keeping records on other economically important traits, such as carcass or feed intake, that need to be included in these genetic evaluations? Some suggestions were obtained from the workshop held in Armidale last year, (Implementing New Genetic Evaluation Procedures for the Pig Industry, February, 1988). Optimization of mating structures to balance the effects of selection and inbreeding should be considered. Since BLUP uses relationships among all animals, this provides an opportunity to develop schemes that find the optimum

balance between the rate of inbreeding and genetic gain. In a recent paper, Webb and Bampton (1988) have recognized this and suggested potential strategies such as increased number of families or a different population structure than has traditionally been in place. Is across herd analysis desired? BLUP can account for herd effects and, therefore across herd analyses can be performed if appropriate ties exist between herds, i.e. related animals exist in two or more herds through either AI or exchange of animals. To optimize this type of analysis an AI reference sire scheme could be developed. Questions to be answered would include: How would reference sires be chosen, and how would those sires be disseminated through herds desiring an across herd genetic evaluation? Could central test station data be used in this type of analysis? If it were, a re-evaluation of how animals were sampled for central testing might be in order to optimize the use of these type of data in an across herd analysis. Finally, collection of more economic information on production costs involved in a pig operation are needed to further extend the economic indexing system developed by Stewart *et al.* (1988) and incorporated in PIGBLUP.

These are just a few of the issues that need to be addressed for the use of BLUP in the future. BLUP is not a "magical procedure" but makes use of proven biological and statistical principles to produce the best prediction of an animal's breeding value currently possible. Given developments in other countries, it behoves Australian pig breeders to use the best animal breeding technology available to ensure that the Australian pig industry remains competitive in the 21st century.

PRACTICAL EXPERIENCE WITH PIGBLUP

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The introduction of BLUP procedures to the Australian Pig Industry is exciting to me as a pure bred breeder endeavouring to improve my stock to meet the requirements of a changing industry.

Those contemplating the use of PIGBLUP on their farm must first accept that they are not going to fully understand the complexities of the PIGBLUP program. First there are a lot of new terms that you must become conversant with, e.g. estimated breeding values (EBVs) for the traits of growth rate, back fat and litter size, which we generally use in selection of replacement breeding stock. Then we are confronted with results labelled genetic trends which have been split up into several categories. There are trends for production which have been very conveniently divided into two areas, genetic and environmental. Also we have the same results for reproduction.

It soon becomes obvious that PIGBLUP is a lot more complicated than any previous form of improvement program. Equally obvious is the realization that as producers we are not going to understand the complex mathematics which make PIGBLUP work. Hence we must believe in the principles of BLUP and accept that science has presented us with a powerful new tool which has the capability of improving the efficiency of selection and improving the rate of genetic improvement. This point cannot be over stressed, because if we cannot accept the principles involved and believe that they will assist in improving your breeding program PIGBLUP will not work effectively for you.

The next step is equally important. We as users must accept that the scientists at The Animal Genetics and Breeding Unit (AGBU) who have done the development work have got their sums right. We must assume that the parameters of heritability that they have used are correct. I am assured that every possible effort was made to ensure that we have the most accurate assessment possible. We as producers must trust the scientists who have developed the PIGBLUP program and have a continuing close liaison with them.

At the time of accepting the invitation to present this paper I believed that the program would be commercially available at the time of writing this paper. Unfortunately this has not eventuated as every thing is in a state of "limbo" while the necessary contractual arrangements with the commercialization are being completed. My experience is therefore based on the developmental phase of PIGBLUP.

Our first experience was that our computer records were not as good as we believed. As PIGBLUP has the capability of processing information over a 10-year period, it is essential that each animal has its own unique number. The common practice of reusing numbers after a suitable time lapse creates chaos with the program. You realise something is wrong when the exclusion report says that the sow has farrowed before she was born! A year identifier is probably the best method of over coming this problem. It is also necessary to have a means of identifying animals foreign to the farm which will ensure that their records are not confused with those produced on the farm. We prefix all introduced animal numbers with a # and a letter designating the farm of origin, e.g. #A1234.

It very quickly becomes apparent that accuracy of recording enters a new dimension as any errors will effect the EBVs of that animal and also of its relatives. Therefore an extremely high level of accuracy is required if we wish to have accurate results.

The PIGBLUP program has the capacity to generate a massive quantity of reports. If we assume we have a typical 100 sow herd and records for the maximum 10 years, we can anticipate a text file of records in the order of 1-1.5 m bytes in size containing up to 25,000 records. This will be made up with three types of records for each animal. First, an animal record which contains identification number, breed, sire, dam, date of birth, size of litter and number of litters. Second, a production record which contains identity, growth rate and back fat. Third, the animals litter records which are sorted by parity number and contain identity, parity number, mating date, service sire identity, farrowing date, piglets born alive and dead.

Whenever additional information is added and processed all EBVs are updated and can be printed. If all animals with a record which have been in the herd over the period are included along with the animals available for selection as replacement or sale are printed regularly, then the piles of reports seem to accumulate. Hence I believe careful management of the printing of reports will be required to ensure that they are used effectively.

Having produced accurate EBVs for the animals available for selection, we are faced with the problem of how to decide on the relative importance of growth rate, back fat and litter size. Incorporating this information into a single index is logically the next step.

Indexes are generally fixed and don't cater for individual requirements of our herd. Professor Terry Stewart has developed a customers index which collates all the EBVs into a single figure expressed in dollars/litter. This index will enable a breeder to concentrate his selections in a particular direction, e.g. development of a prolific female line with emphasis on mothering ability. This will mean every farm could be selecting with different emphasis on the traits included in the index. These ideally would be related to the market requirements of the particular area.

If we look at the inputs required for this index on the farm as listed in Table 1, the advantages and flexibility will become apparent. The index has default values but a farm can input its own values. It is not recommended that these be changed regularly as this could effect genetic progress. So the values used should be estimated production costs and returns over a two-year period if possible.

The ability to input the premiums (if any) and penalties on the market prices which apply in your area of selling are of great benefit. Likewise the input of production costs and performance data pertaining to a particular piggery will maximise the effectiveness of the index. Also the development of specialised lines will be possible by manipulating the percentages of terminal or maternal sires and replacement gilts.

This customizing of an index for a particular farm is possibly the greatest advance to date in our endeavour to make good genetic progress. The disadvantage will be that purchasers of stock will find it more difficult to compare stock on different farms.

In conclusion I believe we are entering a new era of genetic improvement as science and computers give breeders more accurate information on which to base their selections. However the old practices of selecting for soundness, conformation and temperament will still be important and should not be overlooked. I was told as a young breeder by our local veterinarian and I quote "Breeders have a responsibility to pass on to the next generation sound stock, free of genetic deformities."

Table 1. Pig index economic data

Base carcass market price (\$/kg)	2.15
Premium for P ₂ fat class 1 (8 mm or less) (\$)	0.00
Premium for P ₂ fat class 2 (9-16 mm) (\$)	0.00
Premium for P ₂ fat class 3 (17-19 mm) (\$)	-0.12
Premium for P ₂ fat class 4 (20-24 mm) (\$)	-0.24
Premium for P ₂ fat class 5 (25+ mm) (\$)	-0.40
Cost for feed in feeder (\$/kg)	0.27
Cost for day labour and housing (\$/pig/day)	0.25
Number of pigs born alive/litter	9.9
Pre-weaning mortality (%)	14.0
Post-weaning mortality (%)	3.0
Average daily gain to market weight (g/day)	560
Mean P ₂ fat depth (mm)	12
Is feed restricted in grow/finish to reduce P ₂ fat?	No
Feed conversion, (kg feed/kg live weight)	3.2
Target market carcass weight (kg)	68
Dressing per cent (%)	76
Boars sold (or used) as terminal sires (%)	5
Boars sold (or used) as maternal sires (%)	5
Boars sold (or used) as slaughter boars (%)	80
Gilts sold (or used) as replacement gilts (%)	10
Gilts sold (or used) as slaughter gilts (%)	90

Symposium continued on next page

PERFORMANCE TESTING AND SELECTION FOR EFFICIENT LEAN GROWTH

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Selection versus production environments

Increasingly, the selection of pig breeding stock takes place in an environment removed from production. Selection is carried out in central testing stations or seed stock producing herds while production takes place on farms. There is growing concern that the best genotypes selected in the testing environment may not be the best for the production environment. From Falconer (1985), a desirable testing environment is one in which $rh_t/h_p > 1$ where h_t^2 is the heritability of a trait (for example growth rate) measured in the testing environment, h_p^2 is the heritability of the same trait in the production environment and r is the genetic correlation between the expressions of the trait in the two environments. One of the arguments for separating the testing from the production environment is that h_t^2 can be made greater than h_p^2 and $r=1$.

There are a number of studies which attempted to estimate r by comparing the performance of boars in a central testing station with the performance of their offspring on the farm. The latest of these studies which includes a review of previous work is that of Merks (1988). Generally r has been found to be lower than expected, particularly for growth traits. He attributes this to true sire x environment interaction but also to deficiencies in the studies. One component of the environment likely to differ between the selection and production sectors is the method of feeding. The following experiments of ours provide some information in this area.

Alternative pathways of improvement

A common breeding objective and a major determinant of profit in pig production is the efficiency with which food is converted to the growth of lean tissue. Physiological models have been constructed for pigs (Whittemore and Fawcett, 1976) which suggest pathways along which genetic improvement of this trait could be made. Two of these are a reduction in food intake to limit the amount converted to fat tissue, and a diversion of part of the food consumed away from fat and toward lean deposition.

Genetic improvement of the efficiency of lean growth could follow one or more pathways. The extent to which each responds depends on the performance testing conditions under which efficiency is measured, its heritability and its variability.

Selection for growth on liberal feeding

The traditional method of performance testing pigs for selection for efficient lean growth has been over a fixed weight range, e.g. 25-90 kg, under conditions of liberal feeding which permit individuals to express their appetites. To investigate the effect of this type of selection, McPhee (1981) established a selected-line and a genetically stable control-line from the same base herd. In the selected-line, pigs were fed liberally during performance testing from 25-85 kg live weight and selected for efficient lean growth.

After five generations, pigs from the selected-line were compared with pigs from the control-line on *ad libitum* feeding to measure genetic response. The results are given in Table 2.

Table 2. Comparison of selected and control pigs on *ad libitum* feeding after five generations of selection on liberal feeding

	Growth rate (kg/day)	Food conversion ratio	P ₂ fat (mm)	Food intake (kg/day)
Selected	0.78	2.67 ^x	18.7 ^x	2.07 ^x
Control	0.81	2.78 ^y	21.8 ^y	2.24 ^y

^{x,y}differ at P<0.01

Although there was no apparent improvement in growth rate, there was a considerable decline in fat, and in daily food intake. This clearly shows that the reduced appetite path to improved efficiency of lean growth was followed by the selection process.

The conditions of liberal feeding during performance testing and emphasis on efficiency and leanness used in this experiment mirror those used in central testing stations throughout the world and similar trends in lowered appetite have been observed in some pig populations using these stations. The Danish Landrace is a prime example of this. It is a breed with a low appetite, an extremely lean carcass and only a moderate growth rate. It is now widely felt that this change to appetite is undesirable.

We need to approach a point where efficiency is highest on *ad libitum* feeding then increase appetite further. On the way to this goal efficiency on commercial farms can be kept at a high level by restricted feeding practices. Good appetite is also a desirable attribute of lactating sows and may be important if growth hormones, which depress appetite, become widely used in pig production.

Selection for growth on restricted feeding

Clearly we need to follow some other pathway to improve lean growth efficiency. A change in the way food energy is partitioned in favour of more lean and less fat deposition seems a promising approach. With this aim, Kielanowski (1968) suggested selecting pigs for lean tissue growth rate after a performance test of set duration and food intake. He expected animals to be favoured which grew quickly because they favoured lean over fat deposition, the former tissue having a lower energy cost than the latter. It was difficult to predict the effect on appetite of this form of selection.

After some encouraging results in simulation studies with mice (McPhee and Trappett, 1987), a pig selection experiment was set up to test this hypothesis. A full account of this experiment is given by MCPhee *et al.* (1988). Briefly, from a common base herd two genetically similar lines, a control and a selected-line, were established. Pigs in the selected-line were performance tested over a 12-week period commencing at 25 kg live weight. During the test all pigs were fed the same amount of food. At the end of the test, the weight of lean in the ham was estimated from live weight and ultra-sonic fat depth measurements. This has been shown to be highly correlated with total lean in the carcass (Evans and Kempster, 1979). Those with the highest estimated lean in their hams were selected as parents. Selection proceeded in this way for 5 generations. In the latter part of the trial, litters were split and tested on both the test feeding scale and on *ad libitum* feeding and this permitted the estimation of genetic parameters on both levels of feeding. They are given in Table 3.

Standard errors are high but the tendency is for higher heritabilities for all traits on scale than on *ad libitum* feeding, a negative genetic correlation between growth rate and fat on scale feeding compared with a positive correlation between these traits on *ad libitum* feeding. This conforms with Kielanowski's expectation that, in the absence of appetite variation, the fastest growing pigs will be those which convert the available food into lean rather than fat.

Table 3. Statistics of traits on two levels of feeding (SE in parentheses)

	Scale	Heritability (<i>ad libitum</i>)
Growth rate	0.41 (0.15)	0.28 (0.19)
Fat	0.60 (0.17)	0.34 (0.19)
Ham lean	0.43 (0.15)	0.28 (0.19)
		Genetic correlation
Growth rate x fat	-0.22 (0.20)	0.35 (0.35)
Growth rate x ham lean	0.93 (0.03)	0.99 (0.01)
Fat x ham lean	-0.55 (0.15)	0.34 (0.34)

Responses in the selected-line, measured from the difference between its performance and that of the control-line on scale and *ad libitum* feeding in the final stages of the experiment, are shown in Table 4.

Table 4. Response to five generations of selection on restricted feed measured as the difference between selected and control line means on scale and *ad libitum* feeding

Feeding scale Trait	Significance of effect (P<)		Line	Line x feed
	Scale	<i>Ad libitum</i>		
Growth rate (g/day)	82	160	0.01	0.01
Food intake (g/day)	-	150	0.01	0.01
Food conversion ratio	-0.16	-0.27	0.01	0.01
Fat (mm)	-2.25	-2.51	0.01	ns
Ham lean (kg)	0.47	1.01	0.01	0.01

For growth rate, food conversion ratio and ham lean weight, there were considerably greater responses on *ad libitum* than on scale feeding. For fat, the response was about the same on both levels of feeding. As predicted, the rate of growth of lean relative to that of fat has been increased. The most interesting observation was the rise in voluntary food intake of the selected-line despite the absence of direct selection for this trait.

Practical relevance

These comparisons between the feeding regimes of estimates of heritability, correlation and selection response suggest that a feeding scale which limits appetite expression may be a more suitable performance testing regimen than *ad libitum* feeding for the selection of pig breeding stock whose descendants are grown to slaughter on *ad libitum* feeding.

This poses practical difficulties for on-farm performance testing and selection schemes. Under commercial conditions, pigs are fed in pen groups and variation in individual food intakes can be quite large. The elimination of appetite variation during growth necessitates the provision of individual feeding facilities, a luxury usually afforded only by specialised seed stock producers or performance testing stations.

There are known to be variations in feeding practices in pig herds and the results of this study suggest that these could cause variations in heritabilities and genetic correlations of traits under selection. This could reduce the effectiveness of industry wide genetic improvement programmes based on BLUP procedures (e.g. PIGBLUP) which assume set values for heritabilities. Any loss of efficiency from this source would be small however when compared with the effect of introducing animals of unknown

breeding value (Keele *et al.*, 1988). A glance at the parentage of pigs born in Australian stud herds shows a very high rate of introduction of breeding stock from other herds and overseas. Under these circumstances and considering the reasonably high heritability of lean growth and efficiency traits, BLUP procedures may lose their theoretical advantage of 15-20% over simple phenotypic selection as used in the present study.

Symposium continued on next page

METHODS AND SUCCESS OF SELECTION FOR LITTER SIZE

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Introduction

One extra piglet weaned/litter is worth approximately \$75/sow unit if grower shed space was fully utilized previously, or \$150/sow unit if grower shed space is not a limiting factor (author's estimates, based on 1987 production costs). These estimates place the importance of litter size in some financial context. The benefits are less if the production schedule has already allowed for the current litter size. About half the value from improving litter size comes from reducing the overhead cost of maintaining sows, and half through an increase in the volume of production.

In the past more attention has been given to methods other than selection to improve litter size (i.e. crossbreeding and selection between breeds, avoiding inbreeding, nutrition and mating management, and piglet care) because these methods were seen to have a greater impact.

It is an appropriate time to review whether to include litter size as a selection criterion, particularly given two recent developments; computer software based on BLUP technology which increases the scope to use family information, and improvements in carcass quality which means that less selection pressure could be warranted in this area (with selection pressure diverted to litter size).

Predicted selection response

Quantitative genetic theory predicts the annual response to selection (R) to be:

$$R = i\sigma r h^2/L$$

where i is the selection intensity, σ the phenotypic standard deviation, r the relationship between the selected individual and the individual on whose record selection was based (e.g. $r=1/2$ between daughter and dam), and h^2 is the heritability of the trait.

The heritability of litter size is low compared to growth and carcass traits. A review by Legault (1970) of 24 different publications shows estimates as high as 0.59, with a mean estimate of 0.10. The two largest studies, Strang and King (1970) and Legault (1970) gave heritability estimates of 0.07 ± 0.02 and 0.086 ± 0.035 , respectively. Heritability estimates are strictly applicable only to the population used to derive the estimates. Nevertheless it is worth noting that those studies giving heritability estimates considerably higher than 0.10 were generally based on relatively few animals and/or were accompanied by large standard errors. American estimates generally come in slightly higher than European estimates.

Some attempts have been made to give reasons for the low heritability of litter size. The most common explanation being that litter size has long been subjected to natural selection and as a consequence the genetic variation has been reduced.

Falconer (1960) suggested that with mice a negative environmental correlation exists between a daughter and her dam's litter size. That is, mice reared in large litters produced smaller litters. Revelle and Robinson (1973), in an attempt to distinguish between possible genetic and maternal effects in gilt litters, compared heritability estimates based on daughter-dam regression (0.13 ± 0.06 , 750 pairs) and grand-daughter

grand-dam regression (0.28 ± 0.26 , 539 pairs). In view of the standard errors, the results were not conclusive though the mean estimates provide some evidence of a negative maternal effect. Nelson and Robinson (1973) used cross fostering to establish the effect of being reared in a small (six pigs) or large litter (14 pigs). More pigs (1.8) were born alive to pigs reared in small litters, although there were insufficient data in this study for an adequate statistical test.

Half-sib analysis of data would not be biased by maternal effects, as is the case for daughter-dam regression. Smith and Strang (1979) re-analyzed the data of Strang and King (1970) using half-sib analysis. The resultant heritability estimates were no higher using half-sib analysis, than daughter-dam regression.

The evidence for a substantial maternal effect on litter size is not conclusive. Given also that most of the selection response comes through the selection of males, the issue seems minor. However, there is still merit in cross fostering piglets to standardize litter size in the nucleus for a number of reasons, including eliminating the possible maternal effects on growth and carcass traits as well as litter size.

Litter size is a very variable trait (coefficient of variation of 25-28%) which compensates for the low heritability. The fact that litter size is a sex limited trait and that selection is based on parental performance reduces further the potential for a selection response.

Realized selection response

The predicted response, using the previous equation, assuming 1 in 20 selection on males ($i_m=1.867$), 1 in 4 in females ($i_f=1.214$), h^2 of 10%, σ of 2.5 and L of 1 year, is 0.19 pigs/litter/year. The predicted response looks reasonably good. The more relevant point is whether it can be achieved in practice.

Only one experiment involving a selection line for litter size and a control line, has been reported (Ollivier and Bolet, 1981). The results of 10 generations have been published. The observed response over the first five generations was 0.15 pigs/litter/generation. The trend did not continue over the subsequent generations, with the trend over the whole experiment of 10 generations showing no response.

Among the reasons for the lack of response in this experiment are a failure to achieve a high selection intensity ($i_m+i_f=1.1$) and a realized heritability of only 2%.

Selection options

The remainder of this paper is given to discussing selection methods, within a breed or population, which may give a greater response than simple mass selection.

Repeated records

Basing selection on two or more litter records will increase the effective heritability (by $n/(1+t(n-1))$ where n is the number of records and t is the repeatability). However σ is reduced (by the square root of $(1+t(n-1))/n$) and the generation length is increased. On balance the extra benefits from using repeated records is very modest, with the greatest response coming from using the first two parity records (Table 5).

Information on relatives

The idea of using family information to increase the response of traits of low heritability, is not new. However until the development of the BLUP technology, the method has not been a realistic alternative.

Estimates of the increase in selection accuracy by using family information has been published by Avalos and Smith (1987). The authors considered a number of situations. In the example where all available information was used, and a heritability

of 10% was assumed, the accuracy of selection was increased by 71%. This brings the response to selection from 0.19 to 0.33 pigs/litter/year. Avalos and Smith assume higher selection intensities than in the above calculations and in fact they estimate a response of 0.59 pigs/litter/year in one particular situation.

Table 5. Value of repeated records

n	h^2	σ	L	Response
1	0.10	2.5	1	0.193
2	0.172	1.89	1.25	0.201
3	0.231	1.65	1.5	0.195

(based on a repeatability of 0.15)

Some consideration needs to be given to the implication regarding inbreeding. Under simple mass selection the rate of inbreeding increases slightly with the heritability of the trait under selection. But with family selection, more emphasis is given to information on relatives if the heritability is low. Consequently the rate of inbreeding is increased substantially when selecting on family information for traits of low heritability - the very traits likely to be affected most by inbreeding.

Some restriction on male selections may be necessary to reduce inbreeding rates. Any restrictions will reduce the selection response. Avalos and Smith estimated a 13% loss in response if a maximum of one male was selected/litter, and a 32% loss if using a maximum of one male/sire group. Toro *et al.* (1988) suggest that the inbreeding depression that results with no restriction on sire selections will over compensate for the extra response in litter size.

Goddard (personal communication) has suggested that the final selection decision should take account of a boar's estimated breeding value and his average relationship to the rest of the herd. Boars closely related to other boars and sows would be penalized and this tends to increase the number of boars selected and reduce inbreeding.

The significance of the inbreeding issue will depend also on the degree of selection placed on litter size relative to the growth and carcass traits, and the crossbreeding program following on from the nucleus lines. More precise guidelines are needed if breeders are to use family information to select for litter size.

Hyperprolific sow lines

Legault and Gruand (1976) proposed that a selection response of one pig/litter could be achieved by applying a very high selection intensity by screening a larger population than the nucleus herd itself. The system involves an upgrading process relying on crossing prolific dams with sons of prolific dams. The principal has been applied successfully in commercial breeding companies to achieve a gain of one pig/litter.

The method provides a "one generation" response. It is a useful starting point for a breeder wishing to improve litter size by selection, but is less useful as a basis for an ongoing selection program because it relies on using accumulated records. The generation length would need to be extended too far in the second and subsequent generations to give time to collect the litter records on the next generation of dams.

Practical considerations are that the screening should be limited to populations that are not significantly inferior in other production traits or in health status, to the nucleus herd. For these reasons the concept has been applied most successfully by breeding companies that limit the screening process to their multiplier units only.

Progeny testing

Leukkunen (1984) considered the progeny testing of AI boars on the basis of their daughters' farrowing results. Assuming the same selection intensity as with family selection, Avalos and Smith (1987) estimated that the rates of response would generally be lower than with family selection. They also noted that the population size needed to sustain the progeny testing scheme would be 5000-15,000 sows/sire selected.

Indirect selection

The prospect of selecting for ovulation rate to increase litter size has received some attention. Of special note is the work undertaken by Zimmerman and Cunningham (1975) who selected for ovulation rate over five generations. A response of 0.25 ova shed/generation was achieved, relative to the control line, which represented a realized heritability of 0.52. Although a substantial response in ovulation rate was achieved, there was no correlated response in litter size (England *et al.*, 1977)

Many aspects of reproductive activity in both sexes may be under similar genetic control so it may be possible to assess the potential prolificacy of an individual from hormone levels or the product of hormones.

It is worth noting that various testes measurements appear to be moderately heritable (Toelle *et al.*, 1984). Proud (1976) reported that selection for ovulation rate resulted in a correlated response in the rate of testicular growth in boars. Bates *et al.* (1986) reported that genetic correlations among litter mates indicated that selection for decreased age of maturity in gilts would increase luteinizing hormone concentration in boar offspring. Hemsworth (personal communication) has found that the number of copulations by a boar in a mating test is significantly correlated with the litter size of their daughters at their first farrowing. Hemsworth has also found a significant correlation between testicle size of boars and the receptivity of their daughter at mating.

These papers confirm a connection between aspect of reproduction in both sexes though there is no indications at this stage that there are any useful methods of indirect selection for sires which can be justified in practice.

Hennessy (personal communication) has preliminary results indicating a correlation between adrenal response and growth and feed conversion and to a lesser extent with reproduction.

Overall prolificacy

The major purpose of this paper is to focus discussion on the opportunities to improve litter size by selection. More appropriate measures of overall prolificacy need to account for the time interval between litters and breeding performance over the lifespan of stock. Selection criterion such as pigs produced/age or time unit is more encompassing than litter size. Age at puberty is the only sow performance trait that is reasonably heritable (0.53 ± 0.13 , Young *et al.* {1978}).

Net economic performance

Methods of improving production and reproduction traits simultaneously have been considered by Smith (1964). Morris (1975) in applying the theory developed by Smith, calculated the inclusion of reproductive performance with production traits would increase the rate of genetic improvement by only 0.55%. Moav and Hill (1966) using profit equations concluded that the average commercial standard of reproductive performance may be sufficiently high that selection for production traits alone is almost as efficient as using an optimum index. They point out that as herd mean performance increases, the marginal value of further improvements decrease. Clark and Smith (1979) concluded that selection within specialized sire and dam lines offered no practical advantage to the UK pig industry. However in the US pig industry, where higher fixed reproductive costs, lower feed costs, lower average litter size, and higher

heritability estimates are reported, they suggest that selection within specialized lines becomes more worthwhile. More recently Avalos and Smith (1987) calculated that with family selection, including litter size along with growth and carcass traits improved the economic response by only 5% in general purpose lines and 18% in specialized lines.

On present information it appears the best opportunity to improve litter size by selection is by utilizing family records (using BLUP technology) in specialized dam lines. The economic benefits from using this procedure are useful if not dramatic. However these benefits will increase as the marginal returns from reducing backfat further declines. Developments in use of pig growth hormones, for example, may give substantial gains in carcass quality. This in turn will increase the incentive to divert selection pressure to reproductive traits.

SYMPOSIUM CONCLUSION

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A person running a pig breeding program is faced with three types of problems:

- (1) what data to collect;
- (2) how to analyze this data to estimate breeding values for profit;
- (3) how to utilize the animals selected on their EBVs in the breeding program.

The introduction of BLUP methodology for calculating EBVs provides a useful framework within which to discuss these three problems, but it certainly doesn't represent a solution of them in itself. The main benefit of BLUP methodology for computing EBVs is that, it allows all information to be condensed into a single "best" estimate. The ultimate extension of this principle would be to produce only a single EBV for "profit". However, farmers vary in the importance they attach to individual traits in determining profit. Therefore a better approach is to produce EBVs for traits which directly affect profit and which have the same biological meaning from farm to farm e.g. growth rate. The farmer can then apply his own economic weights to these EBVs in order to produce his own EBV for profit.

Assigning economic weights in a logical manner is not easy. Therefore the procedure to help farmers do this developed at AGBU by Stewart and described in the second paper in this symposium appears to be very useful and worthwhile.

There are some traits which are not economically important themselves but which may be useful indicators for other traits. For instance data on testis size may help in estimating breeding value for litter size (fourth paper in the symposium). A multi-trait BLUP can incorporate this information into the EBVs for the important trait. If this is done there is little point in producing EBVs for the indicator trait since it has no direct affect on profit.

Decisions concerning which traits to record can be approached by their effect on the accuracy of EBVs for profit and on generation interval, as was done in the fourth symposium paper. This applied both to traits which directly affect profit (e.g. piglets/sow/year) and to traits which are only useful as aids in estimating breeding value for another traits (e.g. testis size). Thus we can consider whether it would be better to have EBVs for piglets/sow/year instead of litter size and whether this should take account of data on testes size.

A multi-trait approach can also help us to deal with the problems raised in the third paper in the symposium. For instance, growth rate under restricted and *ad libitum* feeding can be regarded as two different but correlated traits. The approach is then to decide which traits are of direct economic value (e.g. growth rate under restricted feeding or perhaps food conversion ratio) and therefore should have EBVs calculated for them and what data to collect in order to maximize the accuracy of these EBVs.

In order to answer these questions and to carry out the multivariate BLUP analysis good estimates of genetic parameters, such as heritabilities and genetic correlations, are required.

Although BLUP is a powerful technique for estimating breeding values it cannot perform magic or extract good information from bad data. For instance, if one pig(s) is given preferential treatment then his EBV will be biased and this will affect the EBVs of all his relatives as well. This might happen unwittingly if a new line of pigs is given extra human attention or if two halves of a shed have different micro-climates.

Proper designation of management groups is vital to producing unbiased EBVs.

Estimates of breeding value, such as a simple performance test, which do not allow comparison of boars or sows in different groups tend to produce rather rigid breeding programs. For instance, the number of boars and sows to be selected from each batch and the maximum time to keep them in the breeding herd are all fixed in advance. BLUP estimates of breeding value allow a more flexible approach in which the policy is simply to select the best regardless of where they come from. For instance an exceptionally good boar can be retained in the herd for longer than usual; as information on relatives accumulates a sow may be culled where otherwise she would have been retained; a purchased boar can be compared with home boars.

However, the older, rigid breeding programs took into account inbreeding in determining their optimum design. A policy of simply selecting the boars and sows with the highest EBVs amongst all those available does not. Thus inbreeding rates could rise as indicated in the first paper in the symposium. I doubt if mate selection alone will overcome this problem and it may erode a considerable part of the benefits of more accurate BLUP EBVs.

In dairy breeding programs Goddard (1985) found that simply selecting the bulls with the best EBV did not lead to a good compromise between inbreeding and selection intensity. What is needed is a flexible mechanism to combine EBVs and inbreeding when selection decisions are made. (In the fourth paper reference was made to a suggestion of mine in this direction).

The ability of BLUP to condense all information into a single EBV for each trait also simplifies the selection process. In the absence of such an EBV the farmer must try to combine information himself; for instance to compare two boars, one with a superior performance test but inferior pedigree to the other. It is unlikely that the farmer will weight all sources of information optimally in making his final decision. Thus even if BLUP EBVs are only slightly more accurate than a performance test alone, they will probably lead to better selection decisions.

References

- AVALOS, E., and SMITH, C. (1987). Genetic improvement of litter size in pigs. *Animal Production*. 44:153-164.
- BATES, R.O., BUCHANAN, D.S., JOHNSON, R.K., WETTERMAN, R.P., FENT, R.W., and HUTCHENS, L.K. (1986). Genetic parameter estimates for reproductive traits of male and female littermate swine. *Journal of Animal Science*. 63:377-385.
- BELONSKY, G.M. and KENNEDY, B.W.(1988). Selection on individual phenotype and best linear unbiased predictor of breeding value in a closed swine herd. *Journal of Animal Science*. 66:1124-1131.
- CLARK, P.K., and SMITH, C. (1979). Profit equations and specialized lines for pigs. *British Society of Animal Production* (Winter Meeting 1979), paper number 32.
- ENGLAND, M.E., YOUNG, L.D., CUNNINGHAM, P.J., and ZIMMERMAN, D.R. (1977). Ovulation rate in swine. Correlated response in litter traits. Abstracts of 69th Annual Meeting of the American Society of Animal Science, p. 18.
- EVANS, D.G. and KEMPSTER, A.J. (1979). A comparison of different predictors of the lean content of pigs carcasses. Two predictors for use in population studies and experiments. *Animal Production*. 28:97-108.
- FALCONER, D.S. (1985). "Introduction to Quantitative Genetics" second edition (Longman: London and New York).
- GODDARD, M.E. (1985). Policy of selecting bulls to breed bulls. *Animal Production*. 44:29-38.
- GODDARD, M.E., LEWER, R.P., GRASER, H.U., TIER, B. and JONES, L.P. (1988). Contract on "Genetic evaluation of Cattle and Sheep". *Animal Production in Australia*. 17:93-102.
- GRASER, H.U. and KLASSEN, D.J.(1988). Costs and technology involved in estimating breeding value for the pig industry. *Proceedings of Australian Association of Animal Breeding and Genetics*. 7:214-218.
- KEELE, J.W., JOHNSON, R.K., YOUNG, L.D. and SOCHA, T.E. (1988). Comparison of methods of predicting breeding values of swine. *Journal of Animal Science*. 66:3040-3048.

- KIELANOWSKI, J. (1968). The method of pig progeny testing applied in Poland. 1. General principles and physiological background. (Proceedings of the Meeting of the Sub-Commission on Pig Progeny Testing, ninth Study Meeting of the European Association of Animal Production: Dublin).
- KLASSEN, D.J., BRANDT, H. and MAKI-TANILA, A. (1988). Genetic parameters for Australian pig field data. *Proceedings of Australian Association of Animal Breeding and Genetics*. 7:505-508.
- KLASSEN, D.J. (1988). The value of BLUP indexes to pig breeders. *The Pig Farmer*. 22:32.
- LEGAULT, C. (1970). Statistical and genetical study of the performance and fattening and carcass characters of the pig. *Annales Genetique et de Selection Animale*. 2:209.
- LEGAULT, C. and GRUAND, J. (1976). Improvements in litter size in sows by the creation of hyperprolific lines and the use of artificial insemination. *Journees Recherche Porcine en France*. 8:201-206.
- LEUKKUNEN, A. (1984). Progeny testing of AI boars on the basis of their daughters farrowing results. *Acta Agriculturae Scandinavica*. 34:300-312.
- LONG, T.E. and JOHNSON, R.K. (1988). Effects of pedigree errors or selection of data on three methods of estimating breeding values for litter size, backfat and average daily gain in swine. *Proceedings of National Swine Improvement Federation*. 13:10-15.
- McPHEE, C.P. (1981). Selection for efficient lean growth in a pig herd. *Australian Journal of Agricultural Research*. 32:681-690.
- McPHEE, C.P., RATHMELL, G.A., DANIELS, L.J. and CAMERON, N.D. (1988). Selection in pigs for increased lean growth rate on a time-based feeding scale. *Animal Production*. 47:149-156.
- McPHEE, C.P. and TRAPPETT, P.C. (1987). Growth and body composition changes in mice selected for high post-weaning weight gain on two levels of feeding. *Theoretical and Applied Genetics*. 73:926-931.
- MERKS, J.W.M. (1988). Genotype by environment interactions in pig breeding programmes (IVO Report B-310 Zeist: The Netherlands).
- MOAV, R. and HILL, W.G. (1966). Specialized sire and dam lines. IV Selection within lines. *Animal Production*. 8:375-390.
- OLLIVIER, L., and BOLET, G. (1981). Selection for litter size in pigs: results of a 10 generation selection experiment. *Journees de la Recherche Porcine en France*. 13:261-267.
- PROUD, C., DONAVAN, D., KINSEY, R., CUNNINGHAM, P.J. and ZIMMERMAN, D.R. (1976). Testicular growth in boars as influenced by selection for ovulation rate. *Journal of Animal Science*. 42:1361-1362.
- REVELLE, T.J., and ROBINSON, O.W. (1973). An explanation for the low heritability of litter size in swine. *Journal of Animal Science*. 37:668-675.
- SMITH, C. (1964). The use of specialized sire and dam lines in the selection for meat production. *Animal Production*. 6:337-344.
- SMITH, C., and STRANG, G.S. (1979). A note on the heritability of litter size. *Animal Production*. 8:403-406.
- STEWART, T.S., KLASSEN, D.J. and HAMMOND, K. (1988). "Developing a Custom Selection Index for Your Herd" (Animal Genetics and Breeding Unit: Armidale).
- STRANG, G.S., and KING, J.W.B. (1970). Litter productivity in Large White pigs. 2. Heritability and repeatability estimates. *Animal Production*. 12:235-243.
- TOELLE, V.D., JOHNSON, B.H., and ROBISON, O.W. (1984). Genetic parameters for testis traits in swine. *Journal of Animal Science*. 59: 967-973.
- TORO, M.A., SILIO, I., RODRIGANEZ, J., and DOBAO M. TERESA. (1988). Inbreeding and family index selection for prolificacy in pigs. *Animal Production*. 46:79-85.
- WEBB, A.J. and BAMPTON, P.R. (1988). In "Animal Breeding Opportunities". *An Occasional publication of the British Society of Animal Production jointly with the British Poultry Breeders Roundtable*. 12:111-128.
- WHITTEMORE, C.T. and FAWCETT, R.H. (1976). Theoretical aspects of a flexible model to simulate protein and lipid growth in pigs. *Animal Production*. 22:87-90.
- YOUNG, L.D., PUMREY, R.A. CUNNINGHAM, P.J., and ZIMMERMAN, D.R. (1978). Heritability and genetic and phenotypic correlations for pre-breeding traits, reproductive traits and principal components. *Journal of Animal Science*. 46:937-949.
- ZIMMERMAN, D.R., and CUNNINGHAM, P.J. (1975). Selection for ovulation rate in swine: Population procedures and ovulation response. *Journal of Animal Science*. 40:61-69.

A SYMPOSIUM - THE EFFECTIVENESS OF CURRENT PIG VACCINES

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Introduction

The trend in recent years towards increased productivity by intensification in the pig industry has resulted in changes in management procedures. Intensification has also increased risk factors and disposition towards disease. Previously adopted antibiotic regimens aimed at reducing disease may no longer be acceptable because the pork consumer is becoming more conscious of the dangers of antibiotic residues in meat and the adverse effects it may have on human health. The trend will inevitably be towards the use of vaccines to diminish and prevent disease on pig farms. Some pig producers may have unrealistic expectations of vaccines. Vaccines are not miracle cures. Most vaccines being sold on the market have been extensively evaluated and should afford some level of protection. There are circumstances under which vaccines will fail to confer total protection. If so, it is important to realise that the problem may not reside solely in the vaccine formulation, but may be due to mitigating factors. It is the purpose of this symposium to illustrate some of these mitigating factors in the context of the disease process and the delicate balance that exists between the host animal and the bacteria which resides on or within its tissues.

