MANIPULATING PIG PRODUCTION VII

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1. Swine - Australasia - Congresses. 2. Swine - Australia - Congresses. 3. Swine - Research - Congresses. 4. Swine - Production - Congresses. 5. Swine - Nutrition - Congresses. 6. Swine - Genetics - Congresses. 7. Swine – Metabolism – Conferences. 8. Swine – Physiology – Congresses. 9. Swine - Reproduction - Congresses. 10. Swine - Health - Congresses. 11. Meat - Quality - Congresses. 12. Agriculture - Environment - Congresses. I. Cranwell, P.D. Peter Donald, 1937 - , II. Title.

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CONTENTS

A.C. DUNKIN MEMORIAL LECTURE

VARIATION IN PIG PRODUCTION

HOUSING AND ENVIRONMENT

ONE-PAGE PAPERS:

NUTRITIVE VALUE OF FEEDSTUFFS AND ENZYMES

ONE-PAGE PAPERS:

GENETICS, ANIMAL BREEDING AND BEHAVIOUR

SYMPOSIUM I: Development and implementation of genetic improvement technologies in pig breeding

ONE-PAGE PAPERS:

REPRODUCTION AND NUTRITION

ONE-PAGE PAPERS:

GENETICS AND MEAT QUALITY

REVIEW III

ONE-PAGE PAPERS

PHYSIOLOGY, ENDOCRINOLOGY, NUTRITION AND GROWTH

ONE-PAGE PAPERS:

MANAGEMENT OF THE YOUNG PIG

REVIEW IV:

LIPID METABOLISM

REVIEW V:

ONE-PAGE PAPERS:

MEAT QUALITY AND BODY COMPOSITION

ONE-PAGE PAPERS:

ANIMAL HEALTH

SYMPOSIUM II: Antibiotics in pig production

ONE-PAGE PAPERS:

NUTRIENT UTILIZATION, PHYSIOLOGY AND HEALTH

REVIEW VI:

ONE PAGE PAPERS

ANIMAL HEALTH AND WASTE UTILIZATION

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Conference sponsors, listed below, are gratefully acknowledged for their continued financial support of APSA. For the first time, the Pig Research and Development Corporation has taken the position as Principal Sponsor of the conference and without their inputs it is unlikely the APSA conference would be possible.

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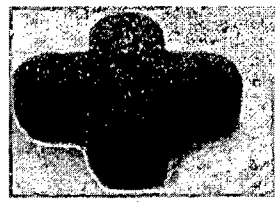

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ACKNOWLEDGEMENT TO REFEREES

The Proceedings, 'Manipulating Pig Production VII', contains 106 one-page papers, six Reviews and two Symposia, a total of 279 pages. As is the policy of the Association, all one-page papers, Reviews and papers in Symposia were reviewed by external referees (at least two per paper). The committee of APSA and the editor gratefully acknowledges the assistance generously given during 1999 by the following referees and by those who wish to remain anonymous or who were inadvertently omitted from the list.

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PREFACE

Manipulating Pig Production VII records the proceedings of the Seventh Biennial Conference of the Australasian Pig Science Association (APSA). The APSA conferences are now seen as a major pig research event on the world research calendar and are well attended by leading scientists from Australia and overseas. The APSA conferences bring together a unique collection of minds that in the past have successfully combined their expertise to solve some very complex problems faced by the Australasian pig industries. The proceedings from the conference represent a very concise and informative summary of the status of Australasian pig research. To this end, APSA represents a significant asset to the Australasian pig industries.

Now that the new millennium is nearly here, APSA, like many other associations must reassess its role to ensure that its existence is relevant and justified. As many Australian pig producers clearly recognise the need to remain internationally competitive, there will always be a leading role for pig science. However, the funds available for research, the number of scientists available to conduct the research, and the resources directed towards pig research are under continual pressure. For these reasons I believe the role of organisations such as APSA will become even more significant if the current standard of pig research is to be maintained. These comments are endorsed by that fact that a dwindling number of pig scientists have still managed to submit more than 116 one-page papers to APSA in 1999 clearly indicating the value they place on this Association.

We are proud of the many new innovations introduced at the 1999 conference. A key emphasis has been on the active discussion, and in some instances debate, of presented research findings. Pre-conference workshops, an extended conference program and the introduction of short presentations to support poster presentations ensure there is adequate time for interaction among scientists.