The first paper in the symposium will deal in a general way with the role of vaccination in the host-micro-organism equation. Subsequent papers will discuss the role of vaccines in the light of diseases of economic importance to Australia. These diseases have been selected on the basis of net revenue loss incurred per sow according to the blueprint on Pig Health Research prepared recently by the Australian Pig Industry Research Council. Enzootic pneumonia and swine dysentery represent two disease systems of high priority to the industry and will be reviewed in the second and third papers, respectively. Vaccination against swine erysipelas and leptospirosis will be presented in the fourth and fifth papers. The final paper in this symposium will highlight two pathogens of pigs, *Campylobacter* and *Streptococcus spp.*, for which there are no currently available vaccines.

Symposium continued on next page

THE ROLE OF PIG VACCINES IN THE HOST - MICRO-ORGANISM EQUATION

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Introduction

According to the report commissioned by the Australian Pig Research Council on "A Blue Print for Pig Health Research" by Cutler and Gardner (1988), pig producers invariably list as a high priority problems of disease or a lack of good health in pigs as the major contributory factor for decreased productivity and reduced profit. A ranking system based on net revenue loss (NRL)/sow has been established to assess the relative importance of various disease syndromes commonly encountered by pig producers both large and small. For example, post-weaning colibacillosis, pneumonia and swine dysentery are ranked very high with NRLs >\$50/sow. Ranked next are diseases such as neonatal diarrhoea, *Campylobacter spp.* and atrophic rhinitis which cause net revenue losses between \$10-50/sow. Diseases caused by *Erysipelothrix rhusiopathiae* and parvovirus are economically less damaging (NRL <\$10/sow). Although the economic significance of other diseases such as coccidiosis, leptospirosis, *Streptococcus spp.* and encephalomyocarditis virus infections have not been adequately assessed, it is acknowledged that the industry should maintain a level of scientific awareness and expertise with these and other pathogens that may potentially affect productivity and profit in the pig industry.

Herd health, infection and disease

Since pig producers often perceive disease as the major cause of decreased profitability, there is a natural tendency to resort to preventative measures such as vaccination or the use of antibiotics in feed supplements. While such practices may offer some short term advantages, the disease syndrome responsible for decreased profitability is very often not eliminated and can frequently recur. It is probably far more important to recognise that genetic (Gibbons *et al.*, 1977), behavioural (Seabrook, 1988), environmental (Armstrong and Cline, 1977) and nutritional factors (Lecce *et al.*, 1983) all contribute towards the health of the pig.

Falkow (1987) defines infection as the persistent presence of a micro-organism on the surface or within the tissue of the host animal (e.g. *E. coli* in the gut, *E. rhusiopathiae* in the tonsils, *Pasteurella spp.* or *Haemophilus spp.* in the respiratory tract). The presence of micro-organisms in association with host tissues will not always lead to clinical illness or disease. A precarious and delicate balance exists between the continued presence of the micro-organism and the well-being of the host. This is because the growth of the bacteria or micro-organism is continuously held in check by the immune system of the pig.

Tipping the host-micro-organism equation

The host-micro-organism equation can be shifted by changes in environmental parameters which induce either trauma or stress (Barnett and Hemsworth, 1986). Management policies can also tip the host-micro-organism equation. For example, the indiscriminate administration of antibiotics will not only result in the accumulation of

unacceptably high levels of antibiotic residues in meat products but also shift the ecological composition of the gut microflora (Walton, 1988). A consequence of dietary antibiotics is the elimination of harmless bacteria (Lorian, 1986) which may otherwise provide an effective barrier to colonization by antibiotic-resistant and more invasive pathogenic strains (Sprunt and Leidy, 1988). Alternatively, antibiotic-resistant strains may be selected and these may then have the opportunity to proliferate rapidly beyond host containment. Seldom recognised in the host-micro-organism equation is the complex interaction that occurs between bacteria in the skin, oropharynx, gut and reproductive tract. *In vitro*, some species of bacteria enhance and some inhibit the growth of others quite independently of the host immune system (Yurdusev *et al.*, 1989) while others happily coexist. *In vivo*, the coexisting species provide a dynamic matrix at a site that apparently limits invasion by a foreign bacterium or overgrowth of a minority member of the matrix. Disturbing this balance will culminate in overgrowth or "superinfection" by the advantaged colonizing strain.

Appraisal of some commonly used pig vaccines

Of all the economically important bacterial diseases of pigs listed in Cutler and Gardner's blueprint, only killed or inactivated whole cell vaccines against colibacillosis, erysipelas and leptospirosis are being sold on a commercial scale in Australia. Of these, *E. coli* bacterins have proven to be the most effective under field conditions primarily because research in the last 10 years has amassed extensive information on the pathogenesis of this microbe. Since pathogenic strains of *E. coli* rely on the pilus for attachment to cell surfaces (Choi, 1988), antibodies directed against the pilus or its ancillary adhesive factors (Leite *et al.*, 1988) will prevent binding to and colonization of the gut epithelial cells by the pathogen. Anti-pilus antibodies are readily generated using bacterins because the pilus is present in large numbers on the surface of the bacteria. When administered as a vaccine, the pilus determinants are the most dominant and hence the pig immune system is engaged in the production of anti-pilus antibodies (To *et al.*, 1984). It is for this reason, that recombinant subunit vaccines consisting of genetically engineered subunit pilus antigens have also proved to be successful in field trials (Greenwood *et al.*, 1988).

Bacterial pathogens such as *E. rhusiopathiae* do not possess pilus nor mediate attachment to cells via adhesins. The identification of virulence determinants of *E. rhusiopathiae* has proven to be much more difficult. Nevertheless, strains of *E. rhusiopathiae* of varying arthritogenicity have been isolated and shown to differ in their DNA fingerprints even though their antigenic composition are essentially the same (Chin, unpublished data). It is possible to demonstrate using this highly arthritogenic strain as challenge bacteria, that commercially available bacterins do not protect pigs against polyarthritis. The arthritic form of swine erysipelas is a major cause of both total and partial abattoir condemnations and a source of economic loss to the pig producer. As a result of further studies on the antigenic composition of the cell wall of this bacterium (Chin and Eamens 1986), several candidate vaccine antigens have been identified. They are capable of conferring increased as well as absolute protection against arthritis in a homologous challenge (Chin *et al.*, 1989). The protective antigens are being cloned and will eventually be tested as a subunit vaccine similar to that produced for *E. coli* pilin (Chin, unpublished data).

It is important to realise that the humoral arm (i.e. the antibody-producing capability) of the immune system has evolved primarily to neutralize foreign molecules or antigens. Each antigen in turn, may possess one or more determinants or epitopes which will bind only its corresponding antibody. It is not hard to imagine that a killed vaccine composed of bacteria may contain tens of thousands of unique antigens, each with a varying combination of epitopes. The antibody producing capabilities of the

animal will be literally overwhelmed by such an offering and very often, antibodies are produced only against some of the antigens. The phenomenon of antigenic competition and antigenic dominance (Hammerl *et al.*, 1988) has immense bearing on the level of protection afforded by a vaccine, particularly since different pigs and breeds of pigs may perceive different antigens from the same bacterium as being the dominant antigen. If antibodies are generated against dominant antigens that are not virulence determinants, then the pathogenicity of the bacterium is not neutralized and disease can still occur. This is why there is a trend for scientists to investigate pathogenic mechanisms so that antigens vital to the survival of the pathogen may be identified and neutralized. One obvious advantage of this is the possibility of combining only a few antigens from several pathogens to achieve protection against many diseases. The alternative vaccination approach would be to immunize against many diseases at various times, or to combine killed cells of several pathogens in the one vaccine. The former approach is labour intensive while the latter may not always work because of antigenic competition and the possibility that antibodies may be generated against irrelevant antigens.

The pathogen is not a passive target

Vaccination represents a strategy aimed at shifting the host-micro-organism equation in favour of the host. Protection is possible if antibodies generated against a particular bacterial pathogen are successful in limiting the ability of that organism to cause disease. However, in the local environment of the respiratory tract, there are many micro-organisms with pathogenic potential. A vaccine for enzootic pneumonia (Kobish *et al.*, 1987) prepared from a strain of *Mycoplasma hyopneumoniae* may confer protection only against that strain. Other strains present in the ecological niche of the trachea and lung might remain unaffected and, in the absence of the target strain, may themselves become potent pathogens. Similarly, vaccines against *M. hyopneumoniae* (Bolske *et al.*, 1987) may not protect against *M. hyorhinis* or *M. salivarium* (Erickson *et al.*, 1988), nor against other pathogens in the "pneumonia complex" like *Actinobacillus pleuropneumoniae* (Inzana *et al.*, 1988), *Bordetella bronchiseptica*, *Pasteurella multocida* and *Streptococcus suis* (Holt *et al.*, 1988). Unless the commonality or difference in pathogenic mechanism of these organisms are elucidated, the formulation of effective bacterin-vaccines for total protection against all conceivable pathogens of the respiratory tract will be a daunting task.

Many respiratory pathogens appear to share similar pathogenic mechanisms. Like *E. coli*, respiratory pathogens must be able to attach to the mucosal epithelium (Nakai *et al.*, 1988). They generally produce toxins that destroy the epithelial cells (e.g. dermonecrotic toxins produced by *Pasteurella multocida* {Foged *et al.*, 1987} and *Bordetella bronchiseptica* {Nakai *et al.*, 1985}) or cause extensive exfoliation of the cilia. Antibodies against these components may limit the disease process but may not kill the bacteria. Some bacteria have evolved complex decoy mechanisms to avoid being killed by the host immune system. For example, certain bacteria like *Streptococcus spp.* and *Staphylococcus spp.* possess in their cell wall protein G and protein A acceptors capable of binding host immunoglobulins at the non antibody-binding site. Such bacteria have a coating of ineffectual host antibodies and no longer present themselves as being "foreign". Since the cell surface of the micro-organism provides the interface for the host immune system, it has also been the site where pathogenic mycoplasmas have evolved complex gene duplicating systems (Su *et al.*, 1988) to confuse host immunity. These genes code for essentially the same surface protein but with minor sequence variations. In this way, each subsequent generation of mycoplasma may appear to be slightly different to the pig even though it is still the same strain. It would be impossible to develop a vaccine that could be varied antigenically as rapidly as the

mycoplasma is varying itself. Other bacteria may also produce proteases that are able to destroy host immunoglobulins and hence destroy the protective antibodies generated by the vaccine.

It is quite clear that micro-organisms do not as a rule present themselves as passive targets vulnerable to immunological intervention. As fast as we might seek to shift the host-micro-organism equation in favour of the host, then too will the target bacteria seek to re-establish the equilibrium and may in fact, turn the tables around in its favour. Does this then mean that vaccines will generally be ineffectual? The answer really lies in more research and research must in the future, be carried out by multidisciplinary teams attacking the same problem from different angles. No longer can one pathogen remain the sole research domain of a single researcher. To allow this would be to ignore the complexities of the host-micro-organism equation and to permit costly and ineffective research programs that will not advance our knowledge, nor provide the answers needed to maximize profit where it counts most, on the farm.

ENZOOTIC PNEUMONIA: RECENT ADVANCES AND THE FUTURE

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Introduction

Enzootic pneumonia is probably the most widespread and economically significant disease in any swine producing country in the world (Muirhead, 1979). Economic effects of mycoplasmal pneumonia are complex. The cost of the disease is enormous. In Australia with a relatively small pig industry (approximately 4.5 million pigs slaughtered annually; Anon, 1987) the disease is estimated to cost about \$20 million/annum (Anon, 1988; extrapolation from Pointon *et al.*, 1985). Increased mortality, decreased growth rate, depressed feed conversions (Pointon *et al.*, 1985; Muirhead, 1987; Cutler, 1987), susceptibility to secondary bacterial infections, increased management costs and increased use of antibiotics are the main reasons for the economic significance of enzootic pneumonia.

Aetiology and epidemiology

The primary aetiological agent responsible for enzootic pneumonia is *Mycoplasma hyopneumoniae* although secondary bacterial invaders play a role. *M. hyopneumoniae* may infect piglets while still in the farrowing shed (Pointon *et al.*, 1985) although recent studies do suggest that the majority of animals become infected upon transfer to the multi-age grower sheds (Clark, 1988; Gardner and Hird, 1988; Sheldrake *et al.*, 1989). Results from our study (Sheldrake *et al.*, 1989; Figure 1) indicated that 43 of 44 animals sero-converted with respect to *M. hyopneumoniae* antibodies in a 2-month period commencing at about 3 months of age, which coincided with the transfer from the weaner room to the grower shed. The role and importance of *M. hyorhinis* is not well understood. It may precede *M. hyopneumoniae* infections, and we have found serological evidence of *M. hyorhinis* infection in animals at about 4 weeks of age, with small pneumonia like lesions in a *M. hyopneumoniae*-free herd.

Bacterial infections

A number of bacteria have been associated with respiratory infection and pleurisy in pigs; *Pasteurella multocida*, *Actinobacillus pleuropneumoniae*, *Haemophilus parasuis*, *Streptococcus suis* and *Bordetella bronchiseptica*. *P. multocida* is a common secondary invader found in 20% of healthy lungs, and up to 80% of pneumonic lungs (Backstrom and Hoefling, 1982). *P. multocida* results in purulent bronchopneumonia and sometimes pleuritis and pericarditis (Ross, 1984). *H. parasuis* is commonly isolated from pneumonic lesions although its exact role in the disease process is unknown. *A. pleuropneumoniae* is a recognised primary cause of pleuropneumonia (Ross, 1984). A number of serovars are known and the prevalence of this organism is increasing with intensification of the industry (Cutler, 1987).

Detection

Animals infected with *M. hyopneumoniae* are identified by lung examination at necropsy. This approach is used routinely in abattoir monitoring when the prevalence of enzootic pneumonia in a herd is being assessed. The limitations of this approach are that they require the animal to be slaughtered, and there is no confirmation that *M. hyopneumoniae* may be involved. Techniques which involve culturing *M. hyopneumoniae* organisms from diagnostic lung specimens in laboratory-prepared medium are not successful because of the fastidious nature of the organism. Another approach is the use of fluorescent dyes conjugated to *M. hyopneumoniae*-specific antibodies for mycoplasma detection by fluorescent microscopy. The procedure is relatively labour intensive and expensive. The animal must also be dead before the lung tissue can be sampled. This procedure does not lend itself to surveys in growing pigs.

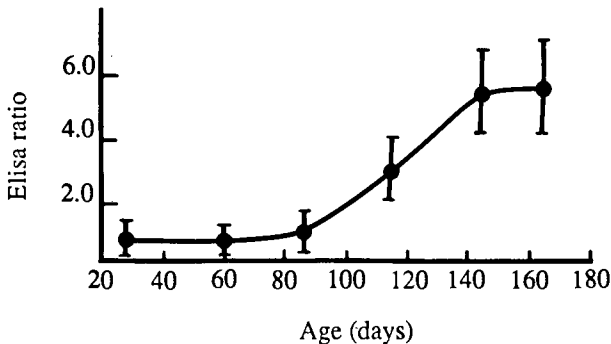


Figure 1. *M. hyopneumoniae* ELISA response for pigs in the field study. Bars represent SD (Sheldrake *et al.*, 1989).

To this end several techniques have been developed in which blood samples are used. These include indirect haemagglutination (Goodwin *et al.*, 1969), complement fixation (Boulanger and L'Ecuyer 1968) and more recently enzyme-linked immunosorbent assay (ELISA) (Bruggman *et al.*, 1977; Nicolet *et al.*, 1980).

The advantage of the ELISA is that it does not require the slaughter of the animal, is extremely cheap to perform (about \$4-6/sample as opposed to about \$110 for immunofluorescent histology) and is specific for *M. hyopneumoniae*, not cross reacting with *M. hyorhinis*. In addition, studies from our laboratory (Sheldrake, unpublished data) indicate that the ELISA is able to detect infection within about 7-10 days of the animals being challenged (Figure 2).

Because the ELISA is so cheap and the sampling procedure simple, large scale epidemiological studies are now possible. Such studies should be possible in any herd by collecting blood samples from a random sample population of animals within the herd. Further developments in ELISA technology are likely in the near future. They will result in the ELISA being sold in a kit form to the consultant veterinarian. The tests will be performed by the veterinarian in his surgery. Results will be available within hours.

Vaccines

The potential value of a vaccine for controlling enzootic pneumonia is significant and numerous studies have assessed both immune response, lung pathology and microflora following immunization. Older breeding animals have a lower prevalence of pneumonia and young animals with symptoms of pneumonia are often free from pneumonia at eventual slaughter (Lannek and Bornfors, 1957). These findings suggested that immune mechanisms may be stimulated to offer long-term protection against further infection with *M. hyopneumoniae*. Several studies (Lannek and Bornfors, 1957; Goodwin *et al.*, 1969) showed significant protection after challenge with *M. hyopneumoniae* in animals previously infected with *M. hyopneumoniae*. In one experiment this interval was extended to about 58 weeks (Goodwin *et al.*, 1969) and still the animals showed a degree of protection, greater than the control group. Further experiments investigated immunizing the sow and the role of colostral antibodies in affording protection to the offspring (Durisic *et al.*, 1975; Kobish *et al.*, 1987). These results, particularly that of the latter study, look encouraging. In the study of Kobish *et al.* (1987), vaccination of sows during gestation resulted in the elimination of micro-organisms from the respiratory tract of 95% of animals. In contrast *M. hyopneumoniae* was isolated from about 90% of piglets born to unvaccinated sows.

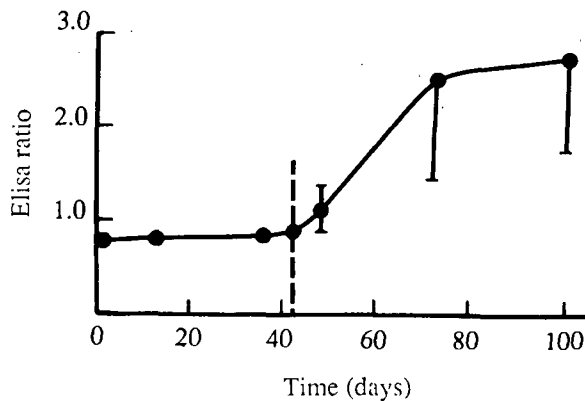


Figure 2. *M. hyopneumoniae* ELISA response of 5 pigs challenged by contact exposure on day 41. Broken line indicates the day of challenge; bars represent SD (Sheldrake *et al.*, 1989).

A number of studies have also investigated the role of parenteral vaccination (Etheridge and Lloyd, 1982; Lam and Switzer, 1971; Goodwin and Whittlestone, 1973; Ross *et al.*, 1984; Weng, 1985) and these results have also appeared encouraging. Overall, it appears likely that a vaccine for controlling *M. hyopneumoniae* will be possible in the near future; it is likely to be a whole cell vaccine although recombinant DNA technology should enable a specific antigen(s) vaccine to be developed.

Controlling pneumonia in the 1990s

There will not be a single panacea for the control of enzootic pneumonia. The effective control of this disease will be based upon:

- (1) sound management and piggery practices;
- (2) regular use of a pneumonia detection kit;
- (3) strategic use of a vaccine.

These three procedures will be required to work as part of a successful pneumonia control programme, probably the most important factor is good husbandry and shed management. Currently many piggeries are able to maintain pneumonia at an acceptable level using only these procedures.

However, where management alone is not able to reduce the prevalence of pneumonia or where the prevalence is unknown, use of a diagnostic test kit on blood samples collected from animals of a certain age will indicate the prevalence. At present, the age at which the pigs should be sampled to act as an indicator for vaccination is not known. However, from the data in Figure 1 it appears that the period shortly after entering the grower shed may be critical. If the prevalence of animals with *M. hyopneumoniae* antibodies is high at this point, then vaccination may be warranted. On the other hand, if the prevalence is low, then the economic gain from vaccination may not be justified.

The aim of this approach is to minimize costs. It is far cheaper to test a sample population of pigs and assess the disease status of the piggery rather than vaccinate all pigs irrespective of the prevalence of the disease. However, epidemiological studies examining the kinetics of enzootic pneumonia in piggeries of different design are needed to establish a recommended age for diagnostic testing of pigs. This age may vary from piggery to piggery. If this is the case, it may be more appropriate to determine the prevalence of infection in slaughter age pigs as a basis for vaccinating younger animals.

Overall, the approach of a structured pneumonia control program is likely to gain wider acceptance in the near future as the move by consumer groups to reduce the levels of antibiotics used in meat production continues.

VACCINES AND THE CONTROL OF SWINE DYSENTERY

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Introduction

Swine dysentery (SD) is a mucohaemorrhagic colitis which is widespread, and a cause of considerable economic loss to the Australian pig industry (Cutler and Gardner, 1988). The condition follows infection with the anaerobic spirochaete *Treponema hyodysenteriae* (Taylor and Alexander, 1971), and mainly occurs in pigs from about 6 weeks of age to near-slaughter age. Economic loss results from deaths, costs of therapeutic and preventive medication, and especially from depression of growth rates. Even where clinical manifestations are suppressed by antimicrobial medication, the disease may periodically reappear and will in any case continue to depress growth rates.

Pathogenesis

Disease occurs following proliferation of the spirochaete within the large intestine. It appears here associated with mucus, and may penetrate the epithelium and be found within the lamina propria. A colonic malabsorption of unknown aetiology ensues (Argenzio *et al.*, 1980).

Synergistic relationships with other anaerobic bacteria may aid in the development of the disease (Whipp *et al.*, 1979), but equally in mouse models other components of the microflora have been shown to exert protective effects (Suenaga and Yamazaki, 1986). Dietary factors may also modify the clinical outcome (Prohaszka and Lukacs, 1984).

The haemolysin of *T. hyodysenteriae* has been suggested as a virulence determinant (Kent and Lemcke, 1984), but natural avirulent isolates also produce haemolysin (Lyson *et al.*, 1982). Endotoxin has also been suggested as being involved in the pathogenesis of SD (Nuessen *et al.*, 1983), although extracts appear to be less biologically active than endotoxin from *Escherichia coli* (Greer and Wannemuehler, 1989).

Immunity

The majority of pigs which recover naturally from SD develop resistance to reinfection with the same strain of *T. hyodysenteriae*, and this protection lasts for at least 16-17 weeks (Joens *et al.*, 1979). Agglutinating antibody appears in both serum and colonic secretions (Joens *et al.*, 1984). Where convalescent sera has been used in Western blot analysis, it has been shown to react amongst other things with several polypeptides common to various strains of *T. hyodysenteriae* (Chatfield *et al.*, 1988), but with greatest reactivity against lipopolysaccharide (LPS) components of the cell wall (Wannemuehler *et al.*, 1988).

T. hyodysenteriae isolates have been divided into serotypes on the basis of their LPS antigens (Baum and Joens, 1979). Where organisms of different serotypes have been used to infect pigs, subsequent challenge into colonic loops has demonstrated that the immunity that developed is serotype specific (i.e. directed at serotype-specific LPS components) (Joens *et al.*, 1983).

Vaccination

Effective vaccines against SD are required to replace or at least supplement current control measures based on antimicrobials. Such therapy is expensive, increasingly less effective as resistance to antimicrobials appears, and is encountering public concern.

There are many difficulties in producing effective vaccines for SD. These include:

- (1) lack of knowledge about which antigens are important in inducing protective immune responses;
- (2) ignorance of the epidemiology of the infection, in particular occurrence and distribution of different antigenic types of the organism;
- (3) insufficient information about mechanisms of immunity, and of methods of inducing immunity within the intestine;
- (4) confusion about the pathogenesis of the infection, and about the role of other components of the microflora, and the diet;
- (5) difficulties in diagnosing the infection, and confusion with other forms of colitis associated with certain other treponemes;
- (6) the fastidious nature of the organisms with resultant problems in growing sufficient bacteria for use in vaccines;
- (7) difficulties in experimentally reproducing the disease, and expense of animals used in vaccine trials.

Despite these problems, the importance of the disease has led to many attempts to produce a vaccine. The first experimental vaccine was an oral laboratory-attenuated avirulent isolate of *T. hyodysenteriae* (Hudson *et al.*, 1974). Unfortunately although the organisms colonized the large intestine, they completely failed to protect against subsequent challenge with virulent isolates of the same strain. Subsequently Hudson *et al.* (1976) used a more complex regime, with live attenuated organisms given orally and intraperitoneally, and formalized virulent treponemes administered intramuscularly with Freund's incomplete adjuvant. On subsequent challenge three weeks later, nine of 18 vaccinated pigs developed SD compared to 16 of 18 control animals. This general approach has recently been refined by Lysons *et al.* (1987), who have used two intramuscular inoculations of oil-adjuvanted bacteria followed by three separate oral doses of avirulent *T. hyodysenteriae*. Using this protocol only 11 of 63 challenged pigs developed SD, and none died. Where the bacterin was used alone, 25 of 43 pigs developed SD, with no deaths. In contrast 43 of 44 unvaccinated control pigs developed SD, and 16 died.

Glock *et al.* (1978) used a different experimental immunizing routine, involving six intravenous injections of formalized *T. hyodysenteriae* at 6-day intervals. Only one of eight vaccinated pigs succumbed to challenge one week after the last inoculation.

Fernie *et al.* (1983) vaccinated pigs with a single intramuscular dose of either formalin-inactivated treponemes or both treponemes and *Campylobacter coli* in Freund's incomplete adjuvant. The vaccine stimulated production of agglutinating antibody, but two of five pigs immunized with *T. hyodysenteriae*, and three of five immunized with both bacteria developed SD following challenge. The duration and severity of the disease was however considerably reduced compared to that seen in control pigs. In Australia, Coloe and Gerraty (1988) have developed a killed whole cell vaccine which is given with adjuvant on two occasions 10-14 days apart. This preparation has been said to confer 90% or more protection. Parizek *et al.* (1985) also developed a bacterin in adjuvant which was given intramuscularly in two doses three weeks apart. Following challenge with the organism used to prepare the bacterin, 11 of 64 (17.2%) pigs developed dysentery compared to 15 of 21 (71.4%) unvaccinated pigs. When challenge

was with an organism of a different serotype from that used to prepare the bacterin, 14 or 15 (35.4%) of 41 vaccinated pigs developed disease compared to 10 or 11 (70%) of 15 control animals. These results indicated that protection was to an extent serotype specific.

Conclusion and further work

To date no vaccination regimen has been able to confer complete protection against SD. Nevertheless, even a demonstrated partial protection, with a reduction in incidence and severity of disease is a worthwhile short-term objective, and could be acceptable to the pig industry.

Vaccination regimens must be practical, and given our poor state of knowledge about protective antigens and immune mechanisms in the gut, single or dual dose bacterins with or without subsequent oral inoculation are the most appropriate current technology. Problems still may arise over LPS serotype specificity, and to that end our work has been directed at determining the number and distribution of different serological types of *T. hyodysenteriae* in Australia. Preliminary results are shown in Table 1.

Table 1. Serogroups of Australian isolates of *Treponema hyodysenteriae*

Origin	Number of isolates	LPS-serogroup ¹					D/B	Untyped
		A	B	C	D	E		
Western Australia	16	2	10	-	-	3	-	1
Victoria	15	-	8	-	-	-	2	5
Queensland	12	-	-	-	-	-	10	2
New South Wales	1	-	1	-	-	-	-	-

¹As defined by Hampson *et al.* (1989)

Organisms from three previously described LPS serogroups have so far been identified in Australia, together with another group apparently sharing antigenic determinants with groups D and B. Other isolates are currently untypable. Consideration should be given to incorporating appropriate isolates from these serogroups in any future swine dysentery vaccines for use in Australia.

Symposium continued on next page

SWINE ERYSIPELAS VACCINES

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Introduction

Swine erysipelas is manifest in one of four forms: A peracute septicaemia with sudden death; an acute septicaemia with fever, lameness and skin diamonds (urticaria); a subacute form of lesser severity with few skin lesions; and a chronic form characterized by skin, cardiac or arthritic symptoms. While the peracute and acute forms cause deaths, abortions and carcass condemnations in Australian pigs, the chronic arthritic form is economically the most damaging for pig-producers. Swine erysipelas is considered the major cause of arthritis condemnations in New South Wales abattoirs. Slaughter records indicate polyarthritis has decreased significantly in NSW since 1974, but single-joint arthritis has not (Table 2), and arthritis condemnations are costing NSW pig producers \$162,000 annually (Cutler and Gardner, 1988). Although 25 serotypes of the causative bacterium, *Erysipelothrix rhusiopathiae*, are recognized, almost all field outbreaks of erysipelas in Australian pigs have been associated with isolates of serotypes 1 or 2 (Eamens *et al.*, 1988).

Table 2. Arthritis condemnation figures for New South Wales pigs 1974-1985 (source: Department of Agriculture New South Wales Annual Reports 1975-1985).

Year	Number killed	Number totally condemned	Number totally condemned for arthritis	Number partially condemned	Number partially condemned for arthritis	Per cent condemned for arthritis
1974-5	772,525	4670	3131 (0.41%)	NA	NA	NA
1975-6	873,529	4280	3131 (0.36%)	NA	NA	NA
1976-7	1,008,797	4552	3367 (0.33%)	NA	NA	NA
1977-8	1,075,791	4096	2928 (0.27%)	NA	NA	NA
1978-9	1,031,705	4093	2947 (0.29%)	NA	NA	NA
1979-80	1,117,818	NA	1982 (0.18%)	NA	NA	NA
1980-1	1,180,013	NA	1678 (0.14%)	NA	NA	NA
1981-2	1,059,955	2109	1329 (0.13%)	10307	6454 (0.61%)	0.74%
1982-3	1,112,173	1557	906 (0.08%)	NA	4352 (0.39%)	0.47%
1983-4	1,103,414	1603	835 (0.08%)	11362	5749 (0.52%)	0.60%
1985	1,302,220	1136	470 (0.04%)	10431	5044 (0.39%)	0.43%

NA= data not available

Vaccines for swine erysipelas

Successful vaccines applied for the control of swine erysipelas have generally been of two types: Killed whole cells of *Erysipelothrix rhusiopathiae* adjuvanted with aluminium hydroxide or oil, or live, attenuated strains of *E. rhusiopathiae*. Both types contain a proportion of culture supernatant.

Killed absorbate and lysate vaccines

In the 1940s, formalin-killed, aluminium hydroxide-adsorbed bacterins were developed for the protection of pigs against acute swine erysipelas (Traub, 1947;

Dinter, 1948), and these were subsequently licensed for use in the United States and Europe. Adsorbed bacterins, given as two doses 1 month apart, afford better protection against acute disease at 3 months post-vaccination than a single dose (72% vs. 55%) (Gouge *et al.*, 1956). The *E. rhusiopathiae* strain and growth medium used for bacterin preparations are considered important. Most aluminium hydroxide-adsorbed products are made from selected strains of serotype 2 grown in a complex medium containing serum. Wood (1979) used a bacterin, prepared according to a method prescribed by USDA, comprising whole cultures (cells plus 50% of supernatant) of four immunogenic strains of serotype 2 grown in horse meat and liver infusion broth supplemented with peptone, ox bile, gelatin, glucose and horse serum. Both cells and culture supernatant contain a soluble, heat-labile antigen which is protective against acute erysipelas (Traub, 1947; Gledhill, 1952). This soluble immunizing antigen, present in whole cell bacterins is apparently composed of glycolipoprotein complexes (White and Verwey, 1970a,b), and about 20% of the total protective activity of the whole culture is present in the supernatant fluid. It has a molecular weight above 200,000 and is inactivated by trypsin and muramidase, but not by lipase or ribonuclease (White and Verwey, 1970a,b). This antigen is believed to be present in several different sizes, made up of aggregates of smaller units, with a sedimentation coefficient of 3.5S. It may reaggregate when detergents are removed or in the presence of phosphate buffer and a relatively lower pH (White and Verwey, 1970b). The isolated protective antigen in large aggregate form is composed in part of media components including horse serum antigens, which are difficult to remove from the protective subunits.

This immunizing antigen, that protects mice against challenge, can also be isolated directly from the bacterial cell wall following mechanical disruption and/or chemical extraction with alcohol/ether or NaOH (Erler, 1973). It appears as a single precipitating antigen on immunoelectrophoresis and can be extracted by precipitation with 50% saturated ammonium sulphate from culture broth and from alkaline extracts of *E. rhusiopathiae* cells of serotype 1b (Rothe, 1982a,b).

Sawada *et al.* (1987a) showed that formalised culture filtrate of the Koganei 65-0.15 (serotype 2) vaccine strain contains a protective immunogen, derived from the cell wall, that is able to induce higher levels of protective and cross-protective antibody in pigs (by mouse protection studies) against serotype 1a and 2 challenge, than formalised whole culture, formalin-killed cells or washed killed cells (Sawada *et al.*, 1987a). Although each component produces IgM and IgG responses, only IgG antibody is protective for mice (Sawada *et al.*, 1987a; Yokomizo and Isayama, 1972).

Direct vaccination of mice with the various components of broth cultures of the Koganei 65-0.15 strain has confirmed the protective activity of both formalised culture supernatant fluid and formalised bacterial cells (Sawada *et al.*, 1987b). The washing of cells does not affect growth agglutination test titres in pigs, although this procedure reduces their immunogenicity in mice (Sawada *et al.*, 1987b). While formalised whole culture, formalin-killed cells and formalised culture filtrate protected pigs against acute systemic erysipelas, only formalised culture filtrate gave complete protection against urticariae (Sawada *et al.*, 1987b). Formalised culture filtrate vaccine of *E. rhusiopathiae* strain Koganei 65-0.15 can also give good cross-protection of pigs against acute swine erysipelas due to a wide range of serotypes, but not against serotype 20 (Sawada *et al.*, 1978b). Porcine antisera prepared against the culture filtrate has been shown to protect mice against several serotypes but not against serotypes 4, 7, 8, 9, 10, 18, 19 and 20 (Sawada *et al.*, 1987c). Direct vaccination with culture filtrate in mice also gives poorer protection than live, whole-organism vaccine (Sawada and Takahashi 1987a).

Although the antigens responsible for serotype specificity (peptidoglycan) and protection (glycolipoprotein) are different, there is evidence that both types of antigens contain muramic acid (Kalf and White, 1963; White and Verwey, 1970a; Rothe, 1982a, b), probably derived from the cell wall (Feist, 1972). It is possible that these antigens

share specific fragments of cell wall peptidoglycan, which may function to some extent as common antigenic determinants (Wood, 1979) and therefore explain why some cross-protection occurs after vaccination with whole cell bacterins and live attenuated vaccines.

Wood (1979) and Wood *et al.* (1981) showed that swine vaccinated with an adsorbate bacterin of serotype 2 were protected against acute erysipelas following challenge with a virulent strain of serotypes 1, 2, 4 or 11, but not against serotype 9 or 10. Arthritis was not prevented by vaccination, but pigs were better protected against arthritis due to serotypes 1 and 2 than that due to types 9 or 10 (Wood *et al.*, 1981). The use of selected strains of serotype 2 for bacterin production is historically accepted but inadequately explained (Wood, 1979), and arises from work reported by Traub (1947) and Dedie (1949). They had found that some strains elicit a greater immune response in pigs than others, and claimed only serotype 2 strains were appropriate for the production of vaccines, since 35 of 36 such strains produced vaccines which protected mice against homologous challenge, but none of 16 serotype 1, nor 7 of type N did so. Dedie claimed that serotype 2 bacterins were effective against challenge by virulent isolates of serotypes-1 and 2, whereas serotype 1 isolates did not produce effective vaccines, but produced no data to confirm this in pigs, or to indicate that he was referring to more than the 16 type 1 strains he had studied in mice. He also reported that not all serotype 2 strains are suitable for vaccine production; highly antigenic isolates were often suitable but lowly antigenic strains seldom were. Likewise, Truszczynski (1961a) showed that a formalin-killed vaccine made from a serotype 2 pig strain grown in horse meat serum broth was protective for mice against challenge with a virulent strain, whereas similar vaccines made from a serotype 1 pig strain or serotype 2 avirulent strains were not protective. In contrast, when one of these avirulent strains was applied as a live vaccine, it was capable of protecting mice and pigs against acute erysipelas, but a second was not protective. White (1962) failed to confirm Dedie's claim of cross-protection for serotype 2 strains using cultures of serotype 1 and 2 grown in nutrient broth and cells washed twice and suspended in saline. He demonstrated protection with vaccines inoculated intravenously as cell suspensions or intramuscularly as oil-adjuvanted vaccine (Freund's incomplete) against homologous but not heterologous challenge, with intermediate protection against isologous challenge.

The importance of the choice of *E. rhusiopathiae* strain for bacterin production was demonstrated by Wood (1979), who showed that bacterins made according to the USDA method but from other, pathogenic isolates of serotypes 1, 2, 4, 9, 10 and 11, afforded poor immunity in mice and no cross-immunity in pigs; limited protection was provided against homologous challenge of pigs for serotypes 1, 2, 4 and 10 while bacterins from serotype 9 and 11 were not protective even against homologous challenge.

Delpy and Hars (1953) developed a lysate bacterin in France using *E. rhusiopathiae* cells lysed with saponin and freeze dried. They reported it was efficacious in mice challenge experiments and in field trials with pigs using a single dose, and the vaccine was subsequently licenced for use in the United States in 1955 (Wood, 1984). The presence of saponin in such preparations may have some additional activity, since saponins and their derivatives may be potent adjuvants as well as producing vaccine site reactions (Vanselow, 1987).

Oil-based vaccines

Oil-adjuvanted bacterins for erysipelas have not been widely used. Freund's incomplete adjuvant was used by White (1962) who found pig protection against acute erysipelas due to homologous but not heterologous strain challenge with serotype 1 and 2 isolates. In similar trials, Shuman *et al.* (1965a, b) used Freund's incomplete adjuvant to examine protection against acute and chronic erysipelas due to serotype 1 or 2

challenge. They observed that pigs were protected against acute erysipelas following challenge with either vaccine or challenge strain, but arthritis was observed in some vaccinated pigs after either homologous or heterologous challenge.

Water-in-oil preparations have problems of high viscosity, a delayed host reaction and breakdown on storage, but these can be partly overcome by the inclusion of a purified stabilizer (emulsifier) (Herbert, 1978). An *E. rhusiopathiae* water-in-oil emulsion bacterin was shown to give superior duration of protection compared to a formalin-killed, aluminium hydroxide-adsorbed bacterin (Jungk and Murdock, 1957; Murdock and Jungk, 1957). In 8 week-old weaners or 4 to 14 day-old sucker pigs, the water-in-oil emulsion bacterin was protective against acute erysipelas for 8 months compared to protection for less than 4 months in many adsorbate vaccinates. Two undesirable features of the emulsion bacterin were the tendency to cause a persistent local reaction and the ability to cause hypersensitivity to bacterin proteins.

Cross (1979) used an oil-based *E. rhusiopathiae* bacterin to vaccinate pigs, and demonstrated superior protection against arthritis in comparison with an aluminium hydroxide-adsorbed product. The combination of aluminium hydroxide and oil-adjuvant for *E. rhusiopathiae* was assessed by Seimenis *et al.* (1984). Using aluminium hydroxide gel and a mixture of mineral oil and emulsifier, they demonstrated improved immunogenicity in mice and pigs challenged 21 days after vaccination. Milic *et al.* (1986) prepared oil-based, lysate and adsorbate vaccines from the same strain of *E. rhusiopathiae* and showed that the oil-based preparation gave the best protection of mice and the greatest and most persistent antibody titres in pigs. They found the adsorbate vaccine was the least efficacious in mice and pigs.

Live vaccines

Live attenuated vaccines have been developed using air-drying (Staub, 1939) or by passage in media containing acridine dyes (Kondo and Sugimura, 1935; Sandstedt and Lehnert, 1944). An avirulent vaccine was licenced in the United States in 1955 for parenteral or oral inoculation (Wood, 1984), and aerosol vaccination was developed in 1958 and has been widely practiced in eastern Europe (Kaden and Beer, 1982; Kaden *et al.*, 1985). Dedie (1949) indicated that, unlike strains used in adsorbed bacterins, serotype specificity of the vaccine strain appears to be of little importance in the potential of living virulent strains to induce immunity, and this may also apply to living attenuated strains. For example, in China testing of a live vaccine made from the attenuated serotype 1a strain G₄T₁₀ has shown cross-protection against most pathogenic serotypes in mice and pigs (Pan *et al.*, 1986). Most experimental work has found that two doses of live vaccine are required for optimal effect (Ray, 1958; Konyaev and Shcherbinn, 1980; Naidenova *et al.*, 1986). In East Germany, a live aerosol vaccine was introduced for field use in 1982. This was shown to protect pigs within 6 to 10 days, and when used in 5 to 6 week-old pigs, and immunity lasted through the fattening period (Kaden *et al.*, 1985).

Wasinski (1976) found great variation in the virulence of 4 attenuated *E. rhusiopathiae* strains (A70, St 56, St Fr, VR-2) of serotypes 1, 2 and N developed in Europe. After 16 passages, all retained their order of virulence in mice and pigs and increased significantly in virulence for mice. All except one (VR-2) became more virulent in pigs. One strain (St Fr) had such innate virulence and a marked increase in its virulence on passage that it would have presented a distinct epizootic hazard.

An acriflavine-fast attenuated strain of serotype 2 (Koganei 65-0.15) given by intradermal or subcutaneous challenge, has been studied extensively in Japan. It is protective for pigs against acute erysipelas (localized or generalized) following challenge with single pathogenic strains of most serotypes, but not against serotype 20, and some strains of serotype 8, 9 and 10, which can induce localized lesions in vaccinates (Takahashi *et al.*, 1984; Sawada and Takahashi, 1987b). In mice, this vaccine failed to

prevent 20% to 30% mortality due to serotype 10, 14, 20 or 22 infection, but provided immunity against strains of other serotypes (Takahashi *et al.*, 1984; Sawada and Takahashi, 1987b).

Work with formalised culture filtrates and cells of Koganei 65-0.15 suggests the antigens which induce protection against acute erysipelas are similar for both killed and live vaccines. Since several workers have shown that multiplication of the bacteria is essential for the establishment of immunity induced by live erysipelas vaccine, the protective antigens, such as those present in culture filtrates, may be bacterial products resulting from bacterial lysis in the host (Sawada *et al.*, 1987c).

Inhibitory effect of maternal antibody

The degree and duration of passive immunity in piglets has been related to the immune status of the sow (Wood, 1986). Artificial intradermal challenge experiments suggest that passive immunity against acute urticarial swine erysipelas lasts for 6 to 8 weeks (Shuman, 1953; Vasilev *et al.*, 1978), or even 8 to 12 weeks (Chodnik and Stevens, 1962; Wellmann, 1967). Although Chodnik and Stevens (1962) and Wellmann (1967) suggested that agglutinating antibody levels in sucking pigs may be relevant to this protection, later work has shown that agglutinating antibody correlates with antigen challenge, but not with protection (Bailey, 1972; Sawada *et al.*, 1978). Fatal erysipelas may occur in sucking pigs as young as 2 weeks of age, in spite of maternal vaccination before mating and farrowing (Bastianello and Spencer, 1984).

Wellmann (1967) reported that colostral immunity has an inhibitory effect on active vaccination of piglets, which may last for 12 weeks. He showed 9-12 week-old pigs with pre-existing antibody at the time of adsorbate bacterin vaccination (particularly GAT antibody) were less protected, and their antibody level was usually not increased by vaccination. In contrast, Vasilev *et al.* (1978) showed that interfering immunity in piglets after live vaccination of the pregnant sow at 80 days gestation persisted for only 6 weeks, and active immunity against acute erysipelas can be induced by delaying live vaccination to 2 months of age.

Comparative efficacy of vaccines against acute and chronic erysipelas

Neher *et al.* (1958) and Shuman (1959) found no significant difference in the efficacy of avirulent live vaccines and bacterins under experimental conditions, while Freeman (1964) concluded that a commercial bacterin gave slightly better protection than an avirulent vaccine against the effects of acute infection. Konyaev and Shcherbinn (1980) found live VR-2 vaccine, administered parenterally on two occasions provided better protection against intramuscular challenge than an aluminium hydroxide adsorbed bacterin. In contrast, Dushuk *et al.* (1982) found no difference in protection against intradermal challenge after oral or intramuscular vaccination of live VR-2 or an adsorbed VR-2 bacterin. However, oral vaccination required a much higher dose than parenteral vaccination.

Some workers have investigated the protection afforded by vaccines against chronic erysipelas, either by field trials or by artificial challenge procedures. Neher *et al.* (1958) and Freeman (1964) reported that commercial aluminium hydroxide-adsorbed bacterins and live avirulent vaccines prevented high mortality but not infection following intravenous challenge, and did not prevent arthritis. The higher incidence and/or severity of arthritis in vaccinated pigs in these trials led Freeman to postulate that both vaccines may have increased the severity of chronic arthritis. In Australia, Webster and Summers (1977) provided indirect field evidence from Queensland that, although bacterin vaccination was reported to protect against acute erysipelas, it had little effect on the condemnation rate for arthritis and polyarthritis. In contrast, Mercy and Bond (1977) in Western Australia reported that bacterin vaccination significantly reduced polyarthritis but did not affect the prevalence of arthritis. They also noted that they

could find no field evidence to indicate that vaccination increases the prevalence of polyarthritis. Work at Glenfield with a commercially available bacterin has demonstrated the efficacy of such vaccines against acute (urticarial) swine erysipelas, but limited protection against arthritis (Eamens, unpublished data).

Bairey (1972) concluded that vaccine protection tests for acute swine erysipelas in mice and swine were not always highly correlated, referring to a number of pig-protective strains that failed to protect mice and the poor repeatability of the mouse test. Rats have been used to assess the protection of a live, avirulent, acriflavine-resistant Swedish vaccine (strain AV/R 9) against chronic erysipelas (Grabell *et al.*, 1965). Using a moderately virulent challenge strain, 2 of 20 vaccinated rats developed arthritis compared to 65 of 114 unvaccinated controls.

Combined vaccines

Immuno-modulators may have a role in anti-*E. rhusiopathiae* immunity, since killed corynebacterial antigens can augment the immunity of adsorbed *E. rhusiopathiae* vaccines in mice (Kulcsar *et al.*, 1984). Such antigens stimulate both T and B lymphocytes and activate macrophages (Vanselow, 1987).

Trivalent killed vaccines for erysipelas, pasteurellosis and bordetellosis have been marketed for use in pigs in America, and are claimed to be as efficacious as monovalent preparations (McCarthy *et al.*, 1986). Simultaneous inoculation of live attenuated *E. rhusiopathiae* (strain BP-2), swine fever and Aujeszky's disease vaccines was found inferior to inoculation of each component 20 days apart (Naidenova *et al.*, 1986), and booster dosing of the *E. rhusiopathiae* component was needed in either regime. Simultaneous vaccination with live *E. rhusiopathiae* vaccine strains VR-2 or A70 with a killed *E. coli* bacterin confers a similar immunity to monovalent vaccination (Tereszcuk *et al.*, 1974).

Other antigens of *E. rhusiopathiae*

Apart from approaches to characterize the antigens responsible for serotype and for protection against acute disease (White and Verwey, 1970a,b; Rothe, 1982a,b) little detailed systematic analysis of the antigenic components of *E. rhusiopathiae* has been undertaken. Truszczynski (1961b) used freeze-thawing and acid extraction to isolate polysaccharide and nucleoprotein fractions of 4 strains of *E. rhusiopathiae*. All fractions were antigenic in a haemagglutination test, but none were immunogenic in mice and all failed to absorb protective antibodies in horse anti-*E. rhusiopathiae* serum. Although the 2 immunogenic strains of *E. rhusiopathiae* (B and StF) contained two nucleoprotein fractions absent in the 2 non-immunogenic strains (A and StG), no immunogenic potential was attributable to these additional fractions.

Lachmann and Deicher (1986) examined the surface antigenic components of *E. rhusiopathiae* strain T28 (serotype 2) by exposing cells to detergents and characterizing the solubilized antigens by sodium dodecyl sulphate polyacrylamide gel electrophoresis (SDS-PAGE) and immunoblotting procedures. The major surface antigen was described as a polydisperse anionic polysaccharide with a molecular weight of 14,000-22,000, which was tightly bound and could only be extracted with proteinase K. This acidic polysaccharide was believed to represent a capsular polysaccharide, rather than a cell-wall component (due to the absence of muramic acid) or teichoic acid (since no teichoic acids have been described for *E. rhusiopathiae*). Of approximately 20 protein bands resolved by SDS-PAGE, the major bands had molecular weights of 64,000 and 48,000. By affinity chromatography and immunoblotting, it was shown that proteins with molecular weights of 78,000, 72,000, 68,000 and 48,000 were surface expressed and immunologically active.

Work is continuing at Glenfield in the development of vaccines for the control of erysipelas arthritis. Early studies have indicated the presence of immunogenic components of *E. rhusiopathiae* that can protect pigs against experimentally-induced urticariae and arthritis due to homologous challenge.

Symposium continued on next page

THE ROLE AND EFFECTIVENESS OF ANTILEPTOSPIRAL VACCINES IN PIGS

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Introduction

Leptospirosis, caused by the bacterium *Leptospira interrogans*, is responsible in Australia for loss of production and of profitability as a result of abortions, stillbirths and the birth of weak pigs. Leptospirosis is also a zoonosis, and human infection derived from pig contact occurs in abattoirs and on pig farms. The porcine disease is characterized by infection of the proximal renal tubules, with the result that bacteria are shed in urine for a period of weeks or months. Leptospiral killed vaccines prepared from whole leptospiral cells have been available commercially for many years, and the experimental use of attenuated live vaccines has also been reported (Hanson *et al.*, 1972).

The species *L. interrogans* consists of more than 200 serovars within 19 serogroups, and the vaccine must include one serovar from each serogroup for which protection is desired. Two killed vaccines designed for pig use are currently marketed in Australia. These are bivalent vaccines, prepared from serovars *pomona* and *tarassovi*. They are generally given as two doses four weeks apart, with boosters each six months or once during every farrowing cycle. Immunity is considered to last six months or more.

Typically only breeding female animals are routinely vaccinated on Australian pig farms. However colostral antileptospiral antibodies are passed from vaccinated sows to their progeny, and provide the piglets with temporary protection. The purpose of vaccination may be to protect breeding females against reproductive losses, to contribute to disease control in the pig herd, or to reduce the risk of human infection.

Evaluating vaccination

Leptospirosis vaccines are frequently used with the primary intention of preventing reproductive losses, and a vaccination program designed for this purpose may not necessarily reduce the spread of infection within a piggery or lead to the control or eradication of the disease. Thus a vaccination program could be assessed according to its success in preventing abortions and stillbirths, or according to its ability to prevent renal infection. Another criterion of success could be the effect on the prevalence of infection within the herd. Even the effect on pig-derived human infection could be used as a criterion of evaluation, but this would be difficult to measure.

Evaluating the effectiveness of vaccination, by whatever criterion, is not straightforward. Experimental studies in which pigs are vaccinated and subsequently challenged are of value, but they are expensive and so the numbers of animals used may be limited. Furthermore, evaluation under controlled laboratory conditions necessarily ignores many factors that can affect vaccine effectiveness in the field, including variation in the infective dose received, the route of infection, the infecting strain, the pig's genetic make-up, management and husbandry, and the environment. The use of a natural challenge received from infected "in-contact" pigs can improve the value of such experimental studies, but some of the problems in interpretation remain.

Studies of the effectiveness of vaccination on farms can be a valuable supplement to experimental vaccination and challenge.

The effectiveness of vaccination

A limited amount of vaccine evaluation has been carried out under Australian conditions. Cargill and Davos (1981) in South Australia found that vaccination reduced the frequency of renal leptospirosis but by no means eliminated it. They vaccinated groups of 15 male pigs with each of two commercially available killed bivalent vaccines prepared from serovars *pomona* and *tarassovi*. These pigs were vaccinated at 12 to 14 weeks of age, with a booster after four weeks. Subgroups of five from each group of fifteen, with nonvaccinated controls, were exposed to infected contact animals four, seven and 12 months after the booster. Overall leptospiral bacteria were demonstrated in the kidney by silver staining in 16/30 vaccinated animals and 12/14 controls. They were cultured from the kidneys of 10/30 vaccinated animals and 6/14 controls. Even four months after challenge, 4/10 vaccinated animals were demonstrably infected.

Whyte *et al.* (1982) also evaluated two commercial bivalent vaccines, administered as two doses at seven or ten months of age, for their ability to deal with a challenge carried by in-contact infected animals. Vaccination again reduced the frequency of but did not eliminate renal leptospirosis and urinary shedding, one vaccine being more effective than the other. Despite this, vaccination was very effective in controlling reproductive losses.

Palit *et al.* (1988) subsequently evaluated a new aluminium-adsorbed bivalent vaccine prepared from serovars *pomona* and *tarassovi* by administering it as two doses, four weeks apart, initially at 12 to 14 weeks of age. In one experiment, pigs were challenged four weeks after the second dose of vaccine, and in the other six months after the second dose. Complete protection (0/3 and 0/11 animals infected, compared with 4/4 and 7/7 controls) was observed. Another commercial vaccine protected only 6/8 pigs after six months, and the lesser effectiveness of that product was associated with a weak antibody response to the second dose of vaccine.

There is little Australian information about the effectiveness of vaccination in the field. Dobson (1974) used a single dose of vaccine two weeks prior to antibiotic administration to eradicate leptospirosis from three South Australian herds of 25-60 sows. Gill and Williamson (1978) eradicated serovar *pomona* from an infected piggery with 250 sows, using vaccination of sows four weeks before farrowing, and of growers at four weeks of age. They noted that the piggery practised a high standard of hygiene.

The finding that leptospiral vaccines provide only incomplete protection has also been made overseas. For example, Bryan (1957) in the United States found that a commercial killed vaccine failed to prevent leptospirosis, but an experimental vaccine did so. Hodges *et al.* (1976) in New Zealand found that two commercial serovar *pomona* vaccines reduced but failed to prevent leptospirosis. Hodges *et al.* (1985) found that 6/19 five-week-old pigs experienced leptospirosis as a result of natural challenge following the use of a commercial vaccine of serovars *pomona* and *hardjo*, designed for bovine use, compared with 19/20 unvaccinated controls. However the vaccine used was considered to be of practical value, as the overall number of urine samples in which bacteria were found was much reduced in the vaccinated group. Edwards and Daines (1979), also in New Zealand, found that vaccination, even in conjunction with antibiotic administration, did not lead to eradication of leptospirosis from a 110-sow piggery.

When does vaccination fail to protect?

Immunity to leptospirosis is recognized as being antibody-mediated (Adler and Faine, 1977). Leptospiral bacteria are destroyed by phagocytic cells following opsonization by circulating antibody (Vinh *et al.*, 1982; McGrath *et al.*, 1984). Leptospirosis infection typically consists of a leptospiraemic phase, during which bacteria circulate and invade many tissues, followed by a longer period of kidney infection during which the bacteria are shed in the urine (leptospiuria). In the pig, leptospiraemia typically lasts up to a few days, but leptospiuria may last from several weeks to more than a year.

Leptospire persist and multiply in the proximal renal tubules, where they are associated with the microvillus border, during the leptospiruric phase of infection. The lumen of the tubule is a privileged location, presumably because phagocytic cells have no access to it. Antibody itself does not readily kill the bacteria, and leptospire may survive and multiply in the presence of urinary antibody (Faine, 1963), even if they are agglutinated by the antibody.

It seems that the primary factor determining whether leptospiral infection persists is whether there is sufficient antibody present to eliminate the infection before renal infection is established. The effectiveness of vaccination is thus likely to be related to the immunogenicity of the vaccine used, insofar as it affects the level of serum antibody at the time of challenge. The factors in vaccine composition that affect immunogenicity have not been fully defined.

The effectiveness of vaccination can be expected to be less if vaccinated pigs are challenged with large numbers of organisms in a highly contaminated environment. Good farm hygiene is thus likely to enhance the value of a vaccination program.

Vaccination cannot be effective unless the vaccine contains the infective serovar, or one that falls within the same serogroup. This has implications for vaccination programs in areas where the full range of infective serovars may not be known, or in circumstances where a previously absent or unimportant pathogenic serovar becomes prominent.

Presumably because of the privileged location provided by the kidney, we find that vaccination does not usually lead to the elimination of infection that is already established, but can only prevent new infection.

Implications for diagnosis

Vaccination against animal diseases frequently compromises the serological diagnosis of disease. Vaccination induces the production of antibodies that may be wrongly taken to indicate infection, giving false positive diagnoses. This is not as great a problem with leptospirosis as it is for some other diseases because the antibodies induced by killed leptospirosis vaccines are typically of low titre and short-lived (Dobson and Davos, 1975).

A more important diagnostic effect of vaccination is to induce false-negative serological reactions. Whyte *et al.* (1982) and Hanson *et al.* (1972) found that vaccinated animals that became infected showed weaker serological responses than unvaccinated controls.

Even the direct effect of vaccination in reducing infection can affect diagnosis, because vaccinated but infected pigs may shed leptospire in the urine in lower numbers than unvaccinated controls. Infection in such vaccinated animals would be more difficult to detect using direct microscopy of urine.

One way in which vaccination of sows may affect diagnosis is by influencing the development of macroscopic lesions in their progeny. Such lesions are used at abattoirs to identify infected herds, and the associated risk of human infection. Jones *et al.*

(1987) experimentally infected eight-week-old pigs, the progeny of vaccinated or unvaccinated sows. Of the progeny of unvaccinated sows, 3/4 developed macroscopic lesions, whereas 0/4 progeny of vaccinated sows (which had therefore received maternal antibody via colostrum) developed lesions although all were clearly infected.

Implications for control

We do not have sufficient information about the effectiveness of leptospiral vaccination in the field, or about the role that it should play in efforts to control the disease or to eliminate it from a piggery. Vaccination of breeding females can be expected to control reproductive losses, at least to a substantial degree. It can also be expected to reduce (although not necessarily eliminate) infection among sows, and the shedding of leptospires in sow urine and subsequent spread to other animals. It can be expected to reduce the likelihood of human infections caused by handling aborted or stillborn piglets, or the afterbirth.

Vaccination of sows will lead to the production of colostral antileptospiral antibodies that will protect the sow's progeny at least until weaning. The period of protection depends on the concentration of antibody in the colostrum, which depends on the sow's serum antibody concentration about the time of farrowing. This in turn depends on when the sow is vaccinated. Vaccination at weaning produces less colostral antibody than vaccination in mid-pregnancy. There is thus considerable variation in the period of protection from one litter to another, but typically maternally-derived protective antibodies persist and prevent infection of the growing pig until eight weeks of age (Millar *et al.*, 1987).

Vaccination of growers is likely to be necessary for control of leptospirosis in heavily infected herds, and it must also be remembered that it is growers that present the major human infection hazard at abattoirs. However maternally derived antibody is likely to reduce the immunogenicity of leptospirosis vaccines if they are given too early in life. The appropriate age for vaccination of the progeny of vaccinated sows has yet to be determined.

Present information does not support the conclusion that vaccination is sufficient as a control measure in all infected herds, or that regular vaccination will necessarily lead to eradication of the disease. Combinations of vaccination and antibiotic treatment may be necessary, in addition to good hygiene to reduce the spread of disease through infected urine and to reduce the degree of natural challenge.

Conclusions

Commercially-available killed leptospiral vaccines have an important part to play in the control of reproductive losses in piggeries and in controlling the spread of infection to other pigs and to human beings. However vaccines vary in effectiveness, and their use does not necessarily prevent animals from becoming infected. Vaccination may complicate diagnosis, and in particular can reduce the degree of the serological response in infected animals.

It is particularly important that vaccines are properly evaluated in the correct target animal, and that they are correctly used by the farmer.

It seems likely that growing pigs as well as breeding females may need to be vaccinated in order to reduce or eliminate the level of infection within a herd. However we do not know at what age growing pigs are best vaccinated, because of the complicating influence of maternally derived antibody. It may be initially useful to use vaccination in conjunction with antibiotics in heavily-infected herds. Evaluation of alternative vaccination-induced control strategies in Australian field situations is desirable.

Symposium continued on next page

PORCINE PROLIFERATIVE ENTEROPATHIES

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The disease

Proliferative enteropathy (PE) in pigs encompasses a group of conditions in which the underlying lesion is hyperplasia of enteric crypt epithelial cells that contain intracytoplasmic curved bacilli, and loss of mucous producing cells. Superimposition of intraluminal haemorrhage, or necrosis and secondary bacterial infection, on the primary crypt cell hyperplasia accounts for the diversity of pathological presentations that have been described (Rowland and Lawson, 1975). Lesions are generally restricted to the ileum and to a lesser extent caecum and proximal spiral colon. With the exception of its haemorrhagic form, PE is not usually a fatal disease and is most commonly associated with reduced weight gain (Rowland and Rowntree, 1972; Jonsson and Martinsson, 1976). The disease PE occurs worldwide but has increased in importance in recent years (Love, 1987). The economic significance of PE to the Australian pig industry is not well established and is complicated by the difficulty in diagnosing PE antemortem. In the Australian Pig Research Council "Blueprint for Pig Health Research", Cutler and Gardner (1988) rank PE (Campylobacter-associated disease) as causing net revenue losses of \$20-50/sow. In abattoir monitoring the prevalence of PE in South Australian pig herds was commonly between five and 20% (maximum 40%) with about one third of herds affected (Pointon, 1989). In a survey in the Burnett region of Queensland, Marr, (1986) found 11 of 31 herds had lesions of PE.

The cause

Traditional concepts of infectious disease are founded on "Koch's Postulates" (Wilson *et al.*, 1983) which were formulated at the time when the role of bacteria in causing disease was starting to be recognised. Essentially these postulates state that the disease-causing organism should be recovered from the lesions of that disease and be cultured outside the body, and pure cultures of the organism should reproduce the disease in susceptible animals. However Koch's postulates have not been fulfilled in PE. It is thought that the constant association of intracellular bacteria with lesions of PE points to their likely role in the aetiology of this disease. Many workers have isolated various *Campylobacter* spp. from cases of PE and on the basis of morphological criteria and antigenic criteria it was considered that *C. sputorum mucosalis* and/or *C. hyointestinalis* were the likely causes of the disease (Lawson and Rowland, 1974; Chang *et al.*, 1984). However, numerous attempts to produce the disease by oral inoculation of *C. sputorum mucosalis* or *C. hyointestinalis* have been unsuccessful (McCartney *et al.*, 1984; Boosinger *et al.*, 1985) while oral inoculation of homogenized affected mucosa have resulted in various degrees of PE (Roberts *et al.*, 1977; Lomax *et al.*, 1982; Mapother *et al.*, 1987; McOrist and Lawson, 1989).

More recent work on the identity of the intralésional bacteria in PE has contradicted some of the earlier findings. The specificity of the immunofluorescence reactions of rabbit antiserum to *C. sputorum mucosalis* has been questioned (Lawson *et al.*, 1985). Key controls using preimmunization serum from the rabbits used for

preparation of antiserum to *C. sputorum mucosalis* had been omitted from the original work of Lawson and Rowland (1974), and it was shown that normal rabbit serum may react with the intralesional bacteria of PE in immunofluorescence tests (Lawson *et al.*, 1985). Intralesional bacteria were enriched by homogenization of affected tissues and differential centrifugation (Lawson *et al.*, 1985) and then used to prepare antiserum. In immunofluorescence tests this antiserum reacted against intralesional bacteria but did not react against *C. sputorum mucosalis*, *C. hyointestinalis* or *C. coli*, suggesting that the intralesional bacteria are antigenically distinct from these *Campylobacter spp.* (Lawson *et al.*, 1985). However, the evidence is contradictory because the immunofluorescence work reported by Chang *et al.* (1984) in which *C. sputorum mucosalis* and *C. hyointestinalis* were identified in the hyperplastic epithelium of PE, did include appropriate preimmunization serum controls. Therefore although PE has been recognised for more than twenty years as an important disease in pigs, its cause is still unknown and there are major inconsistencies in the published work in this area.

How important is proliferative enteropathy to the Australian pig industry?

The common nature of PE in South Australia and the Burnett region of Queensland has been demonstrated by Pointon (1989) and Marr (1986) and it is likely that the prevalence will be similar for other areas of Australia. Traditionally disease tends to be considered as an all or nothing condition but more and more, particularly in intensive livestock production, a disease state which is transient and causing reductions in weight gain may be economically very significant. We have suspected that major losses may be encountered due to subclinical PE and these losses may not be readily identified in commercial piggeries. However, with our current state of knowledge PE can only be accurately diagnosed at necropsy. The lesions of PE that are associated with subclinical PE have probably largely resolved by the time of slaughter. We identified major transient growth setbacks in pigs in an experimental piggery during a period when major losses due to clinical PE were encountered (unpublished data). We were unable to correlate this temporary slump in growth rate with lesions of PE when pigs were slaughtered. However, poor weight gains have been recognised as a manifestation of PE (Rowland and Rowntree, 1972; Jonsson and Martinsson, 1976) and the rapid healing capacity of the gut may account for the failure to identify lesions when pigs are slaughtered a few weeks or more after they have returned to normal levels of growth.

In studies we monitored pigs serologically for reactivity against *C. sputorum mucosalis* in an enzootically-infected piggery (unpublished data), using a microagglutination test developed by Beers (1983). We showed that pigs had no agglutinating antibody prior to ingestion of colostrum; pigs passively acquired antibody after ingestion of colostrum but this fell to undetectable levels by four weeks of age; and pigs developed high titres of antibody from about 11-14 weeks of age. This suggested that pigs are likely to be susceptible to *Campylobacter spp.* during the weaning and post-weaning periods which coincided with the period in which we had observed poor weight gains in our experimental pigs. The relevance of these findings to PE are now uncertain in view of the recent demonstration that the intralesional bacteria in PE appear to be antigenically distinct from *C. sputorum mucosalis* (Lawson *et al.*, 1985; McOrist *et al.*, 1989), however as pointed out earlier there are inconsistencies in the literature on this point.

The future

The information presented here on PE outlines some of the problems that are encountered in a disease system in which the aetiology has not been resolved and experimental production of the disease is not routinely possible. Preparation of an

efficacious vaccine becomes a difficult proposition in this situation. Particular emphasis needs to be given to the expression of the disease in different piggeries and how this coincides with different management systems and factors such as feed medicants and diets. Further studies need to be directed towards characterization of the intralesional bacteria of PE in the Australian context, and defining the host-parasite relationship in disease and non-disease states. The importance of subclinical PE as a cause of reduced weight gain in post-weaning pigs also needs to be assessed.

Symposium continued on next page

STREPTOCOCCUS SUIIS IN AUSTRALIA

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Background

Streptococcus suis is a common pathogen of pigs recognised worldwide. Although in excess of eleven serotypes of *S. suis* are recognised (Sihvonen *et al.*, 1988), two serotypes have most commonly been associated with porcine disease. *Streptococcus suis* type 1 is generally associated with septicaemia, meningitis and arthritis in unweaned pigs (Cook *et al.*, 1988) and *S. suis* type 2 causes a similar spectrum of diseases in weaned pigs (Buddle, 1987). *Streptococcus suis* type 1 is transmitted directly from the sow via the umbilicus or skin abrasions of the sucking pig (Buddle, 1987). Asymptomatic carriers are important in the epidemiology of *S. suis* type 2 infection with organisms recoverable from the tonsils of unaffected pigs (Clifton-Hadley and Alexander, 1980). Stress factors such as poor ventilation and high stocking densities are often associated with *S. suis* disease (Sharrock, 1987). Human infection with *S. suis* type 2 may occur and is usually associated with handling of infected carcasses and pig meat (Muirhead, 1987). Reports from Scandinavia indicate that *S. suis* types 3-11 and other non-typeable alpha-haemolytic streptococci are frequently isolated from cases of bronchopneumonia in pigs (Perch *et al.*, 1983; Sihvonen *et al.*, 1988). There is minimal information on the economic impact and diseases associated with *S. suis* in Australia. In the Australian Pig Research Council "Blueprint for Pig Health and Research", Cutler and Gardner (1988) conclude that research on this disease has been largely neglected.

***Streptococcus suis* serotypes associated with disease in Australia**

Epidemiological studies in Australia indicate that over 50% of normal pigs carry *S. suis* type 1 (Robertson, 1988) and over 70% of normal pigs carry *S. suis* type 2 (Robertson, 1988; Davies and Ossowicz, 1989) in the palatine tonsils. Robertson (1988) indicates that the pathogenicity of different isolates of *S. suis* varies, but currently there is minimal information on virulence factors of *S. suis*, or host factors that influence development of disease. We examined twenty one isolates of *S. suis* recovered from outbreaks of septicaemia and meningitis in weaned pigs in Australian piggeries over a four year period. Over 85% of the isolates (77% of the piggeries) were *S. suis* type 9 and the remainder were *S. suis* type 2 (Gogolewski, Cook and O'Connell, unpublished data). These findings suggest that the Australian situation may differ from the situation in Britain where *S. suis* type 2 is the cause of streptococcal meningitis in weaned pigs (Muirhead, 1987). Alternatively these findings might indicate that new serotypes of *S. suis* are emerging as important causes of disease in Australia. Isolates of *S. suis* should be routinely serotyped to determine which serotypes are most commonly associated with disease in Australia.

In our study, isolates of *S. suis* types 2 and 3 were also recovered from lungs in cases of bronchopneumonia in weaned pigs; more recently we have also recovered *S. suis* types 6 and 8. Recent surveys of *S. suis* isolations in Canada (Touil *et al.*, 1988) and Finland (Sihvonen *et al.*, 1988) indicate that *S. suis* was most commonly recovered from pneumonia and may be isolated in pure culture or in combination with a variety of pulmonary pathogens. This suggests that further studies on the role of *S. suis* in the

pathogenesis of pneumonia in Australian pigs, and the relationship between pulmonary isolates of *S. suis* and septicaemic disease, are needed.

The diversity of streptococcal serotypes that are associated with disease in Australia is also likely to have major implications on protection and vaccination of pigs. Immune protection is likely to be important in *S. suis* in pigs because pigs that have recovered from *S. suis* infection are more resistant to subsequent challenge (Upton, 1986; cited by Holt *et al.*, 1988). *Streptococcus suis* all belong to Lancefield group D but different capsular antigens confer the serotype specificity of isolates (Windsor and Elliot, 1975). Protection has been demonstrated in pigs following repeated intravenous inoculations of live virulent *S. suis* type 2 (Holt *et al.*, 1988). While such immunization protocols will not be useful in commercial piggeries, these findings indicate that a protective vaccine is feasible. Studies are needed to determine whether cross protection exists between different serotypes of *S. suis*.

Sharrock (1987) reports that the success of in-feed penicillin medication at preventing *S. suis* disease, has diverted attention from the paucity of knowledge about *S. suis* infection in Australia. As speculation continues about the desirability of using therapeutic antibiotics as in feed medicants, vaccination may become an important part of a program to control *S. suis* disease.

SYMPOSIUM CONCLUSION

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Most commercially available vaccines against pig diseases are presently sold as bacterins. These vaccines are relatively easy to prepare and would involve isolating the pathogenic strain, inactivating the bacteria and blending the antigen with an appropriate adjuvant. Some of these vaccines may fail to protect as well as one would expect under field conditions for a number of reasons, e.g. antigenic competition or shifts in the host-micro-organism equation brought about by stress etc. Improved and more reliable vaccines can be produced if the pathogenic mechanism of economically important porcine diseases were better understood. It would be feasible under these conditions to fashion vaccines that would generate antibodies able to either neutralize virulence factors or else kill the bacteria. The current generation of bacterins will probably be replaced eventually by subunit vaccines. The quality and reliability of vaccines will be further improved as more is learnt about the pig's immune system and new generation adjuvants are developed.

References

- ADLER, B. and FAINE, S. (1977). Host immunological mechanisms in the resistance of mice to leptospiral infections. *Infection and Immunity*. **17**:67-72.
- ANONYMOUS. (1987). "Australian Agriculture. The Complete Reference on Rural Industry" (National Farmers Federation, Moresope Pty. Ltd.: Camberwell, Victoria, pp. 150-153.
- ANONYMOUS. (1988). "The Cost of Swine Enzootic Pneumonia (SEP)" (Squibb: Noble Park, Victoria).
- ARGENZIO, R.A., WHIPP, S.C. and GLOCK, R.D. (1980). Pathophysiology of swine dysentery: Colonic transport and permeability studies. *Journal of Infectious Disease*. **142**:676-684.
- ARMSTRONG, W.D. and CLINE, T.R. (1977). Effects of various nutrient levels and environmental temperatures on the incidence of colibacillary diarrhoea in pigs: Intestinal fistula and titration studies. *Journal of Animal Science*. **45**:1045-1050.
- BACKSTROM, L. and HOEFLING, D.C. (1982). Respiratory diseases of swine. *Veterinary Clinics of North America: Large animal Practice*. **4**:259-276.
- BAIREY, M.H. (1972). Comments on evaluation of erysipelas vaccine. *Journal of the American Veterinary Medical Association*. **160**:607-608.
- BARNETT, J.L. and HEMSWORTH, P.H. (1986). The impact of handling and environmental factors on the stress response and its consequence in swine. *Laboratory Animal Science*. **36**:366-369.
- BASTIANELLO, S.S. and SPENCER, B.T. (1984). A report of swine erysipelas in a litter of piglets. *Journal of the South African Veterinary Association*. **55**:195-198.
- BAUM, D.H. and JOENS, L.A. (1979). Serotypes of beta-hemolytic *Treponema hyodysenteriae*. *Infection and Immunity*. **25**:792-796.
- BEERS, P.T. (1983). "Studies on Porcine Adenomatosis with Particular Reference to Proliferative Haemorrhagic Enteropathy" (Doctor of Philosophy Thesis, University of Sydney).
- BOLSKE, G., STRANDBERG, N.L., BERGSTROM, K. and JOHANSSON, K.E. (1987). Species-specific antigens of *Mycoplasma hyopneumoniae* and its cross reactions with other porcine mycoplasmas. *Current Microbiology*. **15**:233-239.
- BOOSINGER, T.R., THACKER, H.L. and ARMSTRONG, C.H. (1985). *Campylobacter sputorum mucosalis* and *Campylobacter hyointestinalis* infections in the intestine of gnotobiotic pigs. *American Journal of Veterinary Research*. **46**:2152-2156.
- BOULANGER, P. and L'ECUYER, C. (1968). Enzootic pneumonia of pigs. Complement fixation tests for the detection of mycoplasma antibodies in the serum of immunized rabbits and infected swine. *Canadian Journal of Comparative Medicine*. **32**:547-554.
- BRUGGMANN, S., KELLER, H., BERTSCHINGER, H.U. and ENGBERG, B. (1977). Quantitative detection of antibodies to *Mycoplasma suis pneumoniae* in pigs' sera by an enzyme linked immunosorbent assay. *Veterinary Record*. **101**:109-111.
- BRYAN, H.S. (1957). Studies on leptospirosis in domestic animals. VI. Vaccination of swine with *Leptospira pomona* bacterin. *Veterinary Medicine*. **52**:51-57.