Once again, the research contained within Manipulating Pig Production VII represents the cutting edge of pig production technology. The standard of the papers contained within has been maintained at an extremely high level by the dedicated efforts of the editor, Mr P.D. Cranwell. In 1999 APSA has also introduced the APSA Medal for the best first time presentation at the conference in addition to an APSA Best Poster Award, the outcome being research presentations at the highest possible level.

The Batterham Memorial Award, in honour of the contributions to pig science made by the late Dr E.S. Batterham, is now well established as a highly prestigious award for young pig scientists. The award is made possible by the generous financial support from a number of national and international companies including: BASF, Ridley Agriproducts (including Farmstock), Heartland Lysine (US), Purina Mills (US) and Rhone Poulenc Animal Nutrition.

Many individuals devote a large amount of time to ensure the APSA conference meets the high expectations of pig scientists across the Australasia. I would like to thank the Organising Committee for their efforts over the past two years. The committee consisted of: Dr Colin Cargill (Secretary), Dr Heather Bray (Treasurer), Dr Neil Gannon (Vice President), Dr Frank Dunshea (Past President), Dr John Pluske, Dr Susanne Hermesch, Dr Ian Williams and Dr John Hargreaves. Dr Lyndy Scott and Dr Tony Peacock represented the Pig Research and Development Corporation on the committee over the two years and along with Mr Peter Cranwell were recognised as non-voting members. I am also extremely grateful to Ms Donna Bowden and Ms Janine Benson who were employed through the BECAN Consulting Group to act as the APSA Secretariat. Financial inputs from the many sponsors of APSA in 1999, particularly the Pig Research and Development Corporation are also gratefully acknowledged.

Dr R.J. van Barneveld
President – Australasian Pig Science Association.

A REVIEW - PIG RESEARCH AND DEVELOPMENT: WHAT NEEDS TO BE DONE? WHO SHOULD PAY AND WHO SHOULD DO THE WORK?

W.H. Close

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Abstract

Research has greatly contributed to the considerable improvement in the efficiency of pig production during the past decades, but the rate of improvement in several traits has remained static in recent years. Performance in commercial practice is still well below the genetic capability of the modern animal and solutions must be found to improve productive efficiency, so that pig meat can compete successfully with other meat products. Research should continue to provide a greater understanding of the metabolic, physiological, endocrine and immunological control of growth, reproduction and health status. Consideration must also be given to the effects of production practices on the environment, on improving meat quality and animal welfare and take account of new and emerging technologies. To achieve this, there is a need to initiate applied, strategic, and basic or fundamental research programmes. In the future, it is likely that applied research will be minimally funded from the public purse, but will increasingly depend upon industry funding, since it is the end-user of the results that has most to benefit. In terms of strategic research, an interesting innovation could be the development of a 'stakeholder' or partner-type approach, which will involve both public and private funds and will include all sectors of the industry, including retailer and consumer organisations. Public funding may become further limited and will be increasingly channelled towards innovative and basic research to deepen our understanding of the biology of the pig, and for research related to proposed or newly-implemented legislation, including animal welfare. A balance of applied, strategic and basic research is therefore required with short, medium and long-term objectives to improve the pig industry's efficiency, so that it can compete successfully in the livestock markets of today and tomorrow.

Introduction

During the past 40 years, great progress has been made in the field of animal production in general, and pig production in particular. The unending priority of feeding an ever-increasing world population and providing high quality protein to ensure adequate nourishment has made meat an essential and staple component of diets for humans. In many countries, pig meat has provided the majority of the protein needs of many individuals. Pig meat production has therefore played a major role and has become a global industry in meeting the nutritional needs and desires of the human population.

Research has greatly contributed to this advancement by providing a greater understanding of the biology of the pig and by adapting and applying this knowledge to improve the efficiency of pig production (Table 1). The largest improvements have been in the reproductive performance of the sow and in the overall efficiency with which feed is utilised to promote carcass gain. This has been achieved largely by advances in the genetic potential of the animal for lean tissue growth rate and reproductive traits, improvements in nutritional knowledge and dietary formulation, developments in management, husbandry, housing and stockmanship, as well as a better understanding of the health care requirements of the animal at the different stages of development. Nevertheless, the rate of improvement in several traits over the last 10 years has remained relatively static.

Despite these developments, the performance of pigs on many farms is well below their genetic capability. For example, the modern growing/finishing pig