- BUDDLE, J.R. (1987). Sudden deaths in pigs: Diagnostic criteria and actions. (Proceedings number 95, Post Graduate Committee in Veterinary Science, University of Sydney), pp. 319-341.
- CARGILL, C.F. and DAVOS, D.E. (1981). Renal leptospirosis in vaccinated pigs. *Australian Veterinary Journal*. 57:236-238.
- CHANG, K., KURTZ, H.J., WARD, G.E. and GEBHART, C.J. (1984). Immunofluorescent demonstration of *Campylobacter hyointestinalis* and *Campylobacter sputorum mucosalis* in swine intestines with lesions of proliferative enteritis. *American Journal of Veterinary Research*. 45:703-710.
- CHATFIELD, S.N., FERNIE, D.S., PENN, C. and DOUGAN, G. (1988). Identification of the major antigens of *Treponema hyodysenteriae* and comparison with those of *Treponema innocens*. *Infection and Immunity*. 56:1070-1075.
- CHIN, J.C. and EAMENS, G.J. (1986). Immunoreactivity of fractionated antigens obtained from an arthritogenic isolate of *E. rhusiopathiae*. *Australian Veterinary Journal*. 63:355-58.
- CHIN, J.C., EAMENS, G.J. and PANG, B. (1989). Swine erysipelas vaccines. (Eighth Australian Biotechnology Conference Proceedings, University of New South Wales: Sydney), pp. 456-461.
- CHODNIK, K.S. and STEVENS, J.W. (1962). Immunity to swine erysipelas; direct challenge with *Erysipelothrix rhusiopathiae* by intradermal route. *Journal of Comparative Pathology*. 72:142-148.
- CHOI, S.H. (1988). Binding mechanism of K88ab pili produced by enterotoxigenic *E. coli*. *Dissertation Abstracts International B*. 48:2868-2869.
- CLARK, L.K. (1988). Stalling Mycoplasma. *Hog Farm Management*. 57(October).
- CLIFTON-HADLEY, F.A. and ALEXANDER, T.J.L. (1980). The carrier site and carrier rate of *Streptococcus suis* type 2 in pigs. *Veterinary Record*. 107:40-41.
- COLOE, P.J. and GERRATY, N.L. (1988). The use of vaccination against swine dysentery to improve health status and productivity in intensively housed pigs. (Proceedings of the tenth International Pig Veterinary Society: Rio de Janeiro, Brazil), p. 123.
- COOK, R.W., JACKSON, A.R.B. and ROSS, A.D. (1988). *Streptococcus suis* type 1 infection of sucking pigs. *Australian Veterinary Journal*. 65:64-65.
- CROSS, G.M. (1979). "A Study of *Erysipelothrix Rhusiopathiae* Polyarthritits in the Pig" (Doctor of Philosophy Thesis, University of Sydney: Sydney).
- CUTLER, R. (1987). Swine respiratory disease. (Proceedings number 95, Post Graduate Committee in Veterinary Science, University of Sydney), pp. 853-887.
- CUTLER, R. and GARDNER, I. (1988). "A Blueprint for Pig Health Research" (Australian Pig Research Council: Canberra, ACT).
- DAVIES, P.R. and OSSOWICZ, C. (1989). The prevalence of *Streptococcus suis* type 2 infection in South Australian pigs. *Australian Advances in Veterinary Science*. pp. 138-139.
- DEDIE, K. (1949). Die saureloslichen Antigene von *Erysipelothrix rhusiopathiae*. *Monatschrift fur Veterinarmedizin*. 4:7-10.
- DELPY, L.P. and HARS, E. (1953). Observations sur le mode d'action des vaccins tues Vaccin solubilise immunigene contre le rouget du Porc. *Bulletin de l'Academie Veterinaire*. 26: 539-546.
- DINTER, Z. (1948). Sur Schutzimpfung gegen Schweinerotlauf mit einer Absorbatvaksine. *Tierarztliche Umschau*. 17:279.
- DOBSON, K.J. (1974). Eradication of leptospirosis in commercial pig herds. *Australian Veterinary Journal*. 50:471.
- DOBSON, K.J. and DAVOS, D.E. (1975). Leptospiral titres in pigs after vaccination. *Australian Veterinary Journal*. 51:443-444.
- DURISIC, S., MAKSIMOVIC, A., VISACKI, J., KNEZEVIC, N. and MARKOVIC, B. (1975). Antibodies in blood, colostral and milk sera of sows inoculated with an experimental vaccine of *Mycoplasma suis pneumoniae*. *Acta Veterinaria Yugoslavia*. 25:189-194.
- DUSHUK, R.V., PODLESNYKH, L.A., ZUBETS, N.A., ISACHENKO, I.V. and YUSOV, E.N. (1982). Oral immunisation against swine erysipelas. *Veterinariya (Moscow)*. 4:26-27.
- EAMENS, G.J., TURNER, M.J. and CATT, R.E. (1988). Serotypes of *Erysipelothrix rhusiopathiae* in Australian pigs, small ruminants, poultry, and captive wild animals and birds. *Australian Veterinary Journal*. 65:249-252.
- EDWARDS, J.D. and DAINES, D. (1979). A leptospirosis outbreak in a piggery. *New Zealand Veterinary Journal*. 27:247-248.
- ERIKSON, B.Z., ROSS, R.F. and BOVE, J.M. (1988). Isolation of *Mycoplasma salivarum* from swine. *Veterinary Microbiology*. 16:385-390.
- ERLER, W. (1973). Serologische, chemische und immunchemische Untersuchungen an Rotlaufbakterien. XIII Mitteilung: Das immunisierende Antigen. *Archiv fur Experimentelle Veterinarmedizin*. 27:321-326.
- ETHRIDGE, J.R. and LLOYD, L.C. (1982). A method for assessing induced resistance to enzootic pneumonia of pigs. *Research in Veterinary Science*. 33:188-191.
- FAINE, S. (1963). Antibody in renal tubules in mice. *Australian Journal of Experimental Biology and Medical Science*. 41:81-92.

- FEIST, H. (1972). Serologische, chemische und immunchemische Untersuchungen an Rotlaufbakterien. XII Mitteilung: Das Murein der Rotlaufbakterien. *Archiv fur Experimentelle Veterinarmedizin*. **26**:825-835.
- FERNIE, D.S., RIPLEY, P.H. and WALKER, P.D. (1983). Swine dysentery: Protection of pigs against infection with *Treponema hyodysenteriae*. *American Journal of Veterinary Research*. **39**:639-642.
- FOGED, N.T., PEDERSEN, K.B. and ELLING, F. (1987). Characterization and biological effect of *Pasteurella multocida* toxin. *Federation of European Microbiological Societies, Microbiology Letters*. **43**:45-51.
- FREEMAN, M.J. (1964). Effects of vaccination on the development of arthritis in swine with erysipelas: Clinical, haematologic and gross pathologic observations. *American Journal of Veterinary Research*. **25**:589-597.
- GARDNER, I.A. and HIRD, D.W. (1989). Host determinants of pneumonia in slaughter weight swine. *American Journal Veterinary Research*. (In press).
- GIBBONS, R.A., SELLWOOD, R. and BURROWS, M. (1977). Inheritance of resistance to neonatal *E. coli* diarrhoea in the pig: Examination of the genetic system. *Theoretical Applied Genetics*. **51**:65-70.
- GILL, I.J. and WILLIAMSON, P.L. (1978). Efficacy of vaccination in eradicating disease caused by *Leptospira interrogans* serotype pomona in an intensive piggery. *Victorian Veterinary Proceedings*. **36**:38-40.
- GLEDHILL, A.W. (1952). The immunising antigens of *Erysipelothrix rhusiopathiae*. The role of the L antigen. *Journal of General Microbiology*. **7**:179-191.
- GOODWIN, R.F.W., HODGSON, R.G., WHITTLESTONE, P. and WOODHAMS, R.L. (1969). Immunity in experimentally induced enzootic pneumonia of pigs. *Journal of Hygiene (Cambridge)*. **67**:193-208.
- GOODWIN, R.F.W. and WHITTLESTONE, P. (1973). Enzootic pneumonia of pigs: Immunization attempts inoculating *Mycoplasma suis pneumoniae* antigen by various routes and with different adjuvants. *British Veterinary Journal*. **129**:456-462.
- GOUGE, H.E., BOLTON, R. and ALSON, M.C. (1956). Laboratory studies on erysipelas. III. Duration of immunity in pigs vaccinated with adsorbed bacterin, and with serum and culture. *American Journal of Veterinary Research*. **17**:135-139.
- GRABELL, I., HANSEN, H.J., THAL, E. and WELLMANN, G. (1965). Chronic *Erysipelothrix rhusiopathiae* infection in laboratory rats. II. Influence of vaccination on the development of disease. *Journal of Comparative Pathology*. **75**:275-279.
- GREENWOOD, P.E., CLARK, S.J., CAHILL, A.D., TREVALLYN-JONES, J. and TZIPORI, S. (1988). Development and protective efficacy of a recombinant-DNA derived fimbrial vaccine against enterotoxigenic colibacillosis in neonatal piglets. *Vaccine*. **6**:389-392.
- GREER, J.M. and WANNEMUEHLER, M.J. (1988). Comparison of the biological responses induced by lipopolysaccharide and endotoxin of *Treponema hyodysenteriae* and *Treponema innocens*. *Infection and Immunity*. **57**:717-723.
- HAMMERL, P., WEGER, R. and THALHAMER, J. (1988). Antigenic competition in the immune response against protein mixtures: Strain-specific non-immunogenicity of *Escherichia coli* antigens. *Molecular Immunology*. **25**:313-320.
- HAMPSON, D.J., MHOMA, J.R.L., COMBS, B. and BUDDLE, J.R. (1989). Proposed revisions to the serological typing system for *Treponema hyodysenteriae*. *Epidemiology and Infection*. **102**:75-84.
- HANSON, L.E., TRIPATHY, D.N. and KILLINGER, A.H. (1972). Current status of leptospirosis immunization in cattle and swine. *Journal of the American Veterinary Medical Association*. **161**:1235-1243.
- HERBERT, W.J. (1978). "Handbook of Experimental Immunology", third edition, pp. A3.1-3.15 (Blackwell Scientific Publications: Oxford).
- HODGES, R.T., STOCKER, R.P. and BUDDLE, J.R. (1976). *Leptospira interrogans* serovar pomona infection and leptospiruria in vaccinated pigs. *New Zealand Veterinary Journal*. **24**:37-39.
- HODGES, R.T., YOUNG, G.W. and THOMSON, J.T.M. (1985). The efficacy of a leptospirosis vaccine in preventing leptospiruria in pigs. *New Zealand Veterinary Journal*. **33**:31-34.
- HOLT, M.E., ENRIGHT, M.R. and ALEXANDER, T.J.L. (1988). Immunization of pigs with live cultures of *Streptococcus suis* type 2. *Research in Veterinary Science*. **45**:349-352.
- HUDSON, M.J., ALEXANDER, T.J.L., LYSONS, R.J. and PRESCOTT, J.F. (1976). Swine dysentery: Protection of pigs by oral and parenteral immunisation with attenuated *Treponema hyodysenteriae*. *Research in Veterinary Science*. **21**:366-367.
- HUDSON, M.J., ALEXANDER, T.J.L., LYSONS, R.J. and WELLSTEAD, P.D. (1974). Swine dysentery: Failure of an attenuated strain of spirocheate given orally to protect pigs against subsequent challenge. *British Veterinary Journal*. **130**:37-40.
- INZANA, T.J., MA, J., WORKMAN, T., GOGOLEWSKI, R. and ANDERSON, P. (1988). Virulence properties and protective efficacy of the capsular polymer of *Haemophilus pleuropneumoniae*. *Infection and Immunity*. **56**:1880-1889.

- JOENS, L.A., DeYOUNG., D.W., CRAMER, J.C. and GLOCK, R.D. (1984). The immune response of the porcine colon to swine dysentery. (Proceedings of the eighth International Pig Veterinary Society: Ghent, Belgium), p. 187.
- 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., WHIPP, S.C., GLOCK, R.D. and NEUSSEN, 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, 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.
- JONSSON, L. and MARTINSSON, K. (1976). Regional ileitis in pigs: Morphological and pathogenetical aspects. *Acta Veterinaria Scandinavica*. **17**:223-232.
- JUNGK, N.K. and MURDOCK, F.M. (1957). An emulsion-type erysipelas bacterin. I. Duration of immunity in pigs vaccinated at weaning. *American Journal of Veterinary Research*. **18**:121-125.
- KADEN, V. and BEER, J. (1982). Aerogene Immunisierung gegen Schweinepest und Rotlauf in Mastanlagen. *Monatschrift für Veterinärmedizin*. **37**:380-384.
- KADEN, V., HELLER, P. and POLSTER, U. (1985). Onset and duration of immunity to swine fever and erysipelas in pigs after aerosol vaccination. *Archiv für Experimentelle Veterinärmedizin*. **39**:730-737.
- KALF, G.F. and WHITE, T.G. (1963). The antigenic components of *Erysipelothrix rhusiopathiae*. II. Purification and chemical characterisation of a type-specific antigen. *Archives of Biochemistry and Biophysics*. **102**:39-47.
- KELLEY, K.W. (1980). Stress and immune function: A bibliographic review. *Annals Research Veterinarian*. **11**:445-78.
- KENT, K.A. and LYSONS, R.M. (1984). Purification and cytotoxic activity of a haemolysin produced by *Treponema hyodysenteriae*. (Proceedings of the eighth International Pig Veterinary Society: Ghent, Belgium), p. 185.
- KOBISH, M., QUILLIEN, L., TILLON, J.P. and WROBLEWSKI, H. (1987). The *Mycoplasma hyopneumoniae* plasma membrane as a vaccine against porcine enzootic pneumonia. *Annales de l'Institut Pasteur, Immunology*. **138**:693-705.
- KONDO, S. and SUGIMURA, K. (1935). Experimental studies regarding living swine erysipelas vaccine II The pathogenicity and immunising property for swine of avirulent swine erysipelas bacilli obtained by treating with tryptaflavin. *Journal of the Japanese Society of Veterinary Science*. **14**:322-339.
- KONYAEV, M.T. and SHCHERBINN, V.K. (1980). Comparison of the efficacy of swine erysipelas vaccines. *Veterinariya (Moscow)*. **3**:33-34.
- KULCSAR, A., PADANYI, M., RETHY, L.A., RETHY, L. and BACSKAI, L. (1984). The influence of immuno-modulants on the development of secondary-type antibacterial (anti *Erysipelothrix rhusiopathiae*) immunity. *Annales Immunologiae Hungaricae*. **24**:171-176.
- LACHMANN, P.G. and DEICHER, H. (1986). Solubilisation and characterisation of surface antigenic components of *Erysipelothrix rhusiopathiae* T28. *Infection and Immunity*. **52**:818-822.
- LAM, K.M. and SWITZER, W.P. (1971). Mycoplasma pneumoniae of swine: Active and passive immunizations. *American Journal Veterinary Research*. **32**:1737-1741.
- LANNEK, N. and BORNFORSS, S. (1957). Immunity to enzootic pneumonia in pigs following recovery from the disease. *Nordisk Veterinär Medicin*. **9**:91-98.
- LAWSON, G.H.K. and ROWLAND, A.C. (1974). Intestinal adenomatosis in the pig: A bacteriological study. *Research in Veterinary Science*. **17**:331-336.
- LAWSON, G.H.K., ROWLAND, A.C. and MacINTYRE, N. (1985). Demonstration of a new intracellular antigen in porcine intestinal adenomatosis and hamster proliferative ileitis. *Veterinary Microbiology*. **10**:303-313.
- LECCE, J.G., CLARE, D.A. and BALSBAUGH, R.K. (1983). Effect of dietary regimen on rotavirus-E. coli weaning diarrhoea of pigs. *Journal Clinical Microbiology*. **17**:869-895.
- LEITE, D.S., YANO, T. and PSETANA De CASTRO, A.F. (1988). Production, purification and partial characterization of a new adhesive factor (F42) produced by enterotoxigenic *E. coli* isolated from pigs. *Annales de l'Institut Pasteur, Microbiology*. **139**:295-306.
- LOMAX, L.G., GLOCK, R.D. and HOGAN, J.E. (1982). Experimentally induced porcine proliferative enteritis in specific-pathogen-free pigs. *American Journal of Veterinary Research*. **43**:1615-1621.
- LORIAN, V. (1986). A survey of drug resistance in salmonellae isolated from animals in England and Wales in 1982 and 1983. *British Veterinary Journal*. **142**:371-380.
- LOVE, R.J. (1987). Campylobacter associated conditions. (Proceedings number 95, Post Graduate Committee in Veterinary Science, University of Sydney), pp. 1037-1043.
- LYSONS, R.M., BURROWS, M.R., JONES, P.W. and COLLINS, P. (1987). Swine dysentery, a new and effective vaccine. *Pig Veterinary Society Proceedings*. **18**:87-92.

- MAPOTHER, M.E., JOENS, L.A. and GLOCK, R.D. (1987). Investigations into the aetiology of porcine proliferative enteritis. *Veterinary Record*. **121**:86.
- MARR, G.V. (1986). Porcine intestinal adenomatosis/necrotic enteritis: The incidence in pig herds in the Burnett region of Queensland. *Australian Advances in Veterinary Science*. p. 98.
- MCCARTHY, D.H., PORTER, D.B., DOUGLASS, M.S. and SLUSSER, C.A. (1986). Preventing atrophic rhinitis, erysipelas, and pasteurellosis in pigs. *Veterinary Medicine/Small Animal Clinician*. **81**:1169-1174.
- MCCARTNEY, E., LAWSON, G.H.K. and ROWLAND, A.C. (1984). Behaviour of *Campylobacter sputorum mucosalis* in gnotobiotic pigs. *Research in Veterinary Science*. **36**:290-297.
- MCGRATH, A.G., ADLER, B., VINH, T. and FAINE, S. (1984). Phagocytosis of virulent and avirulent leptospire by guinea-pig and human polymorphonuclear leukocytes *in vitro*. *Pathology*. **16**:243-249.
- MCORIST, S., BOID, R. and LAWSON, G.H.K. (1989). Antigenic analysis of *Campylobacter* species and an intracellular *Campylobacter*-like organism associated with porcine proliferative enteropathies. *Infection and Immunity*. **57**:957-962.
- MCORIST, S. and LAWSON, G.H.K. (1989). Reproduction of proliferative enteritis in gnotobiotic pigs. *Research in Veterinary Science*. **46**:27-33.
- MERCY, A.R. and BOND, M.P. (1977). Vaccination of pigs against *Erysipelothrix rhusiopathiae*. *Australian Veterinary Journal*. **53**:600.
- MILIC, L., PETRICEVIC, S., MIJATOV, L. and TRBIC, B. (1986). Comparison of the immune response to adsorbed, lysate and oil-based vaccines against swine erysipelas. *Veterinarski Glasnik*. **40**:229-235.
- MILLAR, B.D., CHAPPEL, R.J. and ADLER, B. (1987). Detection of leptospire in biological fluids using DNA hybridisation. *Veterinary Microbiology*. **15**:71-78.
- PALIT, A., COX, J., HOLLINGWORTH, J. and SHEERS, J. (1988). Prevention of *Leptospira interrogans* serovar *pomona* infection in domestic pigs by vaccination. *Australian Veterinary Journal*. **65**:289-290.
- MUIRHEAD, M.R. (1979). Respiratory diseases of pigs. *British Veterinary Journal*. **135**:497-508.
- MUIRHEAD, M.R. (1987). Respiratory Diseases. (Proceedings number 95, Post Graduate Committee in Veterinary Science, University of Sydney), pp. 561-603.
- MURDOCK, F.M. and JUNGK, N.K. (1957). An emulsion-type erysipelas bacterin. II. Duration of immunity following vaccination of newborn pigs. *American Journal of Veterinary Research*. **18**:126-132.
- NAIDENOVA, N., MOTOVSKI, A., IORDANOV, S., DIMITROV, K., IOTOV, M., STOEV, I., PETROV, P.G., KURSHELOVA, R. and VESELINOVA, I. (1986). Simultaneous immunisation of piglets against swine fever, erysipelas and Aujeszky's disease. *Veterinarno Meditsinski Nauki*. **23**:9-13.
- NAKAI, T., KUME, K., YOSHIKAWA, H., OYAMADA, T. and YOSHIKAWA, T. (1988). Adherence of *Pasteurella multocida* or *Bordetella bronchiseptica* to the swine nasal epithelial cell *in vitro*. *Infection and Immunity*. **56**:234-240.
- NAKAI, T., SAWATA, A. and KUME, K. (1985). Intracellular locations of dermonecrotic toxins in *Pasteurella multocida* and *Bordetella bronchiseptica*. *American Journal of Veterinary Research*. **46**:870-874.
- NEHER, G.M., SWENSON, C.B., DOYLE, L.P. and SIKES, D. (1958). The incidence of arthritis in swine following vaccination for swine erysipelas. *American Journal of Veterinary Research*. **19**:5-14.
- NICOLET, J., PAROZ, P. and BRUGGMANN, S. (1980). Tween 20 soluble proteins of *Mycoplasma hyopneumoniae* as antigen for an enzyme linked immunosorbent assay. *Research in Veterinary Science*. **29**:305-309.
- 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.
- PAN N.Z., WANG, N. and LU, W. (1986). Potency tests of the swine erysipelas vaccine prepared with the attenuated strain G4T10 against various serotypes of *Erysipelothrix rhusiopathiae*. *Chinese Journal of Veterinary Medicine*. **12**:2-5.
- PARIZEK, R., STEWART, R., BROWN, K. and BLEVINS, D. (1985). Protection against swine dysentery with an inactivated *Treponema hyodysenteriae* bacterin. *Veterinary Medicine*. **80**:80-86.
- PERCH, B., PEDERSEN, K.B. and HENRICHSEN, J. (1983). Serology of capsulated streptococci pathogenic for pigs: Six new serotypes of *Streptococcus suis*. *Journal of Clinical Microbiology*. **17**:993-996.
- POINTON, A.M. (1989). *Campylobacter* associated intestinal pathology in pigs. *Australian Veterinary Journal*. **66**:90-91.
- POINTON, A.M., BYRT, D. and HEAP, P. (1985). Effect of enzootic pneumonia of pigs on growth performance. *Australian Veterinary Journal*. **62**:13-18.

- PROHASZKA, L. and LUKACS, K. (1984). Influence of the diet on the antibacterial effect of volatile fatty acids and on the development of swine dysentery. *Zentralblatt für Veterinär Medizin, Reihe B.* 31:779-785.
- RAY, J.D. (1958). Present status of prophylactic vaccination in swine erysipelas. *Journal of the American Veterinary Medical Association.* 132:365-368.
- ROBERTS, L., ROWLAND, A.C. and LAWSON, G.H.K. (1977). Experimental reproduction of porcine intestinal adenomatosis and necrotic enteritis. *Veterinary Record.* 100:12-13.
- ROBERTSON, I. (1988). Is *Streptococcus suis* a problem in Australia? *Australian Pork Journal.* April, pp. 20-23.
- ROSS, R.F. (1984). Chronic pneumonia of swine with emphasis on mycoplasmal pneumonia. (Proceedings American Association of Swine Practitioners: Kansas City, Mo), pp. 79-96.
- ROSS, R.F., ZIMMERMANN-ERICKSON, B.J. and YOUNG, T.F. (1984). Characteristics of protective activity of *Mycoplasma hyopneumoniae* vaccine. *American Journal Veterinary Research.* 45:1899-1905.
- ROTHER, F. (1982a). Das protektive Antigen des Rotlaufbakteriums (*Erysipelothrix rhusiopathiae*). 1. Mitteilung: Spezifischer Nachweis des protektiven Antigens. *Archiv für Experimentelle Veterinärmedizin.* 36:243-253.
- ROTHER, F. (1982b). Das protektive Antigen des Rotlaufbakteriums (*Erysipelothrix rhusiopathiae*). 2. Mitteilung: Die weitere Charakterisierung des protektiven Antigens. *Archiv für Experimentelle Veterinärmedizin.* 36:255-267.
- ROWLAND, A.C. and LAWSON, G.H.K. (1975). Porcine intestinal adenomatosis in the pig: A possible relationship between necrotic enteritis, regional ileitis and proliferative haemorrhagic enteropathy. *Veterinary Record.* 97:178-180.
- ROWLAND, A.C. and ROWNTREE, P.G.M. (1972). A haemorrhagic bowel syndrome associated with intestinal adenomatosis in the pig. *Veterinary Record.* 91:235-241.
- SANDSTEDT, H. and LEHNERT, E. (1944). Erfarenheter av under 1943 utförda ympningar mot rodsjuka hos svin. *Scandinavisk Veterinär-tidskrift.* 34:129-136.
- SAWADA, T., MURAMATSU, M. and SETO, K. (1978). Application of the killed-cell agglutination test with the Marienfelde strain of *Erysipelothrix rhusiopathiae* to vaccinated pigs. *Annual Report of the National Veterinary Assay Laboratory.* 15:3-11.
- SAWADA, T. and TAKAHASHI, T. (1987a). Cross protection of mice and swine inoculated with culture filtrate of attenuated *Erysipelothrix rhusiopathiae* and challenge exposed to strains of various serovars. *American Journal of Veterinary Research.* 48:37-42.
- SAWADA, T. and TAKAHASHI, T. (1987b). Cross protection of mice and swine given live-organism vaccine against challenge exposure with strains of *Erysipelothrix rhusiopathiae* representing ten serovars. *American Journal of Veterinary Research.* 48:81-84.
- SAWADA, T., TAKAHASHI, T. and SETO, K. (1987b). Immunogenicity of different fractions from broth culture of an attenuated strain of *Erysipelothrix rhusiopathiae* in mice and swine. *Japanese Journal of Veterinary Science.* 49:151-154.
- SAWADA, T., TAKAHASHI, T. and TAMURA, Y. (1987a). Protective effect of sera from swine immunised with different fractions from broth culture of an attenuated strain of *Erysipelothrix rhusiopathiae*. *Japanese Journal of Veterinary Science.* 49:37-42.
- SAWADA, T., TAKAHASHI, T. and TAMURA, Y. (1987c). Antiserum against culture filtrate is cross-protective for various serovars of *Erysipelothrix rhusiopathiae*. *Veterinary Microbiology.* 14:87-93.
- SEABROOK, M.F. (1988). The behaviour of the pig stockman and its influence on pig performance and behaviour - a review. *Pig News and Information.* 9:403-406.
- SEIMENIS, A., SKYRIANOS, G., MENASSE, I. and STOFOROS, E. (1984). In "Agriculture - Adjuvants, Interferon and Non-specific Immunity", pp. 203-208 (Commission of the European Communities: Luxembourg).
- SHARROCK, A. (1987). Weaners: *Streptococcus suis* meningitis and septicaemia. (Proceedings number 95, Post Graduate Committee in Veterinary Science, University of Sydney), pp. 1004-1005.
- SHELDRAKE, R.F., GARDNER, I.H., SAUNDERS, M.M. and ROMALIS, L.F. (1989). Serum antibody response to *Mycoplasma hyopneumoniae* measured by ELISA following experimental and natural infection of pigs. *Australian Veterinary Journal.* in press
- SHUMAN, R.D. (1953). Experimental evaluation of culture and serum vaccination for the control of swine erysipelas IV. Gilts vaccinated with culture and serum before breeding, and its immunising effect on their offspring. *Journal of the American Veterinary Medical Association.* 123:431-433.
- SHUMAN, R.D. (1959). Comparative experimental evaluation of swine erysipelas bacterins and vaccines in weanling pigs, with particular reference to the status of their dams. *American Journal of Veterinary Research.* 20:1002-1009.
- SHUMAN, R.D., WOOD, R.L. and CHEVILLE, N.F. (1965a). Sensitisation by *Erysipelothrix rhusiopathiae* (insidiosa) with relation to arthritis in pigs II Pretreatment with dead cells of serotypes A and B and challenge with live homologous or heterologous cells. *Cornell Veterinarian.* 55:387-396.

- SHUMAN, R.D., WOOD R.L. and MONLUX, W.S. (1965b). Sensitisation by *Erysipelothrix rhusiopathiae* (*insidiosa*) with relation to arthritis in pigs. III. Pretreatment with dead cells of serotype B and challenge with live or dead homologous or live heterologous cells. *Cornell Veterinarian*. **55**:397-411.
- SIHVONEN, L., KURL, D.N. and HENDRICHSEN, J. (1988). *Streptococcus suis* isolated from pigs in Finland. *Acta Veterinaria Scandinavica*. **29**:9-13.
- SPRUNT, K. and LEIDY, G. (1988). The use of bacterial interference to prevent infection. *Canadian Journal of Microbiology*. **34**:332-338.
- STAUB, A. (1939). Sur la vaccination contre le rouget du porc. *Comptes Rendus de l'Academie des Sciences (Paris)*. **208**:775-776.
- SU, C., CHAVOYA, A. and BASEMAN, J.B. (1988). Regions of *M. pneumoniae* cytoadhesin P1 structural gene exist as multiple copies. *Infection and Immunity*. **56**:3157-3161.
- SUENAGA, I and YAMAZAKI, T. (1986). Eliminating organisms against *Treponema hyodysenteriae* in the gut of mice. *Zentralblatt für Bakteriologie Infektionskr. Hyg. Reihe A*. **261**: 322-329.
- TAKAHASHI, T., TAKAGI, M., SAWADA, T. and SETO, K. (1984). Cross protection in mice and swine immunised with live erysipelas vaccine to challenge exposure with strains of *Erysipelothrix rhusiopathiae* of various serotypes. *American Journal of Veterinary Research*. **45**:2115-2118.
- TAYLOR, D.J. and ALEXANDER, T.J.L. (1971). The production of dysentery in swine by feeding cultures containing of spirochaete. *British Veterinary Journal*. **127**:58-61.
- TERESZCUK, S., WASINKA, B. and WASINSKI, K. (1974). Studies on simultaneous vaccination of pigs against infectious diseases. I. Simultaneous vaccination against erysipelas and colibacillosis. *Medycyna Weterynaryjna*. **30**:326-329.
- TO, S.C.M., MOON, H.W. and RUNNELS, P.L. (1984). Type 1 pili (F1) of porcine enterotoxigenic *E. coli*: vaccine trial and tests for production in the small intestine during disease. *Infection and Immunity*. **43**:1-5.
- TOUIL, F., HIGGINS, R. and NADEAU, M. (1988). Isolation of *Streptococcus suis* from diseased pigs in Canada. *Veterinary Microbiology*. **17**:171-177.
- TRAUB, E. (1947). Immunisierung gegen Schweinerotlauf mit konzentrierten Adsorbatimpfstoffen. *Monatschrift für Veterinärmedizin*. **2**:165-172.
- TRUSZCZYNSKI, M. (1961a). The antigenic structure of virulent and avirulent strains of *Erysipelothrix rhusiopathiae*. I. Immunobiologic properties. *American Journal of Veterinary Research*. **22**:836-837.
- TRUSZCZYNSKI, M. (1961b). The antigenic structure of virulent and avirulent strains of *Erysipelothrix rhusiopathiae*. II. Immunochemic and serologic investigations. *American Journal of Veterinary Research*. **22**:839-845.
- VANSELOW, B.A. (1987). The application of adjuvants to veterinary medicine. *Veterinary Bulletin*. **57**:881-896.
- VASILEV, V.D., STOEVI, I. and HRISTOVA, H.V. (1978). Study on the immune response in swine vaccinated at an early age with a live vaccine against *Erysipelothrix rhusiopathiae*. *Veterinarno Meditsinski Nauki*. **15**:11-18.
- VINH, T., ADLER, B. and FAINE, S. (1982). The role of macrophages in the protection of mice against leptospirosis: *in vitro* and *in vivo* studies. *Pathology*. **14**:463-468.
- WALTON, J.R. (1988). Bacterial resistance to antibiotics - The present position. *Pig News and Information*. **9**:125-127.
- WANNEMUEHLER, M.J., HUBBARD, R.D. and GREER, J.M. (1988). Characterization of the major outer membrane antigens of *Treponema hyodysenteriae*. *Infection and Immunity*. **56**:3032-3039.
- WASINSKI, K. (1976). Studies on the reversion of virulence in attenuated *Erysipelothrix rhusiopathiae* strains used for the production of live vaccines. *Bulletin of the Veterinary Institute of Pulawy*. **20**:6-12.
- WEBSTER, W.R. and SUMMERS, P.M. (1977). The effect of vaccinating pigs against *Erysipelothrix rhusiopathiae*. *Australian Veterinary Journal*. **53**:151.
- WELLMANN, G. (1967). Beobachtungen bei der Rotlauf-Immunisierung von Schweinen. III. Die Hemmung der aktiven Immunitätsbildung durch die materne Immunität. *Berliner und Münchener Tierärztliche Wochenschrift*. **80**:74-84.
- WENG, C-N (1985). "Serological Studies of Mycoplasma Infections in Pigs" (Doctor of Philosophy Thesis: Cambridge [cited in *Aslib Index to Theses*. **35**(1):435]).
- WHIPP, S.C., ROBINSON, I.M., HARRIS, D.L., GLOCK, R.D., MATHEWS, 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.
- WHITE, R.R. and VERWEY, W.F. (1970a). Isolation and characterisation of a protective antigen-containing particle from culture supernatant fluids of *Erysipelothrix rhusiopathiae*. *Infection and Immunity*. **1**:380-386.
- WHITE, R.R. and VERWEY, W.F. (1970b). Solubilisation and characterisation of a protective antigen of *Erysipelothrix rhusiopathiae*. *Infection and Immunity*. **1**:387-393.

- WHITE, T.G. (1962). Type specificity in the vaccination of pigs with killed *Erysipelothrix rhusiopathiae*. *American Journal of Veterinary Research*. 23:752-755.
- WHITE, T.G. and KALF, G.F. (1961). The antigenic components of *Erysipelothrix rhusiopathiae*. 1. Isolation and serological identification. *Archives of Biochemistry and Biophysics*. 95:458-463.
- WHYTE, P.B.D., RATCLIFF, R.M., CARGILL, C. and DOBSON, K.J. (1982). Protection of pregnant swine by vaccination against leptospira infection. *Australian Veterinary Journal*. 59:41-45.
- WILSON, G., MILES, A. and PARKER, M.T. (1983). "Topley and Wilson's Principles of Bacteriology, Virology and Immunity", seventh edition (The Williams and Wilkins Company: Baltimore).
- WINDSOR, R.S. and ELLIOT, R.D. (1975). Streptococcal infection in young pigs. IV. An outbreak of streptococcal meningitis in weaned pigs. *Journal of Hygiene Cambridge*. 75:69-78.
- WOOD, R.L. (1979). Specificity in response of vaccinated swine and mice to challenge exposure with strains of *Erysipelothrix rhusiopathiae* of various serotypes. *American Journal of Veterinary Research*. 40:795-801.
- WOOD, R.L. (1984). Swine erysipelas - a review of prevalence and research. *Journal of the American Veterinary Medical Association*. 184:944-949.
- WOOD, R.L. (1986). Erysipelas, In "Diseases of Swine", sixth edition, pp. 571-583, eds. A.D. Leman, B. Straw, R.D. Glock, W.L. Mengeling, R.H.C. Penny and E. Scholl (Iowa State University Press: Ames, Iowa).
- WOOD, R.L., BOOTH, R.L. and CUTLIP, R.C. (1981). Susceptibility of vaccinated swine and mice to generalised infection with specific serotypes of *Erysipelothrix rhusiopathiae*. *American Journal of Veterinary Research*. 42:608-614.
- YOKOMIZO, Y. and ISAYAMA, Y. (1972). Antibody activities of IgM and IgG fractions from rabbit anti-*Erysipelothrix rhusiopathiae* sera. *Research in Veterinary Science*. 13:294-296.
- YURDUSEV, N., LADIRE, M., DUCLUZEAU, R. and RAIBAUD, P. (1989). Antagonism exerted by an association of a *Bacteroides thetaotaomicron* strain and a *Fusobacterium necrogenes* strain against *Clostridium perfringens* in gnotobiotic mice and in fecal suspensions incubated *in vitro*. *Infection and Immunity*. 57:724-731.

VACCINATION AGAINST *TREPONEMA HYODYSENTERIAE*: ARTIFICIAL AND NATURAL CHALLENGE RESULTS

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In this paper we describe results of pen and field studies using an inactivated *T. hyodysenteriae* vaccine in a mineral oil adjuvant given to post-weaner pigs. In the pen studies two doses of vaccine were given 10-17 days apart and all animals were challenged 8-15 days after the second vaccination. Blood samples were taken to measure serum antibody titres using a modified ELISA test (Coloe and Smith, unpublished data). Pigs were challenged by feeding an actively growing pure culture of *T. hyodysenteriae* and swine dysentery was diagnosed by clinical assessment and at post-mortem using histopathology and by isolation of *T. hyodysenteriae*. In addition, faecal cultures of all pigs were examined 21 days after challenge. The results of the challenge studies are illustrated in Table 1.

Table 1. Protection of vaccinated pigs challenged with *T. hyodysenteriae*

	Vaccinated	Control
Number of pigs challenged	24	24
Morbidity	0	20 (83%)
Mortality	0	17 (71%)
Isolation from faeces after challenge	0	18 (75%)
Antibody titre at challenge	>1.9	<0.2

In a field trial, pigs nine weeks of age were introduced weekly into a large grower shed and penned in groups of up to 22. Approximately half of the groups were vaccinated at introduction and again two weeks later. The remainder of the pigs were the unvaccinated controls. When all pigs in the shed were on the trial, medication in the feed directed towards *T. hyodysenteriae* was withdrawn. After 122 days without medication in the feed, symptoms of swine dysentery were seen in control animals. At the time of this natural challenge, there were 16 pens of vaccinated pigs and 16 pens of controls. The disease progressed rapidly through the non-vaccinated control animals with high morbidity and mortality. Immediately symptoms of swine dysentery were observed in a pen, those pigs were treated by injection and water medication. Following the clinical outbreak of the disease, pigs from vaccinated and control pens were deliberately mixed to ensure severe challenge conditions. Of 300 vaccinated pigs, none died and none showed clinical signs of swine dysentery. Of 336 control (non-vaccinated) pigs, 250 (74%) had clinical swine dysentery and 32 (9.5%) died. Serum IgG titres averaged over 1.8 OD units in the vaccinated group and less than 0.2 OD units in the controls. The disease was confirmed in the control animals by post-mortem and isolation of *T. hyodysenteriae*.

Therefore, in conclusion, two doses of an inactivated *T. hyodysenteriae* vaccine were highly effective in protecting pigs from either natural or artificial challenge.

A VACCINE TO PREVENT PARVOVIRUS DISEASE IN PIGS

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Porcine parvovirus (PPV) causes reproductive failure characterized by embryonic and foetal death. Reproductive failures occur when a seronegative dam is exposed during the first 70 days of gestation. By 70 days of gestation most foetuses are able to develop a protective immunological response to the virus. The cost of PPV in the Australian herd has been estimated at \$5.80/sow/year (Cutler and Gardner, 1988). In an outbreak the costs are likely to be much greater, and may approach \$50/sow/year. Parsons *et al.* (1986) have examined the economics of PPV vaccination. They claim substantial economic benefits for a PPV vaccination program in breeding herds.

Maternal viraemia is a prerequisite for transplacental infection. An active immunity in the dam is essential to prevent transplacental infection. Maternally derived antibody to PPV has been shown to prevent the development of active immunity following vaccination or infection. A field trial was undertaken to determine the rate of decay of maternally derived antibody to PPV. The antibodies of the dams had been acquired by natural infection. The results showed that most gilts had lost maternally derived antibody to PPV by 21 weeks (Bates *et al.*, 1987). We have developed an inactivated PPV vaccine called Porcine Parvac (CSL). The vaccine virus is grown in cell culture, inactivated with β -propiolactone and adjuvanted with aluminium hydroxide.

The vaccine was tested in laboratory trials. Antibody negative gilts were vaccinated twice, four weeks between doses. Gilts were joined after the second dose. Pregnancy was confirmed and the gilts were challenged with virulent PPV 40-50 days after joining. After 90-110 days gestation, all gilts were sacrificed and foetuses removed. At sacrifice, all foetuses from unvaccinated gilts (seronegative at challenge) were found to be positive for PPV antigen, and positive for haemagglutination inhibition (HI) antibody.

In contrast foetuses collected from vaccinated gilts (HI titres of gilts >128) after challenge contained neither antigen nor antibody at the time of sacrifice. In litters from two unvaccinated gilts, all (12/12) foetuses showed evidence of PPV infection, and 9/12 were mummified. In contrast, in litters from 10 vaccinated gilts no (0/99) foetuses showed evidence of PPV infection. The average litter size was 9.5 from vaccinated gilts and 1.5 from unvaccinated gilts. The geometric mean HI antibody titres were 30 (range <8-256) after one dose of vaccine and 338 (range 128->1024) after the second dose.

Therefore, in conclusion the vaccine has been shown to be effective in preventing PPV reproductive failure in susceptible gilts.

References

- BATES, J., EDWARDS, S., HOLLINGWORTH, J. and PYE, D. (1987). *Australian Advances in Veterinary Science*. 1987:62-64.
- CUTLER, R. and GARDNER, I. (1988). "A Blue Print for Pig Health Research" (Australian Pig Research Council: Canberra).
- PARSONS, T.D., SMITH, G. and GALLIGAN, D.T. (1986). *Preventive Veterinary Medicine*. 4:199-204.

BIOCHEMICAL, SEROLOGICAL AND PATHOLOGICAL STUDIES OF AUSTRALIAN ISOLATES OF *ACTINOBACILLUS PLEUROPNEUMONIAE*

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Porcine pleuropneumonia is caused by the bacterium *Actinobacillus pleuropneumoniae*, formerly known as either *Haemophilus parahaemolyticus* or *Haemophilus pleuropneumoniae*, (Nicolet, 1986). Lack of knowledge about the properties of Australian isolates of *A. pleuropneumoniae*, has limited the development of effective prevention and treatment programmes for this serious disease. To overcome this deficiency we have examined the biochemical, serological and pathological properties of haemophili collected from Australian pigs.

In the biochemical study, 70 isolates of haemophili from Australian pigs were compared with reference strains of porcine haemophili. Forty-eight of the isolates were identified as *A. pleuropneumoniae* and the remaining 22 isolates as *Haemophilus parasuis*.

Forty-one of the *A. pleuropneumoniae* isolates were used to determine the minimal inhibitory concentration (MIC) of 12 antimicrobial agents. Penicillin, neomycin, trimethoprim, trimethoprim-sulphamethoxazole and tetracycline all showed low MIC values, although two isolates were clearly resistant to tetracycline. A wide range of MIC values was encountered with the sulphonamides.

All 48 *A. pleuropneumoniae* isolates were serotyped by a rapid slide agglutination test and/or a gel diffusion test. The results were; serovar-1 23 isolates; serovar-2 5 isolates; serovar-3 5 isolates; serovar-7 14 isolates and 1 non-typeable isolate.

The pathogenicity of 2 isolates each of serovars 1, 2, 3 and 7 were tested by intranasal inoculation into 6-week-old pigs. While all isolates caused pneumonia, both isolates of serovar 1 were clearly more pathogenic than the other serovars tested (serovar 1 isolates killed 12/16 pigs, serovar 2 killed 1/16, serovars 3 and 7 killed 0/14 each).

Our work has established methods for the routine identification of porcine haemophili. Penicillin appears to be the antimicrobial agent of choice for the treatment of porcine pleuropneumonia. We found that at least four serovars of *A. pleuropneumoniae* occur in Australia, with serovar 1 being the most prevalent. Pathogenicity trials demonstrated that serovar 1 isolates are potent pathogens.

References

- NICOLET, J. (1986). In "Diseases of Swine" 6th edition pp. 426-436, eds. A.D. Leman, B. Straw, R.D. Glock, W.L. Mengeling, R.H.C. Penny and E. Scholl (Iowa State University Press: Iowa).

EFFECTS OF THE MHC ON PRODUCTION AND DISEASE RESISTANCE IN PIGS

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The major histocompatibility complex (MHC) is a set of closely-linked genes that exists in all higher animals including mammals and birds. The major role of the MHC is to produce cell-surface proteins (called MHC antigens) which act as biological identity cards, enabling their owner (an individual animal) to distinguish between self and non-self. In this way, MHC antigens play a central role in immunity. Consistent with this important role, certain MHC antigens have been shown in some cases to confer increased or decreased resistance to diseases in humans and in some domestic animals (van der Zijpp and Egberts, 1989). With this background, a research programme was commenced in 1979 to determine whether the MHC of Australian pigs contained any useful genes associated with disease resistance that could be exploited in commercial breeding programmes.

After developing a new skin-transplant technique for generating typing sera, large-scale testing was conducted of hundreds of locally-obtained sera, together with others obtained from Dr. M. Vaiman in France. This eventually led to the identification of 17 different MHC antigens in Australian pigs. Ten of these antigens correspond to internationally-defined antigens. A total of 1,194 Australian pigs have been typed for some or all of the 17 MHC antigens. Included among these were pigs from four relatively large-scale studies.

The first study indicated that the number of MHC antigens common to both boar and sow did not significantly affect litter size, birth weight, or returns to service. The second study showed that MHC antigens do exert some effect on immune response to Erysipelas bacterin and to lysozyme. The third study showed no significant association between MHC antigens and clinical signs following exposure to the Erysipelas pathogen, and no associations of any practical value between MHC antigens and immune response to Erysipelas vaccination. However, there was some degree of consistency between the non-significant associations seen in the two independent Erysipelas studies. The first, second and fourth studies showed some significant but relatively weak associations with production traits, including at least two that showed consistency between Australian and French pigs.

Combining the results of these studies with those obtained in other countries (as summarised by Vaiman *et al.*, 1988), it is concluded that while the MHC does appear to influence various aspects of disease resistance and production, it is not yet possible to recommend the use of MHC antigens as genetic markers in commercial breeding programs.

References

- VAIMAN, M., RENARD, Ch. and BOURGEOUX, N. (1988). In "The Molecular Biology of the MHC of Domestic Animal Species" pp.23-38, eds. C.M. Warner, M.F. Rothschild and S.J. Lamont (Iowa State University Press: Ames).
- Van Der ZIJPP, A.J. and EGBERTS, E. (1989). *Immunology Today*. 10:109-111.

A SYMPOSIUM - NUTRITION - REPRODUCTION INTERACTIONS IN THE BREEDING SOW

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Introduction

The way in which we currently feed breeding sows has been largely determined by research conducted in the 1960s. At that time our expectations of the sows productivity were very different from those which we have now. More importantly the modern sow is very different to her counterpart of 20-30 years ago. In essence, today's sows are leaner, have a lower appetite and increased mature body size.

The purpose of this symposium is to consider recent developments in the nutrition and reproduction of the modern sow. Three areas of nutrition-reproduction interactions will be considered, namely the effects of feeding during the pre-mating period, gestation and lactation on subsequent reproductive performance.

The stimulatory effects of high energy/feed levels prior to mating on ovulation rate are well documented (Table 1). Equally, the adverse relationship between high plane feeding in early gestation and embryo survival rate is well reported, although less well accepted. It is probable that acceptance of this relationship has not been widespread for two reasons: (1) much of the data presented to support the relationship utilized high- or low-plane feeding both pre- and post-mating, and hence confounded the results by altering ovulation rate; (2) approximately 50% of the studies conducted failed to detect significant effects of gestation feeding on embryo survival (it should be noted, however, that no study has reported a beneficial effect of increased feed level on embryo survival rate). In Table 2 data are presented on the relationship between early gestation feed level and embryo survival for studies in which pre-mating feed level was standardized. These data still show an adverse effect of high plane feeding in early gestation on survival of embryos.

Table 1. The influence of pre-mating energy intake on ovulation rate in the pig (adapted from Aherne and Kirkwood, 1985)

	Pre-mating feeding level	
	High	Low
Number of trials	36	30
Energy intake (MJ DE/day)	42.8	23.4
Ovulation rate	13.7	11.8

The effects of nutrient intake during lactation on subsequent reproductive performance of the gilt/sow appear to be more complex and contentious than the above, and are undoubtedly poorly understood at the moment. Overall underfeeding in lactation is usually reported to cause an extension of the weaning to remating interval (Table 3), has little effect on ovulation or conception rates, but may adversely influence subsequent early embryo survival (Table 4). Differences between studies appear to be primarily due to three factors:

- (1) the use of first or subsequent parity sows;
- (2) imposition of various levels of feed, energy and/or protein;
- (3) inadequate feed/energy intakes during lactation in the high treatments see Table 3.

It is the intention of this symposium to review the current state of knowledge on the above interactions that occur between nutrition and reproduction in pigs and the levels of body weight and tissue changes, endocrine parameters and overall sow productivity. The concluding section of the symposium will attempt to integrate this information into a feeding strategy for the breeding sow.

Table 2. The effects of feed level in early gestation on embryo survival in sows that were fed at a standard level pre-mating (summary of 12 experiments adapted from Anderson and Melampy, 1972)

	Gestation feeding level	
	High	Low
Ovulation rate	15.4	15.5
Number of viable embryos	11.8	12.7
Embryo survival (%)	77	82

Symposium continued on next page

Table 3. The effects of nutrition during lactation on the length of weaning to remating interval in gilts and sows

Number of experiments	Number of animals	Sows/gilts	High feed intake ^a	Low feed intake	Mean weaning to remating interval (days)			
					High energy intake ⁺	Low energy intake	High protein intake [#]	Low protein intake
4 ^a	248	gilts	10.5	19.3	-	-	-	-
8 ^b	784	gilts	-	-	12.7	14.6	-	-
4 ^c	190	gilts	-	-	-	-	10.3	16.6
5 ^d	384	sows	5.6	7.1	-	-	-	-
2 ^e	658	sows	-	-	4.4	4.3	-	-
0	-	sows	-	-	-	-	-	-

^a Mean daily DE intake (MJ), gilts 53, sows 81; ⁺ mean daily DE intake (MJ), gilts 64, sows 69; [#] mean daily CP intake (g), gilts 766.

^b King *et al.* (1984); King and Williams (1984a); Armstrong *et al.* (1986); King and Dunkin (1986a).

^c Reese *et al.* (1982) - 2 studies; King and Williams (1984b); Reese *et al.* (1984); Johnson *et al.* (1986); Brendemuhl *et al.* (1987); Kirkwood *et al.* (1987a).

^d King and Williams (1984b); King and Dunkin (1986b); Brendemuhl *et al.* (1987); King and Martin (1989).

^e Henry *et al.* (1984); Hughes *et al.* (1984); Kirkwood *et al.* (1987a,b); Yang *et al.* (1989).

Reese *et al.* (1982); Kirkwood *et al.* (1988).

Table 4. The effects of lactation feed level on subsequent early embryo survival and litter size in sows and gilts

Authors	Number of animals	Sows/gilts	Early embryo survival rate (%)		Subsequent litter size (total)	
			High lactation feed	Low lactation feed	High lactation feed	Low lactation feed
Reese <i>et al.</i> (1982)	44	gilts	-	-	9.4	9.9
Henry <i>et al.</i> (1984)	40	sows	-	-	9.9	8.7
Hughes <i>et al.</i> (1984)	26	sows	70	58	-	-
King and Williams (1984a)	80	gilts	71	72	9.7	9.7
Kirkwood <i>et al.</i> (1987a)	24 48	gilts sows	80	67	-	-
Kirkwood <i>et al.</i> (1987b)	78	sows	83	68	-	-
Kirkwood <i>et al.</i> (1988)	201	sows	-	-	10.6	9.6
Prime <i>et al.</i> (1988)	80	sows	-	-	11.4	11.6

NUTRITIONAL STRATEGIES FOR BREEDING SOWS

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Introduction

In developing nutritional strategies for sows the end point is usually defined as output per unit time (e.g. piglets weaned per sow per year). As the sow will be expected to achieve high levels of performance for several years, feeding strategies need to accommodate long, as well as short term, influences of nutrition. In achieving these aims maternal body condition has been recognized as having particular significance in both short and long term nutrition of sows.

Lactation

Nutrition of sows has often been based on systems involving large weight loss in lactation together with the restoration of tissues during the subsequent pregnancy. Such changes have usually been measured as live weight but it is unlikely that these closely reflect tissue changes. It has been suggested that lactation should be the focal point for the long term nutrition of the sow which should be based on the maximum conservation of tissues at this stage (Cole, 1982). The ability to monitor such condition changes has been helped in practice by condition scoring techniques and the availability of ultrasonic systems.

Lactation and subsequent reproduction

Sows markedly losing condition in lactation may be associated subsequently with poor reproductive performance. While Hardy and Lodge (1969) reported a loss of 1 ovum, together with poorer conception at next oestrus, with every 1 kg loss of weight in lactation, other reports have contradicted this. However, there is considerable evidence of the involvement of body tissues in subsequent reproduction. For example fat loss (e.g. King, 1987) and protein loss (e.g. King and Williams, 1984b) have both been associated with delayed oestrus.

Requirements in lactation

The needs of the sow in lactation are substantial due to the high demands of milk production. In many situations there is a "sparing" effect as a result of the catabolism of body tissues. In fact, a number of recommendations assume a maternal contribution. For example, ARC (1981) assumed that sows would lose 180 g body weight/day and have a requirement of 65-76 MJ DE/day (depending on weaning age and level of milk production) for a 160 kg sow. Similarly Aherne and Kirkwood (1985) suggested an intake of 75 MJ DE/day for a 165 kg sow losing 6.5 kg body weight in a 42 day lactation. Requirements which have assumed no maternal tissue loss have, for example, involved intakes of 66.1 MJ DE/day in the first week to 102.5 MJ DE/day in the fourth week of lactation (Mullan *et al.*, 1989). Recent work at the University of Nottingham has suggested intakes of 85.3, 97.6 and 93.5 MJ DE/day to avoid losses in maternal live weight, P₂ backfat and condition score respectively (Cole, 1989).

Feed intake in lactation

Strategies which avoid large losses of live weight, fat and protein in lactation imply moderately high feed intakes (say >6 kg/day) yet modern sows have reportedly low appetites (in some cases below 3 kg/day). Consequently, it is of importance to understand the factors which influence feed intake in lactation and examine ways in which the knowledge can be used to further the nutritional objective.

For convenience the factors which influence feed intake in the sow can be regarded as the animal, the environment and the diet.

The animal

It has been suggested that modern genotypes may have been selected in such a way that appetite has been reduced. Cole and Chadd (1989) have illustrated this using the relationship between live weight and daily digestible energy intake and it has been implied that such effects may be manifest in the sow (Cole, 1989).

It is well known that sows which are highly fed in pregnancy have reduced feed intake in lactation (Salmon-Legagneur and Rerat, 1962). Similar results have been achieved at the University of Nottingham with modern genotypes (Figure 1). When energy intake rose above 26 MJ DE/day (2 kg food) in pregnancy there was a marked fall in daily intake in lactation. The fall was substantial; voluntary feed intake in lactation decreased by about 25% when energy intake in pregnancy was raised to 35 MJ DE/day.

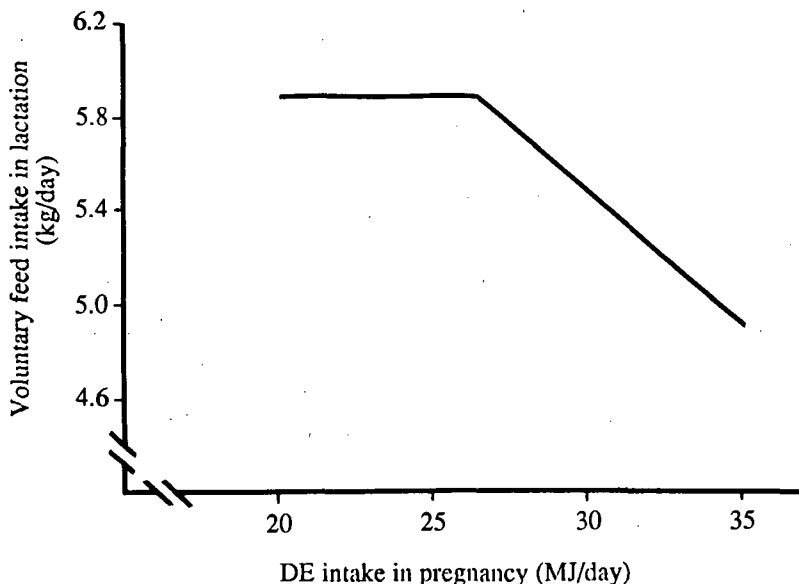


Figure 1. The influence of feed intake in pregnancy on voluntary feed intake in lactation (based on Harker and Cole, unpublished data).

The environment

Environment has a marked effect on feed intake, and high temperatures which result from hot climates or poor building design are a particular problem. Reductions in intake of 120-140 g/day/°C (Cole, 1989) and 100-200 g/day/°C (Lynch, 1989; Stansbury, *et al.*, 1987) have been reported.

The diet

Diet quality has a large effect on appetite and dietary energy plays an important role in feed intake control. It has been suggested that growing pigs eat more of

low energy diets in order to achieve a constant daily energy intake until a point is reached when physical limitation replaces physiological control (Cole *et al.*, 1967). There are suggestions that compensation for dietary quality over the range of physiological control is incomplete with growing pigs (Cole and Chadd, 1989). It was further suggested that there might be a minimum bulk intake needed to avoid gastric hunger sensations (Cole *et al.*, 1972).

The role of diet in feed intake control in the sow has received little attention. Again sows are known to respond to energy and eat more of very poor quality diets (Zoiopolous, 1978). However, when fed higher quality diets there appears to be little compensation with the result that higher daily digestible energy intakes are achieved with diets of higher digestible energy concentration (Table 5).

Clearly the sow has a high requirement for energy and nutrients at this stage which is markedly affected by milk production, for example, accounting for 65-80% of the requirements. Whether the sow eats to a bulk intake in order to try to achieve these high levels or whether animals close to maturity are more influenced by physical rather than physiological mechanisms is not clear.

The influence of dietary protein on feed intake in the sow has been well established (Mahan and Mangan, 1975). While sows ate more of high protein (18%) than low protein (12%) diets, the situation was even worse when those given low protein in lactation also received low protein in pregnancy (9 or 13% vs 17%).

Table 5. Voluntary feed intake in lactation (Cole, 1989)

Dietary DE (MJ/kg)	12.5	13.5	14.5
Lynch (1989)			
Feed intake (kg/day)	4.92	5.08	
DE intake (MJ/day)	61.2	70.3	
Zhu and Cole (unpublished)			
Feed intake (kg/day)	6.01		5.89
DE intake (MJ/day)	75.13		85.41

In situations where feed intake is a problem, high energy and nutrient (particularly protein) density diets are of value, particularly in temperate climates. However, there is a suggestion (Lynch, 1989) that very high protein diets for lactating sows (e.g. 20% vs 14%) may reduce intake at high temperatures (28° C) but increase intake at low temperatures (16° C). Clearly these interactions need further investigation.

Metabolic and tissue status

The requirements of the breeding sow are markedly influenced by reproductive status and it is conventional to consider meeting requirements for the separate reproductive stages. However, there is the further influence of whether the sow is anabolic or catabolic, with the latter having a "sparing" effect on requirements. It is generally assumed that the sow is anabolic in pregnancy and catabolic in lactation. This is not the case, for example Close *et al.* (1985) have shown that sows offered 21 MJ DE/day mobilized fat at day 87 of pregnancy and that losses during late pregnancy could account for up to 4.8 kg fat or 20% of the sows fat reserves. Thus there is a case for taking more account of the potentially catabolic and anabolic phases of the breeding cycle.

Recent work at the University of Nottingham has sought to do this. For example, 26.4 MJ DE/day throughout pregnancy was shown to be adequate for sows

to increase live weight but insufficient to maintain condition score or P₂ backfat from day 90 of pregnancy (Table 6). Raising intake to 39.6 MJ DE/day overcame this, but if sows were merely fed high levels in late pregnancy as part of a redistribution exercise so that they were given commensurately less in lactation, then the gains were lost by the end of the breeding cycle. However, when they received 71 MJ DE/day in lactation the differences were maintained throughout the breeding cycle but those offered only 26 MJ DE/day in pregnancy had lost condition and P₂ fat by weaning.

Little attention has been paid to the change from a catabolic to an anabolic phase. It has been suggested that failure to adjust to an anabolic phase after weaning could be a major factor in the problem of small litters of second parity sows through reduction of ovulation rate (Brooks, 1982). Clearly this is an area requiring further research.

Table 6. The influence of digestible energy intake in late pregnancy on changes in live weight, P₂ fat and condition score (Harker and Cole, unpublished)

	Pregnancy	Pregnancy		
	Days 0-90	A	B	C
Energy intake (MJ DE/day)	26.4	26.4	39.6	52.8
Change in:				
Live weight (kg)	36.2	10.0	18.9	26.1
P ₂ fat (mm)	4.1	-0.8	0.5	1.7
Condition score ¹	0.51	-0.11	0.12	0.13

¹MAFF scale from 0-5

Feeding system

Sows are kept under a wide variety of conditions from intensive sow stalls on concrete to group housing using electronic feeders to outdoor sows. The ways in which these systems influence nutrient requirements is of importance. For example, attention has recently been drawn to differences in the response of sows housed in groups on straw and individually housed sows on concrete (Cole, 1989). The former gained about 10 kg more live weight in the first four weeks of pregnancy which may have resulted from differences in climatic environment, the insulative value of straw and huddling in groups with consequent influences on critical temperature together with any nutritional contribution of straw. However, in this work straw consumption would have accounted for very little of the extra weight gain.

Conclusions

Greater emphasis will need to be placed on the continuum of events from the metabolism of the animal through to the practical application of feeding systems. For example, while attention is being given to relationships between reproduction and tissue status and their modification, it is important to understand the mechanisms involved in such relationships. Knowledge gained to date has relied heavily on short term experiments and particularly, in relation to tissue status, there is a need to generate more information on long term effects. The animal and its environment do not represent static entities and consequently it is important to take account of the changing nature of pig genotypes and the conditions which are being developed for them.

Symposium continued on next page

NUTRITIONAL INFLUENCES ON SOWS

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Introduction

There are two steps in the design of feeding strategies for sows. The first involves setting reproductive targets, for example, the age and weight for mating gilts, the amount of energy during pregnancy required to grow the conceptus without detriment to food intake in the following lactation, and the amount of energy in lactation needed to maintain milk and leave the sow in an acceptable condition at weaning to commence the next reproductive cycle. The second step is setting nutrient allowances to meet these specified targets and this requires a knowledge of the factors that affect the partition of nutrients between maternal tissues, the products of conception, and milk. The effects of some of these factors are now known well enough that animal performance can be predicted with some confidence. In the following paper we will begin by setting some reproductive targets. We will then discuss some of the nutritional implications of meeting these targets and illustrate how factors such as body weight, genotype, and diet are likely to affect the response of the animal.

Reproductive targets

Reproductive efficiency is best measured as the number and weight of piglets weaned from a sow taken from the time she is selected as a gilt from the bacon pen to the time she is slaughtered. This suggests that to maximize efficiency a gilt should be mated as young as possible and then kept in the herd reproducing regularly with the minimum farrowing interval. The interval from weaning to conception has a major effect on the farrowing interval. It decreases with an increase in the length of lactation, parity, plane of nutrition in the previous lactation, or a reduction in litter size (Hughes and Varley, 1980). The results from several recent experiments (King and Williams, 1984b; King and Dunkin, 1986a,b; Mullan and Williams, 1989; Williams, Head and Pearce, unpublished data; Hughes, unpublished data) show that the weaning-to-mating interval is related to live weight of the sow and that she will exhibit the minimum interval of about 5 days if weaned after a lactation of three weeks or more with a weight of 150 kg or greater (Figure 2). If weaned at a live weight of less than 150 kg then the interval increases so that it may take 3 weeks or more if weaning weight is 120 kg. If a target weight for weaning gilts or sows is set at a live weight of 150 kg what are the nutritional implications for designing a feeding strategy?

Lactation

The requirements of the lactating sow for energy and protein have recently been calculated by Mullan, Close and Cole (1989) using the empirical method. These calculations were based on the energy and protein requirement for milk production to sustain different rates of piglet gain plus that required for maintenance of the sow. The example presented in Table 7 is for a sow weighing 160 kg post-partum rearing a litter of 9 piglets with a mean growth rate of 225 g over a 4-week lact-

ation. The amount of energy and protein required to maintain the sow and her milk production was 83 MJ DE and 810 g crude protein/day requiring a mean food intake in excess of 6 kg/day. However, according to published values of the voluntary food intake of sows (Mullan *et al.*, 1989) it would be unlikely for a sow weighing 160 kg post-partum to have a mean intake above 5 kg/day. The animal responds by maintaining milk production at the expense of maternal body reserves, a total weight loss during lactation of approximately 10 kg comprising almost entirely of fat tissue.

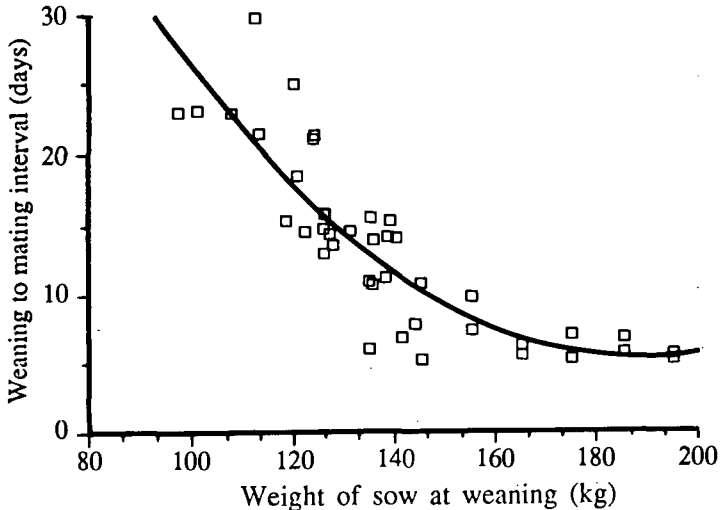


Figure 2. *Weight of sow at weaning and weaning to mating interval. The regression equation was; $Y = 102.17 - 1.0337X + 2.7587 \times 10^{-3}X^2$ ($r^2 = 0.762$).*

Gestation

If it is accepted that, at best, weight losses in lactation are likely to be 10 kg or more in a four-week lactation, then sows should weigh 160 kg at farrowing to achieve the desired weaning weight of 150 kg so the weaning-to-mating interval is minimized. We have used Auspig (Black *et al.*, 1986) to simulate the growth of gilts and their litters from selection until they were weaned of their first litter. The results of the simulation are given in Table 8 and they show the effects of weight at mating, genotype, and body fat on the performance of gilts in pregnancy and lactation. Gilts were selected at 85 kg with either 18 mm (average genotype) or 14 mm (improved genotype) of backfat. They were mated at 100, 120 or 140 kg live weight and fed protein-adequate diets in sufficient amounts so they reached 160 kg at farrowing.

All gilts grew at their prescribed rates in gestation and farrowed at their predetermined weights except the gilts of the average genotype mated at the lightest weight of 100 kg. These animals were fed *ad libitum* but were unable to eat sufficient energy to reach 160 kg at farrowing. There was an interaction between birth weight of piglets and genotype. All piglets from gilts of the average genotype were heavy because these animals consumed enough energy above their maintenance needs so that conceptus growth was never limited at any stage in pregnancy. Conceptus growth was limited in gilts of the improved genotype so that birth weight of the piglets was reduced. The lightest piglets were from gilts that made the smallest maternal gains in pregnancy.

Table 7. Nutrient requirement and body tissue changes during lactation, (litter size =9; no creep feed provided; 160 kg sow post-partum)

Week of lactation	1	2	3	4
Average daily gain of piglets (g)	160	220	260	250
Milk production				
Energy (MJ ME/d)	39	57	68	71
Digestible nitrogen (g/d)	69	99	109	97
Total requirement				
Energy (MJ ME/d)	61	77	88	91
Digestible nitrogen (g/day)	88	116	126	126
Daily intake				
Feed (kg) ¹	3.8	4.9	5.5	5.8
Energy (MJ ME)	49	63	70	74
Digestible nitrogen (g)	85	111	125	131
Daily balance				
Energy (MJ DE)	-12	-14	-18	-17
Digestible nitrogen (g)	-1	-5	-1	+5
Change in body tissue (g/day)				
Fat	-251	-286	-372	-376
Lean	-94	-142	-42	+143
Body weight	-365	-451	-436	-246

¹ Diet containing 13.5 MJ DE/kg and 160 g CP/kg

All sows were fed *ad libitum* in lactation. The improved sows mobilized small amounts of tissue, maintained a good milk supply, weighed more than 150 kg at weaning and returned into oestrus early. Sows of the average genotype also maintained a good milk supply but, in doing so, mobilized large amounts of tissue because their voluntary food intake declined in lactation. Their weight at weaning was well below 150 kg and their subsequent fertility was reduced because the interval from weaning to mating increased.

The reason for the prediction that sows of average genotype are less fertile than sows of the improved genotype is that a target weight of 150 kg at weaning cannot be reached because voluntary food intake in lactation is reduced.

Voluntary food intake - a limit to reproduction?

This symposium has already highlighted some of the factors affecting voluntary food intake of sows in lactation including some work at Nottingham demonstrating that sows which are fed high in pregnancy reduce their food intake in lactation. The interaction between food intake in pregnancy and lactation is puzzling. When energy output is at a maximum during lactation, why should a gilt or sow choose to reduce her food intake and mobilize her body stores? Is it an effect of body weight, that is, the more a sow eats in pregnancy the closer she gets to her mature size, or, is it an effect of body composition, the amount of body fat? At the University of Western Australia we have attempted to resolve this by manipulating the body composition at the same body weight. Gilts were fed diets containing different amounts of protein during pregnancy so that they farrowed at the same body weight but with different amounts of body fat. Body fatness at the beginning of lactation has a large effect on food intake and this is independent of body weight. In heavy sows which weighed 181-183 kg after farrowing the leaner animals with 30 mm of backfat ate 22% more food than the fatter sows with 41 mm of backfat. In the lighter animals (167 to 168 kg) the difference was much greater with the leaner ones

lighter animals (167 to 168 kg) the difference was much greater with the leaner ones eating 60% more energy (Table 9).

Table 8. Auspicious simulation for gilts of average and improved genotypes in pregnancy and lactation (Black, personal communication)

	Genotype					
	Average		Improved		Improved	
Live weight (kg)						
Initial	85	85	85	85	85	85
Mating	100	118	142	101	121	140
Farrowing	156	161	164	158	158	159
Weaning	132	135	136	153	154	155
Backfat (mm)						
Initial	18	18	18	14	14	14
Mating	21	27	38	16	20	25
Farrowing	44	46	47	32	31	31
Weaning	32	33	34	24	23	23
Piglets born alive	9	9	9	9	9	9
Piglet weight (kg)						
Birth	1.46	1.46	1.44	1.33	1.20	1.07
Weaning (4 weeks)	7.51	7.35	7.22	7.59	7.39	7.15
Interval from weaning to mating (days)	14	13	12	5	5	5

The effect of body fat on food intake is shown in Figure 3 and this suggests that once body fat exceeds about one third of body weight then voluntary energy intake decreases. The corollary of this is that the body fat of sows should be controlled before they farrow to ensure adequate intake during lactation to meet a target weight at weaning.

The amount of body fat at farrowing can be manipulated in two main ways. Genotypes vary in body composition as illustrated in Table 8. Body fat can also be manipulated by altering the amount and composition of the diet. At any energy intake, body fat is minimized by feeding a protein-adequate diet, that is, a diet which supplies sufficient amino acids so that they will not limit protein synthesis. As energy intake of a protein-sufficient diet increases animals become fatter because the ratio of fat to protein in the gain increases. The magnitude of the response varies and depends on level of intake. There are large increases in the ratio at either end of the spectrum, that is, close to maintenance and close to *ad libitum* levels of energy intake. There is a large range in the middle where there is little change in the ratio. Reproductive targets, in particular the performance of sows during their first reproductive cycle, can thus be met by the manipulation of feeding strategy and genotype.

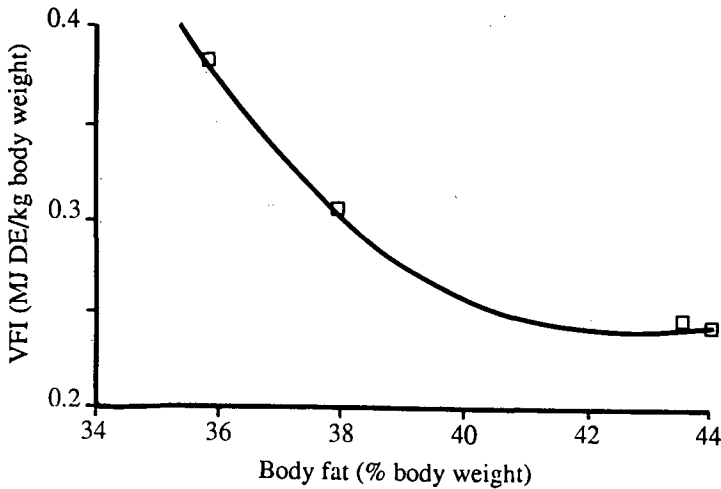


Figure 3. Voluntary food intake (VFI) in lactation vs body fat after farrowing. The regression equation was; $Y = 5.6126 - 0.2521X + 2.9558 \times 10^{-3}X^2$ ($r^2 = 0.999$).

Table 9. Body composition and food intake in lactation (Williams, Head and Pearce, unpublished)

	High Fat	High Lean	Low Fat	Low Lean
Number of sows weaned	15	21	21	22
At mating				
Body weight (kg)	112	108	105	108
Body fat (P ₂ mm)	20	18	20	19
After farrowing				
Body weight (kg)	183	181	168	167
Body fat (P ₂ mm)	41	30	37	26
Body fat (% body weight)	44	38	43	36
Voluntary intake in lactation				
Feed (kg air-dry/day)	3.12	3.81	2.77	4.47
MJ of DE/day	44.3	54.1	39.9	63.5

THE ENDOCRINE BASIS OF NUTRITION - REPRODUCTION INTERACTIONS

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Introduction

The previous sections have provided clear evidence for both direct and indirect effects of nutrition on reproduction. In essence these effects fall into three categories:

- (1) high level feeding during the pre-mating period stimulates higher ovulation rates (the flushing effect);
- (2) low level feeding during lactation results in an elongation of the weaning to oestrus interval and a possible reduction in subsequent litter size;
- (3) high level feeding in early gestation reduces embryo survival rate.

The endocrine basis of each of these effects will be considered below.

Nutrition and ovulation rate

There is ample evidence to suggest that increasing the level of food intake or energy content of the diet above maintenance for a period of 8-14 days prior to oestrus results in an increase in ovulation rate (Aherne and Kirkwood, 1985). Increasing the energy intake of the gilt during the follicular phase of the oestrous cycle has been demonstrated to increase plasma levels of FSH (Rhodes *et al.*, 1987; Flowers *et al.*, 1988) and the pulse frequency of LH (Cox *et al.*, 1987; Rhodes *et al.*, 1987; Flowers *et al.*, 1988). This suggests that flushing enhances ovulation rate by stimulating secretion of gonadotrophins. Furthermore, since episodic release of LH is primarily regulated by hypothalamic GnRH secretion (Clarke and Cummins, 1982) it must be inferred that flushing exerts its primary effect on ovulation rate via an alteration in the hypothalamic GnRH pulse generator.

The mechanism whereby flushing alters the activity of the GnRH pulse generator has yet to be determined. However, metabolic hormones whose levels respond to dietary energy levels are clearly implicated. In particular, plasma insulin levels have been suggested as the primary modulator of energy influences on reproduction since they are closely correlated with energy intake. The identification of insulin binding sites in areas of the hypothalamus known to be involved in GnRH release supports this possibility (van Houten *et al.*, 1979; Landau *et al.*, 1983). Indeed, the provision of exogenous insulin has been shown to significantly raise ovulation rate in gilts, this being associated with increased pulse frequency of LH (Cox *et al.*, 1987).

Insulin mediated changes in GnRH secretion may be increasing ovulation rate via an effect on either follicle recruitment and/or by lessening the rate of follicle atresia (Dailey *et al.*, 1975). The major alterations seen in gonadotrophin secretion in flushed gilts occur during the period 4-2 days prior to oestrus (Rhodes *et al.*, 1987; Flowers *et al.*, 1988), and are preceded by increases in plasma insulin levels. Greater concentrations of FSH between day 3 and 1 before oestrus in flushed gilts (Flowers *et al.*, 1988) may have increased follicle recruitment by extending the period

that FSH levels remained elevated (Baird, 1987).

Evidence is also available to indicate that insulin may act to rescue follicles that could otherwise become atretic over the final stages of maturation. This evidence relies on data indicating that raised insulin levels result in increases in plasma GH (Rainey *et al.*, 1987) and insulin-like growth factor 1 (IGF-1). Both these growth factors, together with insulin, have been shown to have direct effects at the ovarian level in the pig (May and Schomberg, 1981; Britt, 1986; Jia *et al.*, 1986) including synergism with FSH in enhancement of gonadotrophin receptor development and augmentation of steroidogenesis in the developing follicle (Maruo *et al.*, 1988).

In summary flushing appears to raise ovulation rate by increasing plasma insulin and insulin-like growth factor 1 levels. Associated increases in the pulse frequency of GnRH, and thus plasma levels of gonadotrophins, enhance follicle recruitment and growth together with direct effects of insulin and IGF's at the ovarian level. These changes are diagrammatically presented in Figure 4.

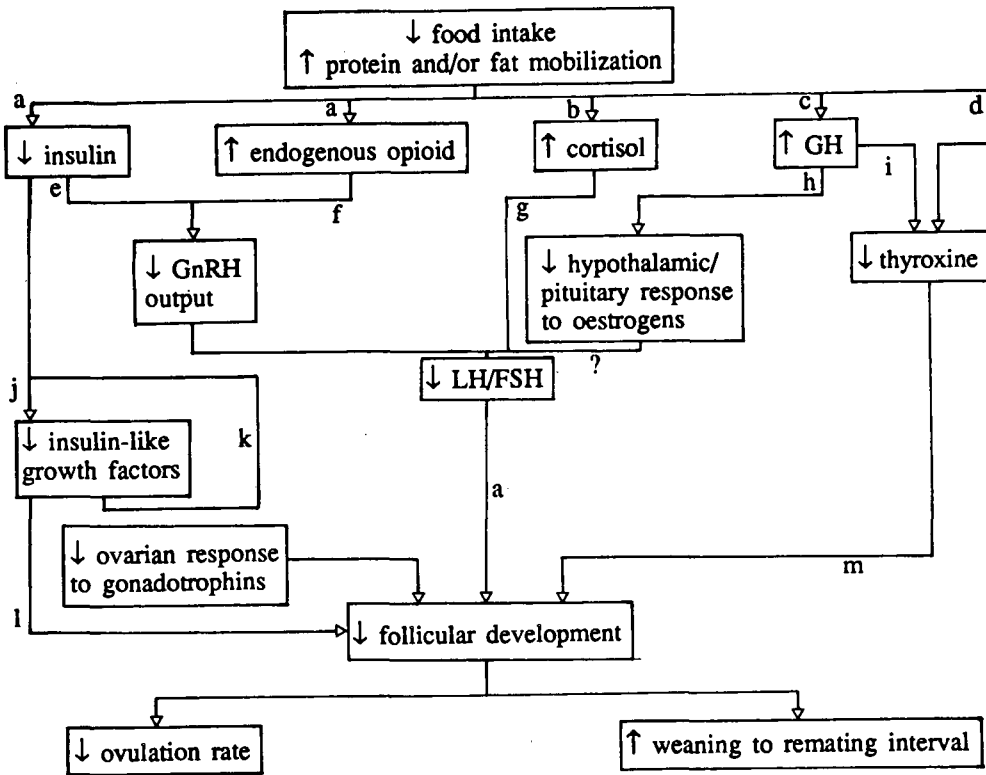


Figure 4. A theoretical model to explain the effects of nutrition on ovulation rate and the length of the weaning to remating interval in sows. (a - Armstrong and Britt, 1987; b - Steiner, 1987; c - McCusker *et al.*, 1985; d - Nelssen *et al.*, 1984; e - Cox *et al.*, 1987; f - Estienne *et al.*, 1988; g - Barb *et al.*, 1982; h - Kirkwood *et al.*, 1988; i - Kirkwood *et al.*, 1989; j - Kahn *et al.*, 1981; k - May and Schomberg, 1981; l - Baranao and Hammond, 1984; m - Maruo *et al.*, 1987; n - Maruo *et al.*, 1988.

Nutrition in early gestation and embryo survival

Table 2, presented in the introduction to this symposium, demonstrated that high plane feeding during early gestation may reduce the rate of embryo survival. Indeed, while approximately half the studies of this relationship have found no effect, the other studies clearly identified an adverse effect of high plane feeding on embryo survival.

The principal controlling mechanism in the development of embryos (and hence their survival) is the secretion of uterine specific proteins (USPs). These in turn are stimulated by the ovarian steroid hormones (Bazer *et al.*, 1982). In particular plasma progesterone levels play a crucial role in stimulating the release of USPs at the appropriate time for embryo support. It is therefore not surprising that altering the plasma levels of ovarian steroid hormones, and hence changing the pattern and level of USP secretions, has an adverse effect on embryo survival rate (Knight *et al.*, 1973).

This is important when we consider the report of Dyck *et al.* (1980) that increased gestation feed intake was associated with a decrease in plasma progesterone concentration and a reduction in embryo survival as shown in Table 10.

Table 10. The effect of feed level in gestation on plasma progesterone levels and embryo survival in sows (from: Dyck *et al.*, 1980)

Feed level (kg/day)	Embryo survival (%)	Mean plasma progesterone concentration (ng/ml)
1.50	82.8	16.7
2.25	78.6	13.8
3.00	71.9	11.8

More recently Grandhi (1988) has reported reduced levels of plasma progesterone and embryo survival in animals fed diets supplemented with fat during gestation. This same author reported no effect of increased lysine levels in gestation on embryo survival. The mechanism underlying the reduction in plasma progesterone levels associated with increased feed or energy intake in early gestation remains to be fully elucidated. However, we present below a possible mechanism whereby high plane feeding in early gestation may adversely influence embryo survival rate (Figure 5).

There is no doubt that high plane feeding during early gestation will result in more rapid live weight and condition gain in sows than does lower plane feeding. Recent studies have also suggested the hypothesis that this increased live weight, or condition, gain will increase both hepatic blood flow and the metabolic clearance rate of progesterone in the pig (Henry, Pickard and Hughes - unpublished data; Symonds and Prime, 1989). Thus, we suggest that any nutritional regime that causes rapid live weight and condition gains in gilts/sows during early gestation is likely to result in reduced embryo survival rates as a consequence of increased hepatic blood flow increasing the MCR of progesterone and hence adversely influencing the secretion of USPs.

Furthermore, it is possible that administration of exogenous progesterone during early gestation may improve embryo survival in sows being fed on a high plane of nutrition, as has been demonstrated in sheep (Parr *et al.*, 1987).

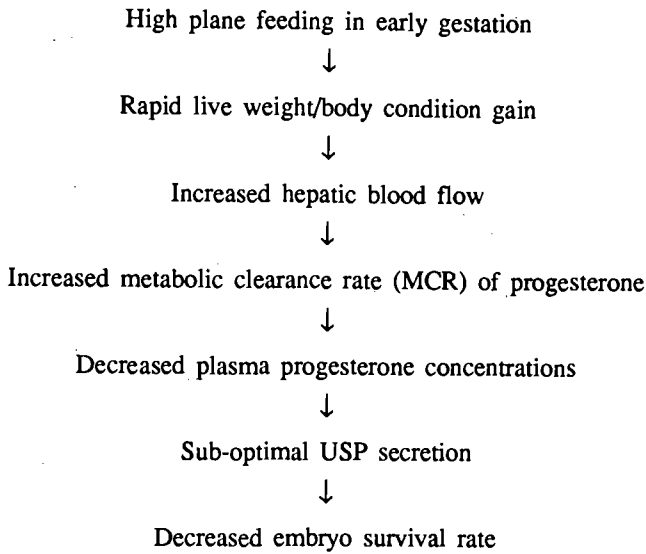


Figure 5. A theoretical model to describe the relationship between nutrition in early gestation and embryo survival rate.

Lactation feeding and subsequent reproduction

During early lactation the sow is normally anoestrous, this being mainly due to suppression of the GnRH pulse generator (Britt *et al.*, 1985). This results in low circulating levels of gonadotrophins and hence minimal follicular development. However, as lactation proceeds a gradual escape from the inhibitory influences of suckling, elevated prolactin levels, and ovarian factors (probably inhibin) begins and the plasma level of FSH and the pulse frequency of LH increases (Stevenson *et al.*, 1981; Britt *et al.*, 1985). This causes an increase in follicular growth in later lactation (Kunavongkrit *et al.*, 1982).

Post-weaning the activity of the GnRH pulse generator increases and the pulse frequency of plasma LH rises rapidly (Cox and Britt 1982a,b; Shaw and Foxcroft, 1985). A concurrent, but smaller, rise also occurs in plasma FSH levels (Shaw and Foxcroft, 1985). These changes induce rapid growth of "selected" follicles and a corresponding depletion of small-sized follicles (Britt *et al.*, 1985). The final result of these alterations in plasma gonadotrophin levels and follicular growth is the appearance of the post-weaning oestrus accompanied by ovulation.

In the case of the sow that is underfed during lactation alterations must occur to the above pattern since the weaning to remating interval is usually delayed (reviewed by Aherne and Kirkwood, 1985). This appears to be particularly true in those sows that have received inadequate levels of dietary protein and have hence had to deplete body protein reserves (Brendemuhl *et al.*, 1987).

Since feeding during the weaning to remating interval was standardized in most of these studies (see papers 1 and 2 in this symposium) this implies that the nutritional effect of lactation feeding on the length of the weaning to remating interval is either (a) due to effects on the follicle population pre-weaning, or (b) an indirect effect operating via the hypothalamic-pituitary-ovarian axis post-weaning. These effects, in turn, may be due to either the dynamic change in body tissues that occurs during lactation or the resultant body weight/condition at the time of weaning.

Nutritional effects during lactation on FSH secretion have not, to our know-

ledge, been investigated in the pig. However, several reports suggest that mean plasma LH level and the pulse frequency of LH secretion are decreased in sows underfed during lactation (Kirkwood *et al.*, 1987a; Mullan and Close, 1989). Additionally, King and Martin (1989) have reported a significant decrease in mean plasma LH level and a non-significant decrease in LH pulse frequency in first litter sows fed a low protein diet during lactation. The data on LH pulse frequencies are particularly interesting in view of the reports of Armstrong and Britt (1984), Armstrong *et al.* (1986) and King and Martin (1989) that the weaning to remating interval was shortest in those sows with the highest LH pulse frequency pre-weaning. Thus, it might be postulated that, since LH pulses are critical in the stimulation of follicular growth during lactation, under-feeding the lactating sow extends the weaning to remating interval via an adverse effect on LH pulse frequency and hence follicle development at weaning.

If this is indeed the case then what mechanism controls the observed changes in LH pulse frequency in response to dietary intake? Although the mechanism linking dietary intake, body condition and metabolic status with reproductive activity is poorly understood, several changes which accompany reduced food intake and increased protein or fat mobilization may be involved and possible inter-relationships between these are illustrated in Figure 4. A central role of reduced insulin levels in underfed animals was suggested by the work of Armstrong and Britt (1987) in which nutritionally induced anoestrus was associated with low basal insulin and resumption of oestrous cyclicity was accompanied by raised levels of insulin. As mentioned earlier, insulin may enhance the activity of the GnRH pulse generator (Cox *et al.*, 1987) and enhance ovarian development directly (May and Schomberg, 1981) or through insulin-like growth factors (Baranao and Hammond, 1984). Increased plasma levels of cortisol (Steiner, 1987) and growth hormone (McCusker *et al.*, 1985) are also seen in underfed animals and have been shown to inhibit the activity of the hypothalamic-pituitary axis at various levels (see Figure 4). Hypothyroidism has been observed in sows fed restricted energy diets in lactation (Nelssen *et al.*, 1984; Brendemuhl *et al.*, 1987) and may inhibit follicular development in pigs (Maruo *et al.*, 1987). Thus there are several areas through which metabolic changes associated with variations in food intake and/or body tissue mobilization can affect follicular development and hence resumption of rebreeding after weaning.

These changes in gonadotrophic secretion will undoubtedly influence the average follicle size of the sow at weaning. However, this may not necessarily influence the length of the weaning to remating interval (see Cox and Britt, 1983). If this is, indeed, the case then other possible routes for lactation feeding to influence the length of the weaning to remating interval must be considered. Two possible pathways are apparent, these being (a) the live weight and body condition of low fed sows will be lower at weaning, and (b) lactation feeding indirectly affects either gonadotrophin secretion post-weaning or the sensitivity of the ovary to gonadotrophic stimulation.

Little is currently known about the relationship between body weight or condition *per se* and the function of the hypothalamic-pituitary-ovarian axis. However, recent work in sheep suggests that animals in good body condition exhibit heightened GnRH pulse generator activity (Rhind *et al.*, 1989) and higher FSH levels (Rhind *et al.*, 1986) relative to their poorer condition counterparts. It is thus possible that the reduced body weight and condition of sows at weaning following low lactation feeding may result in a reduced gonadotrophin release post-weaning. Evidence to support this possibility in the pig is accruing from recent work in Western Australia (Pearce, Paterson, Williams, Urquhart and Pearce, unpublished data), which has shown that the percentage of farrowing weight lost in lactation is correlated with the length of the remating interval ($r=0.48$, $p=0.058$) and early (<13

days) returning first litter sows show greater LH pulsatility on the day of weaning (2.2 pulses/7 h) and the day after weaning (3.8 pulses/7 h) compared to animals showing extended weaning to remating intervals (>32 days) (1.3 and 1.7 pulses/7 h on day of weaning and day after respectively). The inter-relationship of these differences with metabolic hormones is at present being investigated.

Alternatively, it has been suggested by Kirkwood *et al.* (1987a) that the ovaries of sows that have been underfed during lactation are less sensitive to gonadotrophic stimulation than those of high fed sows. This may be due to a lack of sufficient plasma insulin during lactation in low fed sows to fully stimulate either follicular gonadotrophin receptors (Maruo *et al.*, 1988) or ovarian steroidogenesis (Barbieri *et al.*, 1983). The final possibility is that underfeeding during lactation has an indirect influence on GnRH/gonadotrophin secretion post-weaning. Little evidence is currently available in this area, although early weaning has been reported to result in reduced plasma gonadotrophin levels post-weaning (Edwards and Foxcroft, 1983; Kirkwood *et al.*, 1984a,b). Since early weaning involves removal of piglets at a time before the normal "escape" from suckling inhibition on gonadotrophin secretion has begun, this may be an analogous situation to that of the underfed lactating sow.

As described in the first paper in this symposium the adverse effects on weaning to remating interval are not translated into differences in ovulation rate at the post-weaning oestrus. While the reasons for this have not been studied, it may be suggested that this is due to longer weaning to remating intervals allowing an extended flushing period in the low lactation fed sows.

However, several authors have reported adverse effects of underfeeding during lactation on subsequent embryo survival and litter size (see the first paper in this symposium). These differences have only been reported in parity 2-6 sows and not in first parity sows.

The effects of low lactation feeding on embryo survival has been suggested to be the result of sub-optimal LH release at the post-weaning oestrus (Aherne and Kirkwood, 1985) resulting in inadequate luteinization of the corpus luteum (Smith, 1986) with concomitant low plasma progesterone levels in early gestation. Low lactation feeding has indeed been reported to reduce plasma progesterone levels in early pregnancy (Hughes *et al.*, 1984; Kirkwood *et al.*, 1987a) indicating that embryo survival may be affected by a mechanism similar to that outlined in Figure 5. Support for this possibility comes from the observation that injection of GnRH at the post-weaning oestrus improved embryo survival and increased progesterone levels in early gestation in animals fed low levels in lactation (Kirkwood *et al.*, 1987a).

In summary it is proposed that the influences of nutritional variations and concomitant changes in body tissue mobilization affect reproduction in the female pig through one or two mechanisms outlined in Figures 4 and 5. Although these mechanisms explain experimental observations made to date, their confirmation must await further detailed investigation of the endocrinology of nutrition-reproduction interactions in the sow.

NUTRITION REPRODUCTION INTERACTIONS IN THE BREEDING SOW: CONCLUSIONS

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It is clear from the data presented in the three symposium papers that our knowledge of the nutrition of the breeding sows and, more particularly the endocrinology of nutrition-reproduced interactions, has increased markedly in recent years. Nevertheless, many of the crucial interactions between nutrition and reproduction are still incompletely understood. It is therefore pertinent at this stage to summarize (1) what we do know, (2) what we need to find out, and (3) the practical implications of our current knowledge.

The use of flushing to increase ovulation rate has become a commonly-used tool in commercial pig production. It clearly does raise ovulation rates in most circumstances, this probably being achieved via increased insulin and IGF-1 levels as described earlier. In practice the value of raised ovulation rates is questionable in the mature sow (Hughes and Varley, 1980), although suboptimal ovulation rates are likely to limit litter size in the first and second parity animal (see Dyck, 1974; Hughes and Varley, 1980; Love, 1979; Walker *et al.*, 1979). Thus, it is suggested that gilts are fed at least 3 kg/day of a standard grower/finisher diet for at least 10 days prior to mating at second oestrus. In the case of the weaned sow it would seem wise to feed 3 kg/day or more of a lactating sow diet for the duration of the weaning to remating interval. While such a regime may be of little reproductive benefit to the mature sow it is likely to raise litter size in the second parity animal.

Once mated the average gilt/sow loses 20-40% of fertilized ova within the first 2-3 weeks of gestation (Hughes and Varley, 1980). This loss appears to be increased in many cases by overfeeding at this stage. It is suggested that this is due to high energy/high plane feeding reducing plasma progesterone levels and thus adversely influencing uterine specific protein secretion. What remains to be determined is the reason why overfeeding has an adverse effect in some animals but little or no effect in others. Equally, the data presented suggests that the possibility of reversing the effects of overfeeding in early gestation by supplying exogenous ovarian steroid hormones should be examined. Currently, it is suggested that pregnant gilts/sows be fed a "dry sow" diet at a rate no greater than 2 kg/day for the first 2-4 weeks of gestation. Subsequently, feed level appears to have little effect on litter size, although higher plane feeding will undoubtedly raise piglet birth weights (Anderson and Melampy, 1972) and reduce the sows appetite in the following lactation. In the practical situation the adverse effects on voluntary food intake in lactation are likely to be of greater importance than the improvement in piglet birth weight achieved, and hence it is suggested that the sow be fed in the range 1.8-2.2 kg/day of a "dry sow" diet during mid and late gestation. However, if neonatal mortality of piglets is high it may be worthwhile to add a fat supplement to the sows diet at the rate of 200-250 g/day for the last 2-3 weeks of gestation (Moser and Lewis, 1981).

It is clear from the above that achievement of the optimum nutrient intakes by gilts/sows pre-mating and during gestation is relatively easy. Unfortunately this is not the case in the lactating sow. There is no doubt that underfeeding during lactation results in an extension of the weaning to remating interval and may reduce subsequent litter size. The mechanism(s) whereby these effects are brought about have been discussed in detail in the preceding papers. It would appear that underfeeding

results in endocrine changes that may be considered the reverse of flushing (see Figure 4), thus resulting in extended periods from weaning to oestrus. Adverse effects of underfeeding during lactation on subsequent embryo survival rates (and hence litter size) are probably due to a reduction in plasma progesterone levels, as reported earlier for sows overfed in early gestation, although the cause of lowered progesterone levels is likely to be different. The signal(s) that initiate these adverse effects as a result of underfeeding the lactating sow are not well understood. Essentially, tissue mobilization, and its consequent metabolic hormone changes, appears to be the vital cue for alterations to the hypothalamic-pituitary-ovarian axis. However, the roles of live weight and individual tissue changes, and indeed the rate of change in these tissues, remains to be fully elucidated. What is clear is that animals undergoing rapid mobilization of body tissue during lactation are likely to be reproductively inadequate post-weaning. Whether live weight *per se* at weaning is important is yet to be clarified, since most low live weight sows at weaning will also be those sows that have lost most live weight during the preceding lactation. It is therefore suggested that further studies are needed in this area, in particular concentrating on the link between the endocrine status of the animal and nutrient intake, body weight and composition, and rates of tissue change. In addition, apparent differences in response between first and subsequent parity sows need to be further investigated. Lastly the actual levels of intake of both protein and energy must be more closely studied in view of the relatively low levels achieved in most studies in the "high" treatment groups. Interpreting the above summary into practical recommendations there is no doubt that the primary objective in feeding the lactating sow is to maximize food intake. Most studies suggest that energy intakes of 75-100 MJ of DE/day are needed by the sow, this interpreting to 6 kg/day or more of a conventional lactating sow diet. To achieve this with current sow genotypes it is necessary to feed several times/day to appetite. Additionally, gestation food intake must be held low (indeed, it seems likely that a feeding regime designed to minimize fluctuations in the body condition of the sow is ideal), high temperatures must be avoided, high density lactating sow diets should be fed, and the fat content of the sow at farrowing should be controlled.

References

- AGRICULTURAL RESEARCH COUNCIL (1981). "Nutrient Requirements of Pigs" (Commonwealth Agricultural Bureau: Slough, UK).
- AHERNE, F.X. and KIRKWOOD, R.N. (1985). Nutrition and sow prolificacy. *Journal of Reproduction and Fertility*. Supplement 33:169-183.
- ANDERSON, L.L. and MELAMPY, R.M. (1972). Factors affecting ovulation rate in the pig. In "Pig Production", ed. D.J.A. Cole (Butterworths: London).
- ARMSTRONG, J.D. and BRITT, J.H. (1984). Effect of energy restriction during lactation on reproductive performance, energy metabolites and endocrine changes in primiparous sows. (Proceedings of 10th International Congress of Animal Reproduction and Artificial Insemination: Urbana II). Abstract 157.
- ARMSTRONG, J.D. and BRITT, J.H. (1987). Nutritionally-induced anestrus in gilts: Metabolic and endocrine changes associated with cessation and resumption of estrous cycles. *Journal of Animal Science*. 65:508-523.
- ARMSTRONG, J.D., BRITT, J.H., and KRAELING, R.R. (1986). Effect of restriction of energy during lactation on body condition, energy metabolism, endocrine changes and reproductive performance in primiparous sows. *Journal of Animal Science*. 63:1915-1925.
- BAIRD, D.T. (1987). A model for follicular selection and ovulation: Lessons from superovulation. *Journal of Steroid Biochemistry*. 27:15-23.
- BARANAO, J.L.S. and HAMMOND, J.M. (1984). Comparative effects of insulin and insulin-like growth factors on DNA synthesis and differentiation of porcine granulosa cells. *Biochemical and Biophysical Research Communications*. 124:484-490.
- BARB, C.R., KRAELING, R.R., RAMPACEK, G.B., FONDA, E.S. and KISER, T.E. (1982). Inhibition of ovulation and LH secretion in the gilt after treatment with ACTH or hydrocortisone. *Journal of Reproduction and Fertility*. 64:85-92.

- BARBIERI, R.L., MAKRIS, A. and RYAN, K.J. (1983). Effects of insulin on steroidogenesis in cultured porcine ovarian theca. *Fertility and Sterility*. **40**:237-241.
- BAZER, F.W., GEISERT, R.D., THATCHER, W.W. and ROBERTS, R.M. (1982). The establishment and maintenance of pregnancy. In "Control of Pig Reproduction", eds. D.J.A. Cole and G.R. Foxcroft (Butterworths: London).
- BLACK, J.L., CAMPBELL, R.G., WILLIAMS, I.H., JAMES, K.J. and DAVIES, G.T. (1986). Stimulation of energy and amino acid utilization in the pig. *Research and Development in Agriculture*. **3**:121-145.
- BRENDEMUHL, J.H., LEWIS, A.J. and PEO, E.R. Jr. (1987). Effect of protein and energy intake by primiparous sows during lactation on sow and litter performance and sow serum thyroxine and urea concentrations. *Journal of Animal Science*. **64**:1060-1069.
- BRITT, J.H. (1986). Improving sow productivity through management during gestation, lactation and after weaning. *Journal of Animal Science*. **63**:1288-1296.
- BRITT, J.H., ARMSTRONG, J.H., COX, N.M. and ESBENSHADE, K.L. (1985). Control of follicular development during and after lactation in sows. *Journal of Reproduction and Fertility*. Supplement **33**:37-54.
- BROOKS, P.H. (1982). The gilt for breeding and for meat. In "Control of Pig Reproduction", eds. D.J.A. Cole and G.R. Foxcroft (Butterworths: London).
- CLARK, I.J. and CUMMINS, J.T. (1982). The temporal relationship between gonadotropin releasing hormone (GnRH) and luteinizing hormone (LH) secretion in ovariectomised ewes. *Endocrinology*. **111**:1737-1739.
- CLOSE, W.H., NOBLET, J. and HEAVENS, R.P. (1985). Studies on the energy metabolism of the pregnant sow. 2. The partition and utilization of metabolizable energy intake in pregnant and non-pregnant animals. *British Journal of Nutrition*. **53**:267-279.
- COLE, D.J.A. (1982). Nutrition and reproduction. In "Control of Pig Reproduction", eds. D.J.A. Cole and G.R. Foxcroft (Butterworths: London).
- COLE, D.J.A. (1989). Nutritional strategies to optimise reproduction. In "Control of Pig Reproduction III", eds. D.J.A. Cole, G.R. Foxcroft and B.J. Weir (Butterworths: London).
- COLE, D.J.A. and CHADD, S.A. (1989). In "Voluntary Feed Intake of Pigs", eds. M.A. Varley and J.M. Forbes, (Occasional Publication number 13, British Society of Animal Production), (in press).
- COLE, D.J.A., DUCKWORTH, J.E. and HOLMES, W. (1967). Factors affecting feed intake in pigs. 1. The effect of digestible energy content of the diet on intake of castrated male pigs housed in holding pens and metabolism crates. *Animal Production*. **9**:141-148.
- COLE, D.J.A., HARDY, B. and LEWIS, D. (1972). Nutrient density of pig diets. In "Pig Production", ed. D.J.A. Cole (Butterworths: London).
- COX, N.M. and BRITT, J.H. (1982a). Relationship between endogenous gonadotrophin-releasing hormone, gonadotrophins and follicular development after weaning in sows. *Biology of Reproduction*. **27**:70-78.
- COX, N.M. and BRITT, J.H. (1982b). Pulsatile administration of gonadotrophin-releasing hormone to lactating sows: Endocrine changes associated with induction of fertile estrus. *Biology of Reproduction*. **27**:1126-1137.
- COX, N.M. and BRITT, J.H. (1983). Post-weaning estrus in sows is not prevented by electrocauterization of ovarian follicles before weaning. *Journal of Animal Science*. **57** (Supplement 1):34.
- COX, N.M., STUART, M.J., ALTHEN, T.G., BENNETT, W.A. and MILLER, H.W. (1987). Enhancement of ovulation rate in gilts by increasing dietary energy and administering insulin during follicular growth. *Journal of Animal Science*. **64**:507-516.
- DAILEY, R.A., CLARK, J.R., FIRST, N.L., CHAPMAN, A.B. and CASIDA, L.E. (1975). Loss of follicles during the follicular phase of the estrous cycles of swine as affected by genetic group and level of feed intake. *Journal of Animal Science*. **42**:835-841.
- DYCK, G.W. (1974). The effects of stage of pregnancy, mating at first and second estrus after weaning and level of feeding on fetal survival in sows. *Canadian Journal of Animal Science*. **54**:277-285.
- DYCK, G.W., PALMER, W.M. and SIMARAKS, S. (1980). Progesterone and luteinizing hormone concentrations in serum of pregnant gilts on different levels of feed consumption. *Canadian Journal of Animal Science*. **60**:877-884.
- EDWARDS, S. and FOXCROFT, G.R. (1983). Endocrine changes in sows weaned at two stages of lactation. *Journal of Reproduction and Fertility*. **67**:161-172.
- ESTIENNE, M.J., BARB, C.R., KESNER, J.S., KRAELING, R.R. and RAMPACEK, G.B. (1988). Gonadotrophin and prolactin secretion following intraventricular (IVT) administration of morphine in gilts. *Journal of Animal Science*. **66** (Supplement 1):408.

- FLOWERS, B., MARTIN, M.J., CANTLEY, T.C. and DAY, B.N. (1989). Endocrine changes associated with a dietary-induced increase in ovulation rate (flushing) in gilts. *Journal of Animal Science*. **67**:771-778.
- GRANDHI, R.R. (1988). Effects of nutritional flushing, supplemental fat and supplemental lysine from puberty to breeding and during early gestation on reproductive performance of gilts. *Canadian Journal of Animal Science*. **68**:941-951.
- HARDY, B. and LODGE, G.A. (1969). The effect of body composition on ovulation rate in the sow. *Animal Production*. **11**:505-510.
- HENRY, R.W., PICKARD, D.W. and HUGHES, P.E. (1984). The effects of lactation length and food level on subsequent reproductive performance in the sow. *Animal Production*. **38**:527.
- HUGHES, P.E., HENRY, R.W. and PICKARD, D.W. (1984). The effects of lactation food level on subsequent ovulation rate and early embryonic survival in the sow. *Animal Production*. **38**:527.
- HUGHES, P.E. and VARLEY, M.A. (1980). "Reproduction in the Pig" (Butterworths: London).
- JIA, X.C., KALMIJN, J. and HSUEH, A.J.W. (1986). Growth hormone enhances follicle-stimulating hormone-induced differentiation of cultured rat granulosa cells. *Endocrinology*. **118**:1401-1409.
- JOHNSON, L.J., ORR, D.E.Jr., 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.
- KAHN, C.R., BAIRD, K.L., FLIER, J.S., GRUNFELD, C., HARMAN, J.T., HARRISON, L.C., KARLSSON, F.A., KASUGA, M., KING, J.L., LANG, W.C., PODSKALNY, J.M. and VAN OBBERGHEN, E. (1981). Insulin receptors, receptor antibodies and the mechanism of insulin action. *Recent Progress in Hormone Research*. **37**:477-538.
- KING, R.H. (1987). Nutritional anoestrus in young sows. *Pig News and Information*. **8**:15-21.
- KING, R.H. and DUNKIN, A.C. (1986a). 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. and DUNKIN, A.C. (1986b). The effect of nutrition on the reproductive performance of first-litter sows. 4. The relative effects of energy and protein intakes during lactation on the performance of sows and their piglets. *Animal Production*. **43**:319-325.
- KING, R.H. and MARTIN, G.B. (1989). Relationships between protein intake during lactation, LH levels and oestrous activity in first-litter sows. *Animal Reproduction Science*. **19**:283-292.
- 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.
- KING, R.H., WILLIAMS, I.H. and BARKER, I. (1984). The effect of diet during lactation on the reproductive performance of first-litter sows. *Proceedings of Australian Society of Animal Production*. **15**:412-415.
- KIRKWOOD, R.N., BAIDOO, S.K., AHERNE, F.X. and SATHER, A.P. (1987b). The influence of feeding level during lactation on the occurrence and endocrinology of the post-weaning oestrous in sows. *Canadian Journal of Animal Science*. **67**:405-415.
- KIRKWOOD, R.N., LAPWOOD, K.R., SMITH, W.C. and ANDERSON, I.L. (1984a). Plasma concentrations of LH, prolactin, oestradiol-17 β and progesterone in sows weaned after lactating for 10 or 35 days. *Journal of Reproduction and Fertility*. **70**:95-102.
- KIRKWOOD, R.N., LAPWOOD, K.R., SMITH, W.C., MOLLER, K. and GARRICK, D.J. (1984b). Effects of oestradiol benzoate treatment on the reproductive performance and endocrine status of sows after lactations of 10 or 35 days. *Journal of Reproduction and Fertility*. **72**:329-337.
- KIRKWOOD, R.N., LYTHGOE, E.S. and AHERNE, F.X. (1987a). Effect of lactation feed intake and gonadotrophin-releasing hormone on the reproductive performance of sows. *Canadian Journal of Animal Science*. **67**:715-719.
- KIRKWOOD, R.N., MITARU, B.N., GOONERATNE, A.D., BLAIR, R. and THACKER, P.A. (1988). The influence of dietary energy intake during successive lactations on sow prolificacy. *Canadian Journal of Animal Science*. **68**:283-290.
- KIRKWOOD, R.N., THACKER, P.A. and LAARVELD, B. (1989). The influence of growth hormone injections on the endocrine and metabolic status of gilts. *Domestic Animal Endocrinology*. **6**:167-176.
- KNIGHT, J.W., BAZER, F.W. and WALLACE, H.D. (1973). Hormonal regulation of porcine uterine protein secretion. *Journal of Animal Science*. **36**:546-553.
- KUNAVONGKRIT, A., EINARSSON, S. and SETTERGREN, I. (1982). Follicular development in primiparous lactating sows. *Animal Reproduction Science*. **5**:47-56.

- LANDAU, B.R., TAKAOKI, Y., ABRAMS, M.A., GENUTH, S.M., VAN HOUTEN, M., POSSNER, B.I., WHITE, R.J., OHGAKU, S., HORVAT, A. and HEMMELGARN, E. (1983). Binding of insulin by monkey and pig hypothalamus. *Diabetes*. **32**:291-294.
- LOVE, R.J. (1979). Reproductive performance of first parity sows. *Veterinary Record*. **104**:238-240.
- LYNCH, P.B. (1989). In "Voluntary Feed Intake of Pigs", eds. M.A. Varley and J.M. Forbes. Occasional Publication No. 13, British Society of Animal Production (in press).
- MCCUSKER, R.H., WANGSNESS, P.J., GRIEL, L.C. and KAVANAUGH, J.F. (1985). Effects of feeding, fasting and refeeding on growth hormone and insulin in obese pigs. *Physiology and Behaviour*. **35**:383-388.
- MAHAN, D.C. and MANGAN, L.T. (1975). Evaluation of various protein sequences on the nutritional carry-over from gestation to lactation with first-litter sows. *Journal of Nutrition*. **105**:1291-1295.
- MARUO, J., HAYASHI, M., MATSUO, H., UEDA, Y., MORIKAWA, M. and MOCHIZUKI, M. (1988). Comparison of the facilitative roles of insulin and insulin-like growth factor 1 in the functional differentiation of granulosa cells: *In vitro* studies with the porcine model. *Acta Endocrinologica Copenhagen*. **117**:230-240.
- MARUO, T., HAYASHI, M., MATSUO, H., YAMAMOTO, T., OKADA, H. and MOCHIZUKI, M. (1987). The role of thyroid hormone as a biological amplifier of the actions of follicle-stimulating hormone in the functional differentiation of cultured porcine granulosa cells. *Endocrinology*. **121**:1233-1241.
- MAY, J.V. and SCHOMBERG, D.W. (1981). Granulosa cell differentiation *in vitro*: Effect of insulin on growth and functional integrity. *Biology of Reproduction*. **25**:421-431.
- MOSER, B.D. and LEWIS, A.J. (1981). Fat additives to sow diets - A review. *Pig News and Information*. **2**:265-269.
- MULLAN, B.P. and CLOSE, W.H. (1989). The partition of nutrients during lactation and its relationship to reproductive performance. In "Manipulating Pig Production II", p. 302, eds. J.L. Barnett and D.P. Hennessy (Australasian Pig Science Association: Werribee, Australia).
- MULLAN, B.P., CLOSE, W.H. and COLE, D.J.A. (1989). In "Recent Advances in Animal Nutrition - 1989" eds. W. Haresign and D.J.A. Cole (Butterworths: London) (in press).
- MULLAN, B.P. and WILLIAMS, I.H. (1989). *Animal Production*. **48**:449-457.
- NELSEN, J.L., LEWIS, A.J., PEO, E.R.Jr., KITTOK, R.J., KINDER, J.E., ZIMMERMAN, D.R., JOHNSON, R.K. and CRENSHAW, J.D. (1984). Effect of dietary energy intake of sows during lactation on their post-weaning hormone profiles. *Journal of Animal Science*. **59** (Supplement 1):247.
- PARR, R.A., DAVIS, I.F., FAIRCLOUGH, R.J. and MILES, M.A. (1987). Overfeeding during early pregnancy reduces peripheral progesterone concentration and pregnancy rate in sheep. *Journal of Reproduction and Fertility*. **80**:317-320.
- PRIME, G.R., VARLEY, M.A. and SYMONDS, H.W. (1988). The effect of food intake during lactation and early pregnancy on plasma progesterone concentrations and prolificacy in multiparous sows. *Animal Production*. **46**:499.
- RAINEY, M.R., SCHNELLER, H.E. and COX, N.M. (1987). Insulin administration increased ovulation rate, estradiol and growth hormone and changed the course of post-ovulatory progesterone increase in gilts. *Journal of Animal Science*. **65** (Supplement 1):420.
- REESE, D.E., MOSER, B.D., PEO, E.R.Jr., LEWIS, A.J., ZIMMERMAN, D.R., KINDER, J.E. and STROUP, W.W. (1982). Influence of energy intake during lactation on subsequent gestation, lactation and post-weaning performance of sows. *Journal of Animal Science*. **55**:867-872.
- REESE, D.E., PEO, E.R.Jr. and LEWIS, A.J. (1984). Relationship of lactation energy intake and occurrence of post-weaning oestrus to body and backfat composition in sows. *Journal of Animal Science*. **58**:1236-1244.
- RHIND, S.M., LESLIE, I.D., GUNN, R.G. and DONEY, J.M. (1986). Effects of high levels of body condition and food intake on plasma follicle stimulating hormone, luteinizing hormone, prolactin and progesterone profiles around mating in Greyface ewes. *Animal Production*. **43**:101-107.
- RHIND, S.M., McMILLEN, S., McKELVEY, W.A.C. RODRIGUEZ-HERREJON, F.F. and McNEILLY, A.S. (1989). Effect of the body condition of ewes on the secretion of LH and FSH and the pituitary response to gonadotrophin-releasing hormone. *Journal of Endocrinology*. **120**:497-502.
- RHODES, M.T., MINTON, J.E., STEVENSON, J.S. and DAVIS, D.L. (1987). Endocrine changes associated with flushing and altrenogest treatment in gilts. *Journal of Animal Science*. **65**:154
- SALMON-LEGAGNEUR, E. and RERAT, A. (1962). In "Nutrition of Pigs and Poultry" eds. J.T. Morgan and D. Lewis (Butterworths: London).
- SHAW, H.J. and FOXCROFT, G.R. (1985). Relationships between LH, FSH and prolactin secretion and reproductive activity in the weaned sow. *Journal of Reproduction and Fertility*. **75**:17-28.

- SMITH, M.F. (1986). Recent advances in corpus luteum physiology. *Journal of Dairy Science*. **69**:911-926.
- STANSBURY, W.F., McGLONE, J.J. and TRIBBLE, L.F. (1987). Effects of season, floor type, air temperature and snout coolers on sow and litter performance. *Journal of Animal Science*. **65**:1507-1513.
- STEINER, R.A. (1987). Nutritional and metabolic factors in the regulation of reproductive hormone secretion in the primate. *Proceedings of the Nutrition Society*. **46**:159-175.
- STEVENSON, J.S., COX, N.M. and BRITT, J.H. (1981). Role of the ovary in controlling luteinizing hormone, follicle stimulating hormone and prolactin secretion during and after lactation in pigs. *Biology of Reproduction*. **24**:341-353.
- SYMONDS, H.W. and PRIME, G.R. (1989). Influence of volume of food intake on blood flow in the portal vein and the clearance of progesterone from plasma in gilts. *Proceedings of the British Society of Animal Production*. Paper number 13.
- Van HOUTEN, M., NANCE, D.M., GAUTHER, S. and POSNER, B. (1983). Origin of insulin-receptive nerve terminals in rat median eminence. *Endocrinology*. **113**:1393-1398.
- WALKER, N., WATT, D., MACLEOD, A.S., JOHNSON, C.L., BOAZ, T.G. and CALDER, A.F.C. (1979). The effect of weaning at 10, 25 or 40 days on the reproductive performance of sows from the first to the fifth parity. *Journal of Agricultural Science, Cambridge*. **92**:449-456.
- YANG, H., EASTHAM, P.R., PHILLIPS, P. and WHITTEMORE, C.T. (1989). Reproductive performance, body weight and body condition of breeding sows with differing body fatness at parturition, differing nutrition during lactation, and differing litter size. *Animal Production*. **48**:181-201.
- ZOIOPOULUS, P.E. (1978). "Evaluation of Fibrous Ingredients in Diets for Growing and Breeding Pigs (Doctor of Philosophy Thesis, University of Aberdeen: Scotland).

THE PARTITION OF NUTRIENTS DURING LACTATION AND ITS RELATIONSHIP TO REPRODUCTIVE PERFORMANCE

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When the food intake of sows during lactation is insufficient to meet the metabolic requirements of milk production, body reserves are mobilized and this may result in reproductive failure (Mullan and Williams, 1989). The aim of this experiment was to manipulate the use of body reserves by sows during their first lactation in an attempt to investigate how the dynamics of nutrient metabolism may influence reproductive function.

In a 2x2 factorial experiment 28 Landrace x Large White gilts were fed either to appetite (5.5 kg/day) (H) or 3.0 kg/day (L) with litter size adjusted to either six (6) or 12 (12) at each feeding level for a 21-day lactation. Energy and nitrogen balances were conducted and the changes in fat and lean content were calculated accordingly. Blood samples were drawn at 15 min intervals for 12 h via indwelling jugular cannulae.

Energy and nitrogen balance of the sow increased with the increase in feed intake and with the decrease in litter size (Table 1). Over all treatments those sows with a weaning-to-mating interval greater than 7 days had a lower ($P < 0.05$) concentration of LH in the 12 h prior to weaning than those that were mated within 7 days of weaning (0.203 vs 0.318 ng/ml, respectively).

Table 1. Energy and nitrogen balance of sows during lactation (day 5-21) and plasma LH levels at three stages of lactation

	H-6	H-12	L-6	L-12	SEM
Number of animals	7	7	7	7	
Intake - energy (MJ ME/day)	62.0 ^y	66.3 ^y	33.1 ^x	35.2 ^x	2.28
- nitrogen (g/day)	146.9 ^y	147.3 ^y	75.2 ^x	78.2 ^x	5.02
Milk - energy (MJ ME/day)	39.0 ^a	53.6 ^b	35.2 ^a	47.8 ^c	2.38
- nitrogen (g/day)	52.7 ^a	74.5 ^b	47.5 ^a	66.6 ^c	3.13
Balance - energy (MJ ME/day)	+0.6 ^w	-7.8 ^x	-22.9 ^y	-32.9 ^z	2.61
- nitrogen (g/day)	+12.0 ^x	-11.0 ^y	-13.2 ^y	-23.6 ^z	3.75
- fat (g/day)	-94 ^x	-133 ^y	-452 ^y	-629 ^z	46.1
- lean (g/day)	+446 ^x	-242 ^y	-375 ^y	-672 ^z	93.5
Mean plasma LH (ng/ml)					
- day 10	0.309	0.242	0.212	0.613	0.0562
- day 17	0.303	0.440	0.343	0.275	0.0818
- pre-weaning	0.304 ^a	0.205 ^b	0.382 ^a	0.199 ^b	0.0474
- post-weaning	0.570 ^{ab}	0.489 ^{bc}	0.807 ^a	0.281 ^c	0.1326

^{a,b,c}differ at $P < 0.05$; ^{w,x,y,z}differ at $P < 0.01$

The results of this experiment highlight the degree to which sows can mobilize reserves of body fat and body lean to meet the nutritional requirement of milk production. There are also indications that the metabolic state of the sow influenced plasma LH concentration and hence reproductive function.

References

MULLAN, B.P. and WILLIAMS, I.H. (1989). *Animal Production*. 48:449-457.

IMMUNIZATION AGAINST THE α SUBUNIT OF BOVINE INHIBIN CAUSES INCREASED OVULATION RATE IN GILTS

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Litter size in young sows is positively related to ovulation rate (OR) (King and Williams, 1984). Therefore, we have examined the possibility of increasing OR in gilts by immunization against a bovine inhibin α subunit produced by recombinant DNA methods.

A trial was conducted on cross-bred gilts, which, at the commencement of the trial, were 21-24 weeks old and weighed 68-85 kg. Two treatment groups were immunized with a bovine α fusion protein ($b\alpha$) in two different oil based adjuvants. They received doses of 1 mg, delivered intramuscularly in 1 ml formulation, at days 0 and 28. Two control groups were inoculated with a placebo or were unhandled. Gilts were exposed to boars from day 42, observed daily for oestrus and slaughtered five to twelve days after the first observed oestrus. At slaughter, ovaries were collected and OR determined by a count of corpora lutea. Serum samples were assayed for the ability to bind iodinated native bovine inhibin at 1:800 final dilution.

Table 1. Effect of immunization against $b\alpha$

Group	Treatment	% Inhibin binding at day 42	% Inhibin binding at slaughter	Ovulation rate	Interval (days): day 42-oestrus
1 (14)	$b\alpha$	6.6 ^a 4.4-9.1 ²	4.9 ^a 3.2-6.9	15.9 ^a (12) 12.9-19.7	12.3 ^c \pm 2.3 ¹ (12)
2 (15)	$b\alpha$	4.0 ^a 2.0-6.7	3.0 ^a 1.2-5.5	16.5 ^a (14) 14.3-19.0	11.6 ^c \pm 1.7 (13)
3 (15)	placebo	0.2 ^b	0.2 ^b	11.7 ^b (14) 10.7-12.8	8.6 ^c \pm 1.4 (13)
4 (14)	unhandled	Not applicable		12.2 ^b (12) 10.9-13.7	13.3 ^c \pm 2.2 (12)

Values are means; figures in parentheses are number of animals treated or observations made. ^{a,b}differ significantly ($P < 0.01$); ^cdo not differ significantly ($P > 0.1$); ¹SEM; ²95% confidence limits

Ovulation rate of immunized gilts was highly correlated with inhibin binding ($r=0.62$; $P < 0.001$), particularly when weight and age were included in the correlation ($r=0.74$; $P < 0.001$). Immunization against $b\alpha$ caused OR increases of about 35%, presumably as a result of *in vivo* neutralization of porcine inhibin, and caused no deleterious effect on growth rate or onset of oestrus following exposure to boars. Therefore we believe that this treatment could be used to increase prolificacy in gilts and young sows.

References

KING, R.H. and WILLIAMS, I.H. (1984). *Theriogenology*. 21:677-680.

OESTRUS DETECTION IN GILTS EXPOSED TO EXOGENOUS BOAR STIMULI

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The efficiency of detection of oestrus in gilts using the back-pressure test (BPT) is reduced when the gilts are housed adjacent to boars separated by a wire mesh wall rather than opposite boars, separated by a 1 m wide corridor (Hemsworth *et al.*, 1988). This may arise because gilts housed adjacent to boars may habituate to the stimuli from the boar which facilitate the standing response to the BPT (Hemsworth *et al.*, 1988). It is likely that these are olfactory and/or auditory stimuli (Signoret, 1970). Therefore, our objective was to examine whether habituation to auditory and olfactory stimuli from the boar was responsible for this depression in the oestrus detection rate.

Each of 4 groups of 6 gilts was observed concurrently in a 4x4 Latin-square design for 21 days under each of 4 treatments: Gilts were housed either (1) opposite or (2) adjacent to a mature boar or housed in isolation from boars but exposed to either (3) boar pheromones and a broadcast of the boar "courting song" for 5 min in every 30 min for 8 h or (4) pheromones only. The pheromones were delivered to the back of an ovariectomized sow housed in a stall in the gilts' pen. The gilts were checked daily for oestrus using the BPT.

Table 1. Effect of the four treatments on oestrus detection

Treatment	Proportion of gilts detected in oestrus	Proportion of gilts detected in oestrus for more than 1 day	Mean (\pm SE) duration of oestrus (days)
1	23/24 ^b (95.8%)	20/23 ^b (87.0%)	2.3 ^x \pm 0.2
2	18/24 ^a (75.0%)	7/18 ^a (30.9%)	1.1 ^y \pm 0.2
3	24/24 ^b (100.0%)	20/24 ^b (83.3%)	2.5 ^x \pm 0.2
4	24/24 ^b (100.0%)	20/24 ^b (83.3%)	2.7 ^x \pm 0.2

^{a,b}differ at $P < 0.05$; ^{x,y}differ at $P < 0.01$

Exposure of gilts to exogenous boar stimuli did not affect the efficiency of oestrus detection (Table 1). This may suggest that the presence of a boar is necessary for gilts to habituate to boar stimuli to an extent where the efficiency of oestrus detection using the BPT is affected. Alternatively, the boar stimuli may not have been the correct boar stimuli or may not have been applied for long and/or intensely enough for the gilts to habituate and this issue requires further investigation. Nevertheless, we have confirmed that the efficiency of oestrus detection using the BPT is reduced when gilts are housed adjacent to boars.

References

- HEMSWORTH, P.H., WINFIELD, C.G., TILBROOK, A.J., HANSEN, C. and BARNETT, J.L. (1988). *Applied Animal Behaviour Science*. 19:255-264.
 SIGNORET, J.P., (1970). *Journal of Reproduction and Fertility Supplement*. 11:105-117.

LONG DAYS DELAY PUBERTY IN THE GILT

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Attainment of puberty in gilts isolated from boars is delayed in the summer and, while this condition is largely overridden by daily contact with the mature boar, the efficacy of boar contact for inducing puberty is also reduced in the summer (Paterson *et al.*, 1989a,b). The basis for this and other seasonally occurring reproductive dysfunctions in pigs is unknown. In the European wild boar reproductive activity in sows decreases when day-length exceeds 12 h and the young females reach puberty in late autumn when day-length is short (Mauget, 1982). In the present study we investigated the possibility that long day-length is the seasonal factor which delays puberty in the domestic gilt.

On the vernal equinox (September 22, 1988) a total of 35 Large White x Landrace gilts (53.9 days, 17.8 kg) which had been reared together under natural environmental conditions were divided between 2 controlled environment rooms. Initially day-length in both rooms was 12 h (0600-1800 h, 270 lux). In one room the prevailing natural long-day photoperiod was mimicked by increasing day-length 10-15 min/week to a maximum of 14.5 h (December 16-23) then decreasing back to 12 h (March 10-17). In the other room this protocol was reversed to provide a short-day light regime with a minimum day-length of 9.5 h. Temperature was held constant at $23 \pm 1^\circ\text{C}$. From 165 days of age the gilts were inspected daily for signs of oestrus in the absence of boars until they were slaughtered (March 10-17, 224.9 days, 108.3 kg) and their ovaries removed.

Significantly more gilts housed under the short-day light regime reached puberty by slaughter (10/18 vs 1/17, chi-square = 7.84, $P < 0.01$). Mean age at puberty was 209.3 ± 3.13 days for the 10 short-day gilts and the one long-day gilt reached puberty at 228 days.

Paterson *et al.* (1989a,b) showed that among gilts isolated from boars the percentage reaching puberty by 225 days is about 10% in the summer and 50% in the winter when the gilts are housed under normal shed conditions. In the present study we observed a similar pattern when the gilts were housed under controlled light regimes with 5.8% of the gilts on the long-day light regime reaching puberty compared with 55.6% of those on the short-day light regime. This provides compelling evidence that day-length is the environmental factor responsible for the delay in the onset of puberty observed during the summer in the field because puberty was inhibited by long day-length under constant cool temperatures. The clear suppression of reproductive activity by long days observed in this study suggests that day-length may be a contributing factor in other reproductive dysfunctions which follow a seasonal pattern.

References

- MAUGET, R. (1982). In "Control of Pig Reproduction" pp. 509-526, eds. D.J.A. Cole and G.R. Foxcroft (Butterworths: London).
- PATERSON, A.M., HUGHES, P.E. and PEARCE, G.P. (1989a). *Animal Reproduction Science*. **18:293-301**.
- PATERSON, A.M., HUGHES, P.E. and PEARCE, G.P. (1989b). *Animal Reproduction Science*. In press.

THE EFFECT ON PUBERTY OF HOUSING GILTS WITH DRY SOWS

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An increase in the age at puberty, resulting in a reduction in the percentage of available gilts mated/week, has been observed during summer/autumn in a large commercial piggery. In winter and spring, up to 30% of available gilts are mated weekly, whereas in summer/autumn this figure may be as low as 10% (Peacock *et al.*, 1988). A general improvement in reproductive performance during 1987/88 coincided with a management change early in 1987. Rather than being housed in the dry sow shed for a 3-week period before entry into the boar shed, selected gilts went directly to the boar shed from the grower/finisher accommodation. The age at entry into the boar shed remained 26 weeks.

A trial was conducted to determine if accommodation in the dry sow shed before entry into the boar shed had a detrimental effect on the attainment of puberty in gilts. From November 1988 to March 1989, half the selected gilts in a 750 sow unit were accommodated in the dry sow shed for three weeks before entering the boar shed (n=84). In the dry sow shed, gilts were housed between two pens of older sows, with a similar space allowance and group size as those housed in the grower/finisher shed. The rest of the gilts remained in the grower/finisher shed and entered the boar shed at the same time as the gilts from the dry sow shed (n=80). In the boar shed, the two groups were held in separate pens, with a boar on either side. An unpaired t-test was used to compare average time from entry into the boar shed to mating and a 2x2 contingency table used to compare the number of gilts mated within 21 days.

Previous housing affected the time from entry into the boar shed to mating. Gilts from the dry sow shed took 11.9 ± 1.1 days (mean \pm SE) to be mated, whereas those from the grower/finisher shed took 9.3 ± 0.8 days ($P < 0.05$; $t = 1.83$). Seventy per cent of gilts coming from the dry sow shed were mated within 21 days of entry into the boar shed compared with 81% of gilts coming from the grower/finisher shed (chi-square test = 2.69; $P < 0.10$).

These data suggest housing gilts with dry sows before entry into the boar shed is detrimental to the attainment of puberty but do not identify the factor(s) involved. Factors that may be responsible for this delay include the change in diet involved in moving the gilts to the dry sow shed or effects due to the proximity of the sows and gilts. Sows may inhibit reproduction in gilts either by invoking a chronic stress response or perhaps by means of a pheromone. Puberty in gilts reared in crowded conditions is inhibited via some factor contained in the urine (Clark *et al.*, 1985). Evidence that older females can suppress the onset of puberty is available from several rodent and primate species (Vandenbergh, 1983). We suggest that production of an inhibitory pheromone by sows is one explanation for the effects on puberty observed.

References

- CLARK, J.R., FAILLACE, L.S., TRIBBLE, L.F., ORR, D.E. and BELL, R.W. (1985). *Journal of Reproduction and Fertility, Supplement*. 33: 209.
- PEACOCK, A.J., LOVE, R.J. and EVANS, G. (1988). *Australian Advances in Veterinary Science*. 1988:114-117.
- VANDENBERGH, J.G. (1983). In "Pheromones and Reproduction in Mammals" pp. 95-112, ed. J.G. Vandenbergh (Academic Press: London).

THE EFFECT OF GROUP SIZE AND EXPOSURE PEN AREA ON BOAR-INDUCED PUBERTY IN THE GILT

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The boar-effect is thought to be mediated by a synergism between boar primer pheromones and tactile stimuli (Pearce and Hughes, 1987). Decreased puberty stimulation may occur when the frequency of boar-gilt interactions is reduced by either (1) shortening the daily boar exposure period, (2) increasing the number of gilts in the exposure group and/or (3) increasing the size of the exposure pen. The present study investigated the relationship between these variables and the efficacy of the boar-effect.

Two batches each of 48 Large White x Landrace crossbred gilts were allocated to treatment within litter in groups of eight in a 3x2 factorial design involving boar exposure in groups of 8, 4 or 2 gilts in large (L) (22.2 m²) or small (S) (11.1 m²) exposure pens. Daily boar exposure occurred for 20 min in experiment 1 and for 5 min in experiment 2, between mean gilt ages of 160 and 240 days. Interactions between the boar and gilts (defined as gilt's head being within 50 cm of the boar) were assessed in experiment 2 as proportions of instantaneous scans taken every 10 sec from video recordings on days 1, 2, 3, 6, 7 and 8.

Table 1. The effect of treatment on the mean interval to puberty attainment (days)

Treatment	Experiment 1	Experiment 2
(1) 8L	22.2 ± 8.4 (8/8) ¹	30.1 ± 4.7 (8/8)
(2) 8S	17.2 ± 5.7 (8/8)	24.0 ± 1.5 (8/8)
(3) 4L	18.4 ± 6.6 (7/8)	28.0 ± 3.1 (7/8)
(4) 4S	19.6 ± 4.4 (8/8)	22.4 ± 4.9 (7/8)
(5) 2L	28.0 ± 12.9 (6/8)	25.7 ± 4.3 (8/8)
(6) 2S	23.7 ± 6.3 (8/8)	29.6 ± 0.9 (7/8)

¹proportion of gilts reaching puberty by 240 days of age; values are means ± SE

There was no significant effect of treatment on the interval to puberty in either experiment (Table 1). Reducing the period of boar exposure from 20 to 5 min/day resulted in a trend towards later puberty attainment. Fewer ($P < 0.05$) gilt-boar interactions occurred in groups of eight gilts than in groups of four or two (means = 0.308, 0.381 and 0.398, respectively) and in large pens than in small pens (0.333 vs. 0.390). Gilts reaching puberty within 20 days ($n=5$) tended to interact more with the boar than gilts which did not reach puberty until after 40 days of treatment ($n=7$) (54.8 ± 4.03 sec and 46.8 ± 8.5 sec/5 min, respectively).

Sufficient boar contact was achieved to stimulate puberty even in the large exposure pen with eight gilts/group. It is likely that much of the variation in puberty attainment was due to differences in gilt sensitivity to puberty inducing stimuli (Paterson *et al.*, 1989), rather than avoidance or non-perception of the stimuli in large groups and/or pen sizes.

References

- PATERSON, A.M. HUGHES, P.E. and PEARCE, G.P. (1989). *Animal Reproduction Science*. 19: in press.
 PEARCE, G.P. and HUGHES, P.E. (1987). *Animal Production*. 44:293-302.

ADRENAL RESPONSIVENESS AND REPRODUCTIVE PERFORMANCE IN GILTS

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Pigs with a low potential to respond to ACTH have a better growth rate and feed conversion efficiency (Hennessy and Jackson, 1987). To date there is no evidence about the differences in reproductive performance among pigs selected for their inherent adrenal response. This trial was designed to investigate the relationship between inherent adrenocortical response and reproductive performance in gilts.

The trial was conducted during mid-summer to early autumn on four commercial farms. Unmated gilts at approximately 29 weeks of age were challenged with 50 I.U. synthetic ACTH (intramuscularly). One hour after ACTH administration, a blood sample was taken via jugular venipuncture from each gilt. Serum cortisol concentration was determined using a validated radioimmunoassay (Hennessy *et al.*, 1988). The post ACTH serum cortisol concentrations were ranked from the lowest to the highest for each gilt on each farm and the reproductive performance of these gilts was compared.

Table 1. Farrowing rate of gilts falling in either the lowest or highest 10 percentile of the population with respect to adrenal response (figures in parentheses indicate number of pigs).

	Farrowing rate (%)				Average
	Farm A	Farm B	Farm C	Farm D	
High responders	60.0 ^a (24)	44.4 ^a (18)	71.4 (8)	83.3 (12)	61.7 (62)
Low responders	93.8 ^b (24)	94.4 ^b (18)	100.0 (8)	75.0 (12)	90.4 (62)

^{a,b}differ at $P < 0.05$ (chi-square test); chi-square = 4.5 and 10.6 at farms A and B, respectively

In the four farms, there was a trend for gilts with a low adrenal response (LR) to have a higher farrowing rate than gilts with a high adrenal response (HR). On two farms these differences reached statistical significance. The mating rate and litter size (born alive) were not significantly different between the HR and LR on each farm. The average mating rate (%) and born alive litter size were 79.0 and 85.5 and 9.5 and 8.7 for the HR and LR, respectively. Interestingly, there were 16 HR, compared with only 4 LR which subsequently suffered from seasonal infertility i.e. a delayed return (>24 days) or a failure to return to oestrus. In conclusion, gilts with a low adrenal response to ACTH had a tendency for better reproductive performance over the four farms.

References

- HENNESSY, D.P. and JACKSON, P.N. (1987). In "Manipulating Pig Production" p. 23, eds. APSA Committee (Australasian Pig Science Association, Werribee, Victoria: Australia).
 HENNESSY, D.P., STELMASIAK, T., JOHNSTON, N.E., JACKSON, P.N. and OUTCH, K.H. (1988). *American Journal of Veterinary Research*. 49:1276-1283.

A SYMPOSIUM - PRE-MATING MANAGEMENT OF THE GILT

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Introduction

In most piggeries gilts will account for 15-30% of all breeding females and because of their lower reproductive performance (Table 1), replacement gilts have a substantial impact on the overall reproductive performance of the breeding herd. Consequently the gilt has been the focus of considerable research aimed at improving her reproductive performance.

Table 1. A comparison of the reproductive performance of 1158 gilts and 3356 sows over an 18-month period in a commercial piggery (Hemsworth, unpublished data)

	Gilts	Sows
Farrowing rate (%)	84.5	89.9
Litter size - Total	9.48	11.24
- Alive	8.80	9.95

The objective of this symposium is to review the literature on some of the main factors affecting the reproductive performance of gilts and to highlight areas where future research may lead to improvements in reproductive performance. In the first paper, the consequences of the chronological and sexual age at first mating on the immediate and long-term reproductive performance of gilts are considered. The techniques available to control the onset of puberty in gilts are also reviewed in the first paper. The effects of pre-mating nutrition on the short and long-term reproductive performance of gilts are discussed in the second paper. A large component of this paper is the discussion on the implications of body condition on age of puberty and ovulation rate of gilts and on long-term reproductive performance. Thus, the first two papers are addressing the controversial question of the age and stage of development that gilts should be mated.

The factors that influence the efficiency of both detecting oestrous gilts and mating these gilts are considered in the third paper. As expected, social factors play a major role in regulating the sexual behaviour of the female pig and thus a considerable part of this paper is concerned with the influence of social factors.

Therefore, the objective of this symposium is to review the current literature on the pre-mating factors affecting the short and long-term reproductive performance of gilts. It is thus the intention of this symposium to identify some of the present and future opportunities to improve the reproductive performance of gilts.

Symposium continued on next page

AGE AT MATING AND PRODUCTIVITY OF GILTS

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Introduction

The optimum age and weight for first mating of gilts is the subject of debate with the conclusions liable to depend on how the performance of the gilt is assessed, the level of management expertise applied and the physical characteristics of the piggery. This part of the symposium examines the evidence for an effect of age at first mating on the reproductive performance of gilts and then considers management strategies which can be employed to control the onset of puberty and hence the introduction of gilts into the breeding herd.

Reproductive performance

There are numerous reports in the literature that litter size increases with age at first mating (Omtvedt *et al.*, 1965; Strang, 1970; Pay and Davies, 1973; Walker, 1982). In experimental situations, estimates of the increase in litter size for each day mating is delayed range from 0.062 (MacPherson *et al.*, 1977) to 0.026 (Brooks and Cole, 1973) pigs/litter. However for the purpose of deciding on the optimum age at first mating, there are problems with interpreting these studies.

Productivity

While there is little doubt that delaying mating will increase litter size, it must be questioned whether this represents a real increase in the productivity of the gilt and the overall herd. Calculations based on the estimates of the increase in litter size expected from delaying mating show that the extra piglets which result in delaying mating from 190 until 232 days are insufficient to make up for the time lost (Brooks, 1982). Of relevance here is the report by Walker (1982) based on the records of a large commercial piggery. These data show a linear increase in litter size with increasing age at first mating up to 280 days but 72% of the gilts in the herd were mated by 235 days of age because the management found it more profitable.

The other aspect of performance which must be considered is whether there are any long-term effects of mating at different ages. MacPherson *et al.* (1977) found that despite large differences in first litter size between gilts which were mated at different ages (and oestrous cycles), performance over their first three litters was almost identical. Similarly, Brooks and Smith (1980) found that gilts mated at 198 days produced smaller first litters than gilts mated at 237 days, but over five litters the total number of piglets produced was the same. The gilts mated at 198 days consumed 6.2% less food/kg of weaner produced in those five litters. This supports the survey data of Legault and Dagorn (1973) who found that the number of litters produced and the herd life of sows were not affected by age at first mating and that annual sow productivity was reduced by 0.02-0.03 pigs for each day that the average age at first mating was increased.

Sexual age

The second problem with interpreting the data on age and weight at first mating is that most studies have confounded the effect of chronological age with sexual age or the number of previous oestrous cycles the gilt has experienced. In gilts which reach puberty at the same age, reproductive performance at the pubertal oestrus is inferior

to that of gilts in which mating is delayed to a later oestrous cycle, when the gilts are also older and heavier (Brooks and Cole, 1973; Pay and Davies, 1973; MacPherson *et al.*, 1977).

This increase in litter size may be due to an increase in ovulation rate over successive cycles after puberty (Warwick *et al.*, 1951; Paterson and Lindsay, 1980) rather than an effect of age or weight *per se* because when gilts were managed using nutritional manipulation (King, 1989) or boar exposure (Burnett *et al.*, 1988) so that body weight and age at mating at the pubertal oestrus differed widely, litter size was not affected by age or weight. However when gilts were mated at their second oestrus at 198 or 237 days of age, the younger gilts produced smaller litters (Brooks and Smith, 1980) indicating an effect of age (or weight or body composition) independent of the number of oestrous cycles in the post-pubertal gilt as suggested by Kirkwood and Aherne (1985). Similarly when gilts were mated at their first or third oestrous cycle at about 220 days of age (Paterson and Lindsay, 1980; Knott *et al.*, 1984) reproductive performance was not affected by sexual age. In addition these studies showed that mating at the second oestrus at 220 days (Paterson and Lindsay, 1980) or at the third oestrus at 240 days (Knott *et al.*, 1984) gave similar reproductive performance to that when mating took place at 220 days on either first or third oestrus.

The data reviewed show that when gilts are mated at their pubertal oestrus at ages below 190 days, first litter performance is independent of age or body weight. However the level of performance achieved and the possibility that gilts mated at less than 90 kg may have a reduced breeding life (Walker and Burnett, 1984), suggests that mating should be delayed until their second oestrus. As either age at puberty or the number of oestrous cycles increases, the reproductive performance improves such that gilts mated at about 220 days of age will perform similarly whether they are mated at their first or later oestrus. This suggests that there is little to be gained from delaying mating beyond 220 days of age and in most situations mating gilts aged about 200 days and weighing over 100 kg on their second or later oestrus would be the recommended strategy.

Control of puberty

To achieve the aim of mating gilts around 200 days on their second or later oestrous cycle requires that they attain puberty well before 200 days of age. Recent work in our laboratory (Paterson *et al.*, 1989a,b; Paterson and Pearce, 1989a) showed that this will not happen unless gilts receive additional stimulation to induce puberty. This problem was particularly evident in the summer months when less than 10% of unstimulated gilts reached puberty by 225 days of age compared with about 50% in the winter (Paterson and Pearce, 1989b).

The two stimuli which have been most widely studied are contact with mature boars and injection of exogenous hormones. The remainder of this review focuses on the use of these techniques and the effect of age and weight at stimulation on the response obtained.

Contact with boars

Management regimes which include physical contact with a mature boar for about 30 min/day are a potent stimulus to the onset of puberty in the gilt (see Hughes, 1982; Hughes *et al.*, 1989 for reviews). The age and weight of the gilts at the start of boar exposure affects the interval from first contact to puberty. As age at first boar contact increases above 130 days the interval to puberty becomes progressively shorter and less variable (Kirkwood and Hughes, 1979; Burnett *et al.*, 1988) reaching minimal levels around 160 days of age even when the gilts are fed so that body weight is constant in each age group (Hughes and Cole, 1976). The net result of this decrease in the

interval with increasing age is that regardless of when boar exposure starts before about 160 days, the age at puberty will be the same. If first boar exposure is delayed beyond 160 days there is no further significant reduction in the interval to puberty and the mean age at puberty will increase (Hughes and Cole, 1976; Kirkwood and Hughes, 1979; Burnett *et al.*, 1988). This suggests that gilts achieve a threshold age for puberty stimulation at about 160 days of age and having achieved this threshold they are equally responsive to boar stimuli regardless of their age.

When gilts are very young, body weight also appears to play a role in determining the interval to puberty. The gilts studied by Burnett *et al.* (1983) were over 20 kg heavier at the same age than those of Hughes and Cole (1976) and they were more responsive to boar stimulation. In addition, the interval to puberty in these gilts was negatively correlated with growth rate, live weight and backfat thickness which indicates that the rate of sexual maturation in the young gilt, as measured by responsiveness to stimuli which initiate puberty, can be accelerated by a higher rate of growth and increased body fat reserves. Further support for this concept comes from the recent study by King (1989), who found that age at puberty in response to boar exposure at 170 days of age declined linearly as the mean group body weight at introduction increased from 60-118 kg. However the situation with older gilts appears to be different. Knott *et al.* (1984) reported that in groups of gilts which commenced boar stimulation after 170 days of age, variation in weight between 70 and 116 kg had no effect on the time taken to reach puberty. This indicates that there is also a threshold for weight, as there is for age, above which all gilts are equally responsive.

Attainment of these threshold levels of age and weight are prerequisites for successful stimulation of puberty but their attainment alone does not trigger the onset of reproductive activity. The relationship between attainment of puberty, age and weight may be complicated by body composition and Kirkwood and Aherne (1985) have suggested that a minimum fat to lean ratio is required for the attainment of puberty. Burnett *et al.* (1988) reported that quadratic equations including age, weight and backfat levels were more accurate predictors of sexual maturity as measured by response to boars than were simple equations based on age or weight. Similarly King (1989) showed that low body weight and increased backfat in gilts of the same age were associated with delayed onset of puberty.

The data reviewed here and elsewhere (Hughes, 1982; Hughes *et al.*, 1989) suggest that most gilts will have reached the necessary stage of sexual maturation for successful stimulation of puberty by the time they are 160-170 days of age and weigh over 70 kg. This has become, and continues to be, the recommended minimum age and weight for the commencement of boar stimulation. However the study of Burnett *et al.* (1988) with extremely fast growing gilts shows that puberty stimulation and acceptable reproductive performance can be achieved in gilts as young as 130 days of age. Further research with fast growing strains may prove that successful reproduction can be initiated much earlier than is currently believed.

Exogenous hormones

Paterson (1982) reviewed the literature on the induction of puberty with exogenous hormones and concluded that neither combinations of pregnant mare's serum gonadotrophin (PMSG) and human chorionic gonadotrophin (HCG) nor compounds of the steroid oestradiol provided a reliable method for controlling puberty in the gilt. While in most cases these hormones were effective in inducing ovulation, there were many problems associated with their use including failure to show oestrus, poor reproductive performance, failure of corpora lutea to persist in pregnant animals and failure to maintain cyclic activity in unmated gilts.

Studies published since that review have done little to alter the situation. Paterson *et al.* (1984) studied oestrus, ovulation and reproductive performance of gilts treated

with a combination of PMSG and HCG. This treatment was highly effective at inducing a synchronized ovulation but only 45% of the gilts showed behavioural oestrus and the overall pregnancy rate for all gilts treated was 25%. When mating was delayed until the expected time of the second oestrus, only 60% of the gilts that had ovulated in response to PMSG/HCG maintained cyclic activity. Burnett *et al.* (1988) treated groups of gilts ranging from 130-190 days of age with the same combination of PMSG/HCG and found that the incidence of ovulation accompanied by oestrus was high and the mating rate was comparable to that of gilts stimulated by boars. Most of the gilts which failed to respond were in the 130- or 150-days groups suggesting that many of them had not reached the necessary stage of sexual maturity to respond to the gonadotrophins. Overall 72% of those mated were pregnant at day 35 post coitus which represents 62% of the total treated and the distribution of litter size suggested that further losses were likely before farrowing. These problems were not confined to the 130- or 150-day groups and Burnett *et al.* (1988) concluded that although PMSG/HCG was effective in inducing puberty, it was associated with a low conception rate, a highly variable litter size and a high percentage of gilts which failed to maintain cyclic activity following the induction of puberty.

Paterson *et al.* (1984) also treated gilts with oestradiol benzoate which induced ovulation accompanied by oestrus in over 90% of treated gilts. However conception rate was low and only 50% of the treated gilts were pregnant at day 28 post coitus. In animals that were not mated, 33% maintained cyclic activity and were mated at their second oestrus. Similar results were reported by Yang *et al.* (1987a) and Dyck (1988) confirming that oestradiol alone is of limited value for controlling puberty in gilts destined to be mated at either their first or subsequent oestrous cycles.

The problem of maintenance of cyclic activity after induction of ovulation with oestradiol to allow mating at the second oestrus has recently been investigated by Yang *et al.* (1987b). Gilts were induced to ovulate with oestradiol benzoate and then treated with the synthetic progestin allyl trenbolone and/or daily boar exposure during the ensuing luteal phase. While the effect of each factor alone was impossible to assess because no control treatment using oestradiol benzoate alone was included, the combination of oestradiol benzoate and boar exposure resulted in 92% of the gilts maintaining cyclic activity and mating at second oestrus. The reproductive performance of these gilts was acceptable and their mean age at mating was 31 days less than gilts which were stimulated into puberty using boar exposure. It therefore seems that if exogenous hormones have any potential for controlling puberty it may lie in finding techniques which ensure maintenance of cyclic activity after induction of ovulation and then mating them at the second cycle. In this regard the work of Paterson and Lindsay (1981) who showed that continued boar exposure assisted the maintenance of cyclic activity in PMSG/HCG treated gilts and the work of Yang *et al.* (1987b) discussed above, provide useful starting points for further research which may see the development of a reliable system for controlling puberty in gilts with exogenous hormones.

Conclusions

There is little to be gained from delaying first mating in gilts beyond 220 days of age. Provided the gilts are over 100 kg and experiencing their second or later oestrous cycle, age at mating may be reduced to 190-200 days without adversely affecting productivity. There is some evidence from recent studies with strains of gilts which grow very fast that age at mating may be lowered even further but more research, including assessment of lifetime performance, is required before this could be recommended.

There appears to be a threshold of age, weight and perhaps body composition, which must be reached before puberty can occur. Attainment of these threshold levels

alone is not sufficient to trigger attainment of puberty and some stimulus must be given to induce puberty by 180 days of age. Daily exposure to mature boars from about 160 days of age offers the best method for stimulating puberty. Although exogenous hormones can cause ovulation in gilts, the reproductive performance achieved is highly variable and their use cannot be recommended. The possibility of using these ovulatory compounds in conjunction with other treatments in the induced luteal phase to ensure maintenance of cyclic activity and mating at the second oestrus offers a potential technique for commercial application. Further research into this aspect of puberty control should be carried out.

Symposium continued on next page

NUTRITIONAL MANAGEMENT TO IMPROVE THE REPRODUCTIVE PERFORMANCE OF COMMERCIAL GILTS

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Introduction

The management of the gilt and its genetic constitution has changed appreciably over the past 10-15 years. The use of boar stimuli and management stimuli such as mixing and transport stress has allowed gilts to attain puberty at less than six months of age (Hughes, 1982). Economic pressures to mate gilts as early as possible has led to the modern gilt beginning its reproductive life at about 200 days of age. Genetic programs over the past 10-15 years have also placed emphasis on selection for leanness in gilts. The combined effects of these genetic changes and earlier mating mean that gilts now start their breeding lives with less body reserves than in the past.

This part of the symposium will review studies on the effects of nutrition and body composition of gilts on their short term and long term reproductive efficiency and will also discuss the possible mechanisms which regulate and mediate the effects of nutrition on reproduction.

Age at puberty

Nutrition during the rearing period and the consequent body weight and body composition may influence the attainment of puberty. The onset of puberty in gilts has been delayed by the reduction of growth rate through long-term food restriction (Van Lunen and Aherne, 1987; Kirkwood *et al.*, 1986). Similarly, Den Hartog and Noordewier (1984) found that gilts which received increased food intake during rearing reached pubertal oestrus at a younger age than those receiving restricted food intake. However it was not clear from these experiments whether the effect of long-term food restriction have a direct effect on the attainment of puberty or its effects were mediated by its effect on body weight.

There has been little objective information on the interrelationships between body weight, body composition and age at puberty. Knott *et al.* (1984) found that variation in live weight between 70 and 116 kg at 170 days of age failed to affect the age of puberty attainment which suggests that body weight is of no importance in reproductive development. However King (1989) found a linear negative relationship between age at puberty and live weight at 170 days of age. An explanation for these equivocal results may lie in a consideration of the genetic potential of the gilt. During rearing, the gilts in the experiment of Knott *et al.* (1984) were fed *ad libitum* and their growth may have reflected differences in genetic potential or mature size. The inference is that although animals differed in live weight, they were a similar proportion of their mature size and may be expected to show the same response to boar exposure. However King (1989) imposed feed restriction to achieve the differences in live weight and consequently the groups would have represented different proportions of mature body size and may be expected to respond differently to boar exposure. Thus it seems reasonable to hypothesize that puberty occurs when the gilt reaches a certain proportion of its mature body size.

Kirkwood and Aherne (1985) suggested that threshold levels of live weight and more particularly fatness were necessary for the attainment of puberty in gilts.

Furthermore the results of Burnett *et al.* (1988) indicated that the rate of sexual maturation in the gilt can be accelerated by a higher rate of growth and increased body fat reserves. However as a prepubertal gilt becomes heavier it will naturally become fatter and in the experiment of Burnett *et al.* (1988) fatness was confounded with live weight and genotype.

King (1989) studied the effect of body composition on the onset of puberty without confounding it with live weight or genotype and found that an earlier onset of puberty was associated with animals which were not only heavier but tended also to contain a greater amount of lean tissue (King, 1989). This response of the prepubertal gilt to changes in lean tissue has support in the results of both King and Dunkin (1986), and Brendemuhl *et al.* (1987) who showed that the restriction of protein intake during lactation and the consequent mobilization of significant amounts of protein from lean tissue result in prolonged weaning-to-oestrus intervals in first litter sows.

Britt *et al.* (1988) used the nutritionally-induced anoestrous gilt as a model to investigate the effects of nutrient intake and exogenous hormones on cyclic activity and the hypothalamic-hypophyseal-ovarian axis. Again, their results indicate that the nutritionally-induced acyclic condition was related more to the depletion of body protein reserves than body fat (Britt *et al.*, 1988). Thus it seems that lower or diminished body protein reserves may have an adverse effect on the fertility of gilts and young sows.

Although body weight and the proportion of lean tissue may influence the onset of puberty, these factors, in practice, may have little relevance in modern pig production. Selected gilts often reach at least 100 kg by 170 days of age and at this point, live weight or lean mass is unlikely to markedly affect the attainment of puberty.

Ovulation rate/litter size

The fecundity of gilts may be influenced by nutritional management both in the short term and in the long term. Den Hartog (1984) found that, although pubertal ovulation rate was unaffected by feeding level during the rearing period, gilts given a high energy intake during rearing had a higher ovulation rate at subsequent oestruses than gilts given lower feeding levels. Hovell *et al.* (1977), MacPherson *et al.* (1977) and Kirkwood *et al.* (1986) also reported greater ovulation rates or litter sizes in gilts which were given higher food intakes during rearing. Despite heavier gilts reaching puberty earlier, King (1989) found that the pubertal ovulation rate of gilts which were heavier at puberty was greater than that of lighter gilts. Although the results of the above experiments which have examined the effects of nutrient intake during rearing and subsequent body weight on ovulation rate and litter size are not conclusive, there does appear to be a positive relationship between body weight and ovulation rate which is independent of sexual or chronological age.

The effects of short term nutritional changes on ovulation rate and litter size are much more clear cut and consistent. The effect of high levels of feed intake during the oestrous cycle or "flushing" has been studied extensively and these experiments have been reviewed by Den Hartog and Van Kempen (1980). The results of experiments have shown that ovulation rate increases by about two ova in response to increased feed intake during the 14-day period prior to ovulation (Den Hartog and Van Kempen, 1980). Recently, Kirkwood *et al.* (1986) suggested that the effect of flushing occurred only in gilts which were restricted in feed intake during rearing. Consequently Kirkwood *et al.* (1986) suggested that flushing is not a superovulation but merely a normalising of low ovulation rates resulting from poor nutrition.

In the "flushing effect", energy appears to be the dietary component which is limiting ovulation rate (Den Hartog and Van Kempen, 1980). Despite the ovulatory response to flushing being recognised for nearly twenty years, the controlling mechanism for greater ovulation rate in response to increased energy intake is still not clear. Cox

et al. (1987) found that insulin administration prior to ovulation could produce an ovulation rate response similar to that observed with flushing. Flowers *et al.* (1989) studied the hormonal profile in gilts with an increased ovulation rate due to flushing and found that gonadotrophic hormone secretion was increased during the 5-day period prior to oestrus. In addition an increase in the plasma level of insulin occurred approximately two days before the change in gonadotrophic secretion was detected. Flowers *et al.* (1989) suggested two possible mechanisms involved in the flushing effect. Either the stimulatory effect of flushing on gonadotrophic secretion and ovulation rate may have been mediated, in part, through changes in plasma insulin or alternatively, insulin may have a direct effect at the ovarian level in stimulating follicular growth that is independent of gonadotrophic secretion. As ovulation rate is often limiting the fecundity of gilts and young sows (King and Williams, 1984) further investigations are required into the mechanisms controlling the flushing effect which may lead to a greater understanding of the overall hormonal and biochemical control of ovulation rate.

Lifetime reproductive performance

Concern that inadequate body weight and, more particularly, inadequate body fatness could deleteriously affect the long-term reproductive performance and longevity has been expressed by Whittemore *et al.* (1980), following the development of genetically improved lean strains of pigs.

King *et al.* (1984) found that gilts which entered the breeding herd with greater fat reserves were retained in the herd longer and had a shorter average farrowing interval than gilts with less fat reserves. On the other hand, there is evidence that excess body weight and fatness of gilts may adversely affect long-term reproductive performance. Although increased nutrient intake during rearing hastened the onset of puberty in gilts (Den Hartog and Noordewier, 1984), subsequent reproductive performance may be adversely affected. Den Hartog (1984) reported that high levels of energy intake (2.5 maintenance or greater) for gilts during rearing seemed to be detrimental to their conception rate and that more animals given the high energy intakes had to be culled because of locomotion disorders and leg problems.

There is very little quantitative information on the level of fatness that gilts should achieve either at mating or at parturition to ensure that they have sufficient reserves for a long and productive breeding life. Yang *et al.* (1989) compared gilts which contained either 12 or 20 mm P₂ backfat at parturition and concluded that feeding strategies to ensure a target level of P₂ of at least 18-20 mm at parturition will be rewarded by higher levels of production. This recommendation by Yang *et al.* (1989) may apply only to the improved strains of hybrid gilts and the pregnancy/lactation feeding strategy which were used in their experiment. Caution must be exercised in adopting the recommendations of Yang *et al.* (1989) for other types of pigs under various feeding strategies. Different genotypes may have quite different threshold levels of body reserves. Furthermore the minimum levels of body reserves required by the gilt will largely depend upon the feeding strategy employed during subsequent pregnancy and lactation periods.

Until more research is conducted, conservative recommendations only can be made regarding the minimum target levels required by gilts at mating to ensure subsequent life-time reproductive productivity is maximised. Research into this area will be extremely expensive and time-consuming; the two approaches are, firstly to accurately monitor the body reserves of large numbers of gilts and their subsequent long-term reproductive efficiency in commercial piggeries and secondly, to establish large scale, long-term sow experiments, in which gilts with quite extreme amounts and proportions of body reserves at mating are reared, to examine the effect of initial body reserves on long term reproductive performance.

Practical recommendations

Selected replacement gilts should remain on the finisher diet (0.55 g available lysine/MJ digestible energy) between selection and mating. There is no benefit in encouraging gilts to deposit any more fat than is normally deposited under a liberal feeding strategy with protein-adequate diets. Between selection and mating gilts should receive at least 35 MJ digestible energy/day to ensure that the gilt reaches 120-125 kg live weight by 200 days of age. Minimum levels of backfat for gilts at mating have yet to be established. However provided gilts are at least 120-125 kg live weight at mating (Yang *et al.*, 1989) it is likely that the level of body fat and protein reserves will be sufficient to prepare the gilts for a long and productive breeding life. To ensure optimum ovulation rates at mating, gilts should be offered the finisher diet *ad libitum* for about 14 days prior to mating.

DETECTION AND MATING OF OESTROUS GILTS

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It is generally recognized that pig producers have more difficulty in getting gilts mated than sows (English *et al.*, 1982). An examination of the literature indicates that in addition to delayed puberty, poor oestrus detection contributes to mating difficulties in gilts (see Hemsworth, 1982). The incidence and consequences of poor sexual receptivity in those gilts detected in oestrus are unknown, however this condition does occur (Cronin *et al.*, 1982). Nevertheless, sexual receptivity and oestrus detection should be considered under the same behavioural category since receptivity or the standing response is generally the criterion used in the main procedures of oestrus detection (i.e. use of boars or the back-pressure test). The objectives of this part of the symposium are to review the major factors that affect the oestrus detection rate and sexual receptivity of gilts and to identify areas where research is required.

Boar contact

In addition to stimulating the early attainment of puberty in gilts (see paper by Paterson in this symposium), it appears that boar contact is important in maintaining oestrous activity. Paterson and Lindsay (1981) observed a greater percentage of gilts in oestrus at the anticipated time of the second oestrus when gilts were daily introduced to boars after their pubertal oestrus (87%) rather than isolated from boars after their pubertal oestrus (52%). Following slaughter it was found that failure to ovulate, rather than failure to detect oestrus, was responsible for the lower percentage of gilts detected in oestrus in the latter treatment. The proposal that olfactory contact with boars may be involved in the maintenance of oestrous activity is supported by the results of Signoret and Mauleon (1962) and Booth and Baldwin (1983) in which olfactory bulbectomy disrupted the ovarian activity of sexually mature gilts. In the latter study, olfactory stimuli and season were implicated.

The most common procedure of oestrus detection other than the use of boars is the back-pressure test (BPT) or riding test (Signoret, 1970). Females displaying the "standing" or lordosis response for at least 10 sec to pressure on their backs are generally classified as being sexually receptive (Hemsworth *et al.*, 1988). The efficiency of this procedure is dependent on the female receiving intense boar contact at the time of testing. Signoret (1970) reported that the maximum percentage of gilts displaying the standing response to the BPT in the absence of boars was 59% between 24 and 36 h after the start of oestrus. This percentage was increased to 90% by providing the gilts with auditory and olfactory contact with boars and further increased to 100% with the addition of visual and tactile contact with boars. Similarly, Hemsworth *et al.* (1984) demonstrated the importance of intense contact with the boar at the time of conducting the BPT. Testing the gilts at a distance of 1 m or more from the boar, which presumably reduced the amount of boar contact, reduced the efficiency of the test (52% of post-pubertal gilts detected in oestrus compared to 90% when gilts were tested adjacent to boars).

While it appears that boar contact has an important role in stimulating the female's sexual behaviour, there are situations where continuous stimulation from the boar may adversely affect sexual behaviour. Research at our laboratory (Hemsworth *et al.*, 1984; Hemsworth *et al.*, 1986a; Hemsworth *et al.*, 1988) has shown that housing post-pubertal gilts adjacent to boars, with a wire-mesh wall separating them, results in

a low oestrus detection rate with the BPT (when gilts were tested adjacent to boars). It has been proposed that habituation by gilts to the important boar stimuli (e.g. auditory and olfactory stimuli) which facilitate the standing response of the oestrous female to pressure on her back (Signoret, 1970), is responsible for this detection problem (Hemsworth *et al.*, 1988). Recent research has also indicated that this housing procedure which is common in the industry may also produce oestrus detection problems when boars are used for oestrus detection (Hemsworth *et al.*, 1987a). Therefore the results of these studies indicate that the common practice of housing post-pubertal gilts adjacent to boars, with a wire-mesh or barred wall separating them, may adversely affect the sexual behaviour of the gilts to the extent where there are difficulties with oestrus detection.

There is substantial variability in the industry in procedures that use a boar to detect oestrous females and yet these procedures have received little research attention. The study by Hughes *et al.* (1985) indicates that 6-7 month old boars may be less efficient at detecting oestrous gilts than older boars because the younger boars provide the gilts with less olfactory and auditory stimulation. Clearly further research is required to examine the effects of factors such as the sexual motivation and recent mating frequency of the boar, the testing time and the group size of females on the efficiency of oestrus detection using procedures that actively utilize boars.

Space allowance and group size

There is some limited evidence that space allowance and, to a lesser extent, group size of group-housed gilts may influence the efficiency of oestrus detection. Hemsworth *et al.* (1986b) examined the effects of housing groups of adult post-pubertal gilts (6 pigs/group) with a space allowance of 1, 2 or 3 m²/gilt on sexual behaviour. A lower percentage of gilts were detected in oestrus when housed with a space allowance of 1 m²/gilt than with a space allowance of 2 or 3 m²/gilt (oestrus detection rate of 79, 88 and 100%). A significant sustained increase in plasma free corticosteroid concentrations of gilts housed with a space allowance of 1 m²/gilt suggests that a chronic stress response may have impaired expression of oestrus.

In a study of 2,484 gilts at a commercial piggery, Cronin *et al.* (1983) reported that 10.5% of gilts were not mated between 29 and 35 weeks of age. Seventy percent of the unmated gilts had ovulated during this 6-week period, but the majority were not detected in oestrus using the BPT in the presence of boars. Group size and space allowance prior to entry to the mating shed were implicated in this problem because increasing group size (above 50 gilts/pen) and concomitantly reducing space allowance (below 0.9 m²/gilt) were associated with an increase in the percentage of post-pubertal gilts not detected in oestrus (3.6 vs 8.0%). Clearly more comprehensive research is required, but in the meantime it is suggested that post-pubertal gilts around the time of mating should be provided with at least 2 m²/animal. In a survey of 33 Victorian piggeries (Hemsworth, unpublished data) it was found that at 54% of the farms, post-pubertal gilts were often kept in groups with less than 1.5 m²/animal. Therefore, the potential exists to improve oestrus detection by providing oestrous gilts with more space.

In addition to these studies on space allowance, several studies have examined the affects of group size. Christenson and Ford (1979) reported two experiments that examined the influence of group size (8 vs 24 gilts with a constant space allowance of 1.2 m²/gilt) from 6-12 months of age on the sexual behaviour of gilts. In one of the experiments, a higher percentage of crossbred gilts were regularly detected in oestrus when in groups of eight than in groups of 24 animals (96.8 and 85.4%, respectively). Presuming that all gilts had reached puberty, a reduction in the detection rate appears to be responsible for the slightly poorer performance of groups of 24. In the other experiment, using purebred gilts and conducted at a different time of the year, there

was no difference in the percentage of gilts detected in oestrus in groups of 8 or 24. However, the percentage of gilts that were regularly detected in oestrus in both treatments was extremely low (56.3 and 58.7% respectively). In another study at the same laboratory, Christenson (1984) found that a lower percentage of gilts were regularly detected in oestrus between 7 and 9 months of age when housed in groups of 3 rather than 9, 17 or 27 (56.9% vs 78.0, 80.4 and 80.7% respectively). Space allowance for all groups was 1.1 m²/gilt. An increase in the percentage of pre-pubertal gilts and an increase in the percentage of post-pubertal gilts not detected in oestrus were responsible for the poorer performance of gilts in groups of three.

The effects of group sizes of 2, 4 or 8 with a space allowance of 1.4 m²/gilt on the sexual behaviour of post-pubertal gilts have been examined by Barnett *et al.* (1986). Although the plasma free corticosteroid concentrations of gilts in groups of 2 were elevated, there was no significant effect on the oestrus detection rate. Since the oestrus detection rate was low for all group sizes (less than 60%), it is possible that there were no significant treatment effects because of the sub-optimal space allowances. Sub-optimal space allowances in the studies conducted by Christenson (as reflected in the overall low detection rates) may also have masked the treatment effects and so further studies on group size are warranted.

The literature on the effects of group size on the sexual behaviour of female pigs is equivocal. It is difficult to compare studies due to differences in boar contact, age of females, season etc., but there appear to be problems with oestrus detection in small groups (Christenson, 1984) and large groups (Christenson and Ford, 1979; Cronin *et al.*, 1983). The interaction between group size and space allowance must be examined to clarify the optimal social and spatial conditions for group-housed gilts.

Physical conditions at the time of mating

It is common in intensive pig production for matings to be conducted in the boar's accommodation pen even though the conditions in the pen often appear far from ideal for mating. Some recent evidence indicates that the physical conditions at the time of mating may have considerable effects on the sexual behaviour of pigs. Hemsworth *et al.* (1989a) found that the percentage of mating tests which resulted in successful copulations was significantly lower for pigs mating in the boar's accommodation pen than for those mating in a specific mating pen (76% and 88%, respectively). The sexual behaviour of the gilts in the two treatments was similar, but there was a consistent trend for the sexual behaviour of the boars in the treatments to differ, suggesting that the low mating rate of pigs mating in the boar pen treatment may have been mediated through an effect on the sexual behaviour of the boar rather than that of the gilt. The male variables most affected were the time to first mount and the duration of ejaculation. Nevertheless, further research is required not only on the factors in the boar pen treatment that are responsible for these effects but also on the sexual behaviour of the gilt, particularly the intact oestrous gilt. The boar pen treatment in this study is similar to the mating conditions of many commercial pigs in that the floor area is small, the floor may become wet and slippery and the walls are wire-mesh and thus the results have considerable practical implications. Research is presently underway at our laboratory examining the effects of mating conditions on farrowing rate and litter size.

Human contact

In modern animal husbandry little scientific regard has been given to the role of the stockperson and, in particular, his or her behaviour, on production. Several recent experiments have shown that pigs displaying high levels of fear of humans, measured

on the basis of the approach behaviour of pigs to a human in a standard test, may experience a chronic stress response (i.e. sustained elevation of plasma free corticosteroids) with detrimental effects on growth and reproductive performance (Gonyou *et al.*, 1986; Hemsworth, *et al.*, 1981a, 1986c, 1987b). In contrast, Paterson and Pearce (1989a,b) and Pearce *et al.* (1989) found that adrenal morphology and growth rate were not adversely affected by pigs displaying moderate to high levels of fear of humans. Highly significant negative correlations have been found in the pig industry between the level of fear of humans by gilts and sows and the farrowing rate and litter size of the farm (Hemsworth *et al.*, 1981b; Hemsworth *et al.*, 1989b), suggesting that fear of humans may be a serious limitation to the reproductive performance of commercial pigs. Further research is required to confirm this.

Climatic environment

Several studies have reported variation between seasons in the oestrus detection rate of gilts. Christenson (1981) observed that a higher proportion of ovulating gilts were undetected in late summer than in the remainder of the year (16.7 and 8.4%, respectively). Cronin *et al.* (1983) reported that in the spring there was a lower percentage of unmated post-pubertal gilts at 35 weeks of age that had not been detected in oestrus than at other times of the year (3.2 and 6.5%, respectively). The effects of photoperiod and temperature are confounded in these two studies.

There is some limited evidence that indicates that increased environmental temperatures may affect sexual behaviour of gilts. In two out of three trials, Warnick *et al.* (1965) reported that a total of 3 out of 13 gilts (23.1%) were not detected in oestrus at an ambient temperature of 32°C although all had ovulated. Increasing the temperature from 27-33°C slightly decreased the percentage of gilts detected in oestrus (100.0 and 91.3%, respectively; Teague *et al.*, 1968), however it is not known if these undetected gilts had ovulated. In only one of a series of experiments reported by Godfrey *et al.* (1983) have increased temperatures (38°C for 10 h and 32°C for 14 h) reduced the percentage of gilts detected in oestrus (21 vs 4% for control). Again it was not determined whether ovulatory activity or oestrus detection was affected. Several studies have reported that the duration of oestrus was reduced by high temperatures (see review by Paterson and Pett, 1987).

Conclusions

There appears to be considerable potential to utilize our present knowledge of pig behaviour to manipulate both the environment and management procedures to improve the sexual behaviour of commercial gilts. Some of the factors that should be considered included the type and amount of boar contact, space allowance of group-housed gilts, mating conditions and human contact.

SYMPOSIUM CONCLUSION

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The main issues that have been highlighted by this symposium are (1) the age and stage of development when gilts should be mated and (2) the pre-mating management that is necessary to facilitate high reproductive performance of gilts. The main conclusions arising within these two issues are as follows:

(1) Age and stage of development at first mating

- * There is little to be gained from mating gilts after 220 days of age: Increasing chronological age beyond 220 days or increasing sexual age in gilts 220 days or older does not improve reproductive performance.
- * Provided gilts are experiencing at least their second oestrous cycle, gilts can be mated at 190-200 days of age.
- * Selected gilts in modern piggeries should attain 110-120 kg by 190-200 days of age and at this body weight, mating at 190-200 days should not produce any detrimental short or long-term effects on reproduction.
- * The influence of body condition at first mating is a contentious issue. Substantial research is required to examine the effects on long-term reproductive performance of body condition at first mating and of the interaction with long-term nutritional strategies. This research may enable nutritional strategies to be developed that will enable gilts to be mated at considerably earlier ages than 190 days without impairing short or long-term performance.

(2) Pre-mating management

- * The best method available to control the onset of puberty in gilts is with the daily exposure to mature boars from about 160 days. Exogenous hormones can induce puberty but their routine use cannot be recommended because subsequent reproductive performance varies widely.
- * Gilts should be *ad libitum* fed a finisher ration for 14 days prior to mating to increase ovulation rates and thus litter size.
- * Pubertal gilts should be housed near but not adjacent to boars and should be housed in groups with at least 2.0 m²/animal to avoid depressions in their sexual behaviour.
- * Research is required on the factors that influence the efficiency of detection procedures that utilize boars. This is an area where gains in the efficiency of oestrus detection of gilts is likely to occur. The present knowledge of factors influencing the efficiency of the back-pressure test is considerable.
- * To ensure a high mating rate it is important to provide pigs with an environment at the time of mating that is conducive to mating. Factors that inhibit or interfere with the sexual behaviour of pigs will reduce mating rate.

References

- BARNETT, J.L., HEMSWORTH, P.H., WINFIELD, C.G. and HANSEN, C. (1986). Effects of social environment on welfare status and sexual behaviour of female pigs. I. Effects of group size. *Applied Animal Behaviour Science*. **16**:49-257.
- BRENDEMÜHL, J.H., LEWIS, A.J. and PEO, E.R. (1987). Effect of protein and energy intake by primiparous sows during lactation on sow and litter performance and sow serum, thyroxine and urea concentrations. *Journal of Animal Science*. **64**:1060-1069.

- BRITT, J.H., ARMSTRONG, J.D. and COX, N.M. (1988). Metabolic interfaces between nutrition and reproduction in pigs. (Proceedings 11th International Congress on Animal Reproduction and Artificial Insemination), pp. 118-125.
- BOOTH, W.D. and BALDWIN, B.A. (1983). Changes in oestrous cyclicity following olfactory bulbectomy in post-pubertal pigs. *Journal of Reproduction and Fertility*. 67:143-150.
- BROOKS, P.H. (1982). The gilt for breeding and for meat. In "Control of Pig Reproduction", pp. 211-224, eds. D.J.A. Cole and G.R. Foxcroft (Butterworths: London).
- BROOKS, P.H. and COLE, D.J.A. (1973). Meat production from pigs which have farrowed. 1. Reproductive performance and food conversion efficiency. *Animal Production*. 17:305-315.
- BROOKS, P.H. and SMITH, D.A. (1980). The effect of mating age on the reproductive performance, food utilization and live weight change of the female pig. *Livestock Production Science*. 7:67-78.
- BURNETT, P.J., WALKER, N. and KILPATRICK, D.J. (1988). The effect of age and growth traits on puberty and reproductive performance in the gilt. *Animal Production*. 46:427-436.
- CHRISTENSON, R.K. (1981). Influence of confinement and season of the year on puberty and estrous activity of gilts. *Journal of Animal Science*. 52:821-830.
- CHRISTENSON, R.K. (1984). Influence of number of gilts per pen on oestrous traits in confinement-reared gilts. *Theriogenology*. 22:313-320.
- CHRISTENSON, R.K. and FORD, J.J. (1979). Puberty and estrus in confinement-reared gilts. *Journal of Animal Science*. 49:743-551.
- COX, N.M., STUART, M.J., ALTHEN, T.G., BENNETT, W.A. and MILLER, H.W. (1987). Enhancement of ovulation rate in gilts by increasing dietary energy and administering insulin during follicular growth. *Journal of Animal Science*. 64:507-516.
- CRONIN, G.M., HEMSWORTH, P.H. and WINFIELD, C.G. (1982). Oestrous behaviour in relation to fertility and fecundity of gilts. *Animal Reproduction Science*. 5:117-125.
- CRONIN, G.M., HEMSWORTH, P.H., WINFIELD, C.G., MULLER, B. and CHAMLEY, W.A. (1983). The incidence of, and factors associated with, failure to mate by 245 days of age in the gilt. *Animal Reproduction Science*. 5:199-205.
- DEN HARTOG, L.A. (1984). The effect of energy intake during rearing on reproductive traits in gilts. *Netherlands Journal of Agricultural Science*. 32:281-292.
- DEN HARTOG, L.A. and NOORDEWIJER, G.J. (1984). The effect of energy intake on age and puberty in gilts. *Netherlands Journal of Agricultural Science*. 32:263-280.
- DEN HARTOG, L.A. and VAN KEMPEN, G.J.M. (1980). Relation between nutrition and fertility in pigs. *Netherlands Journal of Agricultural Science*. 28:211-227.
- DYCK, G.W. (1988). The effect of estradiol benzoate and boar exposure on the occurrence of estrus and ovulation in the gilt. *Canadian Journal of Animal Science*. 68:377-386.
- ENGLISH, P.R., SMITH, W.J. and MacLEAN, A. (1984). "The Sow - Improving Her Efficiency", 2nd edition, pp. 98-115 (Farming Press: Suffolk, UK).
- FLOWERS, B., MARTIN, M.J., CANTLEY, T.C. and DAY, B.N. (1989). Endocrine changes associated with a dietary-induced increase in ovulation rate (flushing) in gilts. *Journal of Animal Science*. 67:771-778.
- GODFREY, N.W., MERCY, A.R. and EMMS, Y. (1983). The effect of high ambient temperature on reproductive performance in gilts. (Proceedings of the Australian Pig Industry Research Committee Workshop on Reproduction: Tasmania, Australia).
- GONYOU, H.W., HEMSWORTH, P.H. and BARNETT, J.L. (1986). Effects of frequent interactions with humans on growing pigs. *Applied Animal Behaviour Science*. 16:269-278.
- HEMSWORTH, P.H. (1982). Social environment and reproduction. In "Control of Pig Reproduction", pp. 585-601, eds. D.J.A. Cole and G.R. Foxcroft (Butterworths: London).
- HEMSWORTH, P.H., BARNETT, J.L., COLEMAN, C.J. and HANSEN, C. (1989a). A study of the relationships between the attitudinal and behavioural profiles of stockpersons and the level of fear of humans and reproductive performance of commercial pigs. *Applied Animal Behaviour Science*. in press.
- HEMSWORTH, P.H., BARNETT, J.L. and HANSEN, C. (1981a). The influence of handling by humans on the behaviour, growth and corticosteroids in the juvenile female pig. *Hormones and Behaviour*. 15:396-403.
- HEMSWORTH, P.H., BARNETT, J.L. and HANSEN, C. (1987b). The influence of inconsistent handling on the behaviour, growth and corticosteroids of young pigs. *Applied Animal Behaviour Science*. 17:245-252.
- HEMSWORTH, P.H., BARNETT, J.L. and HANSEN, C. (1986c). The influence of handling by humans on the behaviour, reproduction and corticosteroids of male and female pigs. *Applied Animal Behaviour Science*. 15:303-314.
- HEMSWORTH, P.H., BARNETT, J.L., HANSEN, C. and WINFIELD, C.G. (1986b). Effects of social environment on welfare status and sexual behaviour of female pigs. II. Effects of space allowance. *Applied Animal Behaviour Science*. 16:259-267.

- HEMSWORTH, P.H., BRAND, A. and WILLEMS, P.J. (1981b). The behavioural response of sows to the presence of human beings and their productivity. *Livestock Production Science*. 8:67-74.
- HEMSWORTH, P.H., CRONIN, G.M., HANSEN, C. and WINFIELD, C.G. (1984). The effects of estrus detection procedures and intense boar stimulation near the time of oestrus on mating efficiency of the female pig. *Applied Animal Behaviour Science*. 12:339-347.
- HEMSWORTH, P.H., HANSEN, C. and WINFIELD, C.G. (1989b). The influence of mating conditions on the sexual behaviour of male and female pigs. *Applied Animal Behaviour Science*. 23:207-214.
- HEMSWORTH, P.H., WINFIELD, C.G., BARNETT, J.L., SCHIRMER, B. and HANSEN, C. (1986a). A comparison of the effects of two oestrus detection procedures and two housing systems on the oestrus detection rate of female pigs. *Applied Animal Behaviour Science*. 16:345-351.
- HEMSWORTH, P.H., WINFIELD, C.G., TILBROOK, A.J., HANSEN, C. and BARNETT, J.L. (1988). Habituation to boar stimuli: Possible mechanism responsible for the reduced detection rate of oestrous gilts housed adjacent to boars. *Applied Animal Behaviour Science*. 19:255-264.
- HOVELL, F.D.DeB., MacPHERSON, R.M., CROFTS, R.M.J. and PENNIE, K. (1977). The effect of energy intake and mating weight on growth, carcass yield and litter size of female pigs. *Animal Production*. 25:233-245.
- HUGHES, P.E. (1982). Factors affecting the natural attainment of puberty in the gilt. In "Control of Pig Reproduction", pp. 117-138, eds. D.J.A. Cole and G.R. Foxcroft (Butterworths: London).
- HUGHES, P.E. and COLE, D.J.A. (1976). Reproduction in the gilt. 2. The influence of gilt age at boar introduction on the attainment of puberty. *Animal Production*. 23:89-94.
- HUGHES, P.E., HEMSWORTH, P.H. and HANSEN, C. (1985). The effects of supplementary olfactory and auditory stimuli on the stimulus value and mating success of the young boar. *Applied Animal Behaviour Science*. 14:245-252.
- HUGHES, P.E., PEARCE, G.P. and PATERSON, A.M. (1989). Mechanisms mediating the stimulatory effects of the boar on gilt reproduction. In "Control of Pig Reproduction III", in press, eds. G.R. Foxcroft, D.J.A. Cole and B.J. Weir (Journal of Reproduction and Fertility: Cambridge).
- KING, R.H. (1989). Effects of body weight and body composition of gilts at 24 weeks of age on subsequent reproductive performance. *Animal Production*. 49:in press.
- KING, R.H., CLEARY, G.V., MAUGHAN, N. and POWER, C. (1984). The effect of initial fat reserves of gilts on their subsequent reproductive performance. *Proceedings Australian Society of Animal Production*. 15:702.
- KING, R.H. and DUNKIN, A.C. (1986). The effect of nutrition on the reproductive performance of first-litter sows 4. The relative effects of energy and protein intakes during lactation on the performance of sows and their piglets. *Animal Production*. 43:319-325.
- KING, R.H. and WILLIAMS, I.H. (1984). The influence of ovulation rate on subsequent litter size in sows. *Theriogenology*. 21:677-680.
- KIRKWOOD, R.N. and AHERNE, F.X. (1985). Energy intake, body composition and reproductive performance of the gilt. *Journal of Animal Science*. 60:1518-1529.
- KIRKWOOD, R.N., BELTRANENA, E. and AHERNE, F.X. (1986). The influence of plane of feeding on the onset of puberty and ovulation rates at first and second estrus in gilts. *Canadian Journal of Animal Science*. 66:1178-1179.
- KIRKWOOD, R.N. and HUGHES, P.E. (1979). The influence of age at first boar contact on puberty attainment in the gilt. *Animal Production*. 29:231-238.
- KNOTT, R.E., ENGLAND, D.C. and KENNICK, W.H. (1984). Estrus, ovulation, conception and embryo survival in confinement-managed gilts of three weight groups. *Journal of Animal Science*. 58:281-284.
- LEGAULT, C. and DAGORN, J. (1973). Incidence de l'age a la premiere mise-bas sur la productivite de la truie. *Journées Recherche Porcine en France*. 1973, pp. 227-237, (L'Institut Techniique du Proc: Paris).
- MacPHERSON, R.M., HOVELL, F.D.DeB. and JONES, A.S. (1977). Performance of sows first mated at puberty or second or third oestrus, and carcass assessment of once-bred gilts. *Animal Production*. 24:333-342.
- OMTVEDT, I.T., STANISLAW, C.M., and WHATLEY, J.A. (1965). Relationship of gestation length, age and weight at breeding and gestation gain to sow productivity at farrowing. *Journal of Animal Science*. 24:531-535.
- PATERSON, A.M. (1982). The controlled induction of puberty. In "Control of Pig Reproduction", pp. 139-159, eds. D.J.A. Cole and G.R. Foxcroft (Butterworths: London).
- PATERSON, A.M., HUGHES, P.E. and PEARCE, G.P. (1989a). The effect of limiting the number of days of contact with boars, season and herd of origin on the attainment of puberty in gilts. *Animal Reproduction Science*. 18:293-301.
- PATERSON, A.M., HUGHES, P.E. and PEARCE, G.P. (1989b). The effect of season, frequency and duration of contact with boars on the attainment of puberty in gilts. *Animal Reproduction Science*. in press.

- PATERSON, A.M. and LINDSAY, D.R. (1980). Induction of puberty in gilts. 1. The effects of rearing conditions on reproductive performance and response to mature boars after early puberty. *Animal Production*. 31:291-297.
- PATERSON, A.M. and LINDSAY, D.R. (1981). Induction of puberty in gilts. 2. The effect of boars on maintenance of cyclic activity in gilts induced to ovulate with PMSG and HCG. *Animal Production*. 32:51-54.
- PATERSON, A.M. and PEARCE, G.P. (1989a). Boar-induced puberty in gilts handled pleasantly or unpleasantly during rearing. *Applied Animal Behaviour Science*. 22:225-233.
- PATERSON, A.M. and PEARCE, G.P. (1989b). Seasonal effects on puberty in the gilt. In "Control of Pig Reproduction III", in press, eds. G.R. Foxcroft, D.J.A. Cole and B.J. Weir (Journal of Reproduction and Fertility: Cambridge).
- PATERSON, A.M., PEARCE, G.P., FOXCROFT, G.R. and REED, H.C.B. (1984). Reproductive performance of gilts induced into puberty with oestradiol benzoate or a combination of pregnant mare's serum gonadotrophin and human chorionic gonadotrophin. *Animal Production*. 38:121-128.
- PATERSON, A.M. and PETT, D.H. (1987). The role of high ambient temperature in seasonal infertility in the sow. In "Manipulating Pig Production", pp. 48-52, ed. APSA Committee (Australasian Pig Science Association: Werribee, Victoria, Australia).
- PAY, M.G. and DAVIES, T.E. (1973). Growth, food consumption and litter production of female pigs mated at puberty and at low body weights. *Animal Production*. 17:85-91.
- PEARCE, G.P., PATERSON, A.M. and PEARCE, A.N. (1989). The influence of pleasant and unpleasant handling and the provision of toys on the growth and behaviour of male pigs. *Applied Animal Behaviour Science*. 23:27-37.
- SIGNORET, J.P. (1970). Swine behaviour in reproduction. In "Effect of Disease and Stress on Reproductive Efficiency in Swine", pp. 28-45, (Extension Service, University of Nebraska: Nebraska, USA).
- SIGNORET, J.P. and MAULEON, P. (1962). The effect of surgical removal of the olfactory bulbs on the sexual cycle and the genital tract of sows. *Annales de Biologie Animale Biochimie Biophysique*. 2:167-174.
- STRANG, G.S. (1970). Litter productivity in Large White pigs. 1. The relative importance of some sources of variation. *Animal Production*. 12:225-233.
- VAN LUNEN, T.A. and AHERNE, F.X. (1987). Effect of long-term restriction on age at puberty of gilts. *Canadian Journal of Animal Science*. 67:797-801.
- WALKER, I.J. (1982). Reproductive targets and problems in commercial pigs. *Proceedings of the Australian Society of Animal Production*. 14:238-241.
- WALKER, N. and BURNETT, P.J. (1984a). The management of replacement breeding gilts. (Annual Report of the Agricultural Research Institute of Northern Ireland), pp. 14-20.
- WALKER, N. and BURNETT, P.J. (1984b). The induction of puberty in rapidly grown gilts. *Proceedings of the British Society of Animal Production*. 1984, Abstract 84.
- WARNICK, A.C., WALLACE, H.D., PALMER, A.Z., SOSA, E., DVERRE, D.J. and CALDWELL, V.E. (1965). Effect of temperature on early embryo survival in gilts. *Journal of Animal Science*. 24:89-95.
- WARNICK, A.C., WIGGINS, E.L., CASIDA, L.E., GRUMMER, R.H. and CHAPMAN, A.B. (1951). Variation in puberty phenomena in inbred gilts. *Journal of Animal Science*. 10:479-493.
- WHITTEMORE, C.T., FRANKLIN, M.F. and PEARCE, B.S. (1980). Fat changes in breeding sows. *Animal Production*. 31:183-190.
- YANG, H., EASTHAM, P.R., PHILLIPS, P. and WHITTEMORE, C.T. (1989). Reproductive performance, body weight and body condition of breeding sows with different body fatness at parturition, differing nutrition during lactation, and differing litter size. *Animal Production*. 48:181-201.
- YANG, H., RODWAY, R.G. and VARLEY, M.A. (1987a). The influence of different doses of oestradiol benzoate on the attainment of puberty in the gilt. *Animal Production*. 44:285-291.
- YANG, H., VARLEY, M.A. and RODWAY, R.G. (1987b). Effect of allyl-trenbolone on the attainment of puberty in gilts treated with oestradiol. *Animal Production*. 45:503-510.

AUTHOR INDEX

Adamson, D.	71
Adler, B.	256
Anderson, L.M.	186, 188
Annison, E.F.	207, 208
Baglin, M.J.	276
Baigent, D.R.	186, 188
Bates, J.	274
Barnett, J.L.	211
Batterham, E.S.	26, 137, 184, 185
.....	186, 187, 188, 209, 260
Bauman, D.E.	70
Baxter, S.H.	102, 191
Beech, S.A.	26
Bell, A.W.	70
Bird, P.H.	100, 116
Black, J.L.	207, 208, 209
Blackall, P.J.	275
Blackshaw, A.	68
Blackshaw, J.K.	210
Bloor, R.J.	303
Boyd, R.D.	70
Brown, R.W.	303
Brown, S.C.	276
Campbell, R.G.	56, 170
Chappel, R.J.	256
Chin, J.C.	237, 238, 260
.....	263, 265
Clarke, W.A.	137, 184
Close, W.H.	302
Cole, D.J.A.	281
Coloe, P.J.	273
Cook, R.W.	260, 263
Cranwell, P.D.	140, 212, 308
Cronin, G.M.	101, 110, 127
Cutler, R.S.	122, 256
Davies, W.	29
Dettmann, E.B.	187
Dirnbauer, J.	56
Dove, H.	98
Dunshea, F.R.	70
Eaves, L.E.	275
Eamens, G.J.	249
Egan, A.F.	46
Elliott, R.	26
Evans, D.F.	303
Evans, G.	306

Farrell, D.J.	187, 190
Fegan, M.	275
Fernandez, J.	68
Forage, R.G.	303
Fyfe, A.R.	222
Gannon, N.J.	136
Geldard, H.	273
Gerdes, R.	190
Gerraty, N.L.	273
Giles, L.R.	207, 208, 209
Goddard, M.E.	216, 234
Gogolewski, R.P.	260, 263
Gooden, J.M.	207, 208
Greenwood, P.E.	303
Hampson, D.J.	246
Harris, D.M.	70
Harris, P.V.	71
Hartmann, P.E.	72, 99, 100, 116
Haskell, M.J.	215
Hemsworth, P.H.	304, 309, 319, 323
Hennessy, D.P.	212, 308
Holmes, M.A.	72, 116
Hughes, I.P.	29, 276
Hughes, P.E.	277, 290, 296, 307
Huglin, J.	27
Hungerford, J.W.	303
Hutson, G.D.	213, 214, 215
Hutton, K.	138, 176
Jones, R.T.	256
Ketaren, P.P.	187
King, R.H.	69, 98, 315
Krautil, F.L.	38
Leibholz, J.	135, 136
Long, T.	217
Love, R.J.	276, 306
Luxford, B.G.	56, 214
Mawhinney, H.	28
McAlpine, B.	26
McNamara, P.S.	70
McPhee, C.P.	225
Millar, B.D.	256
Moran, C.	29
Morgan, I.R.	38
Morris, S.J.	100
Moughan, P.J.	140
Mullan, B.P.	285, 302
Neill, A.R.	189
Nicholas, F.W.	29, 276

Oksbjerg, N.	68
O'Shea, J.M.	56
Partridge, I.G.	160
Paterson, A.M.	305, 310
Peacock, A.J.	306
Pearce, G.P.	290, 306, 307
Pearson, G.	66, 67
Peters, R.T.	189
Prime, R.W.	122
Purchas, R.W.	66, 67
Rogers, R.J.	275
Saini, H.S.	185
Shay, B.J.	46
Sheldrake, R.F.	242
Shorthose, W.R.	31, 71
Smith, D.R.	56
Smith, M.	27
Smith, N.A.	99
Smith, W.C.	66, 67
Spicer, E.M.	122
Takken, G.	28
Tarrant, P.V.	1
Thornton, R.F.	30, 31, 61, 71, 189
Tilbrook, A.J.	304
Toner, M.S.	98
Treacy, D.A.	229
Trueman, K.F.	275
Tsonis, C.G.	303
Tucker, R.G.	207, 208
Wan, S.S.	308
Wang, Y.H.	135
Warner, R.D.	69
White, E.	188
Wilkinson, J.L.	214
Williams, I.H.	285
Williams, K.C.	71, 189
Willis, G.L.	276
Xian, J.	190
Zhang, S.H.	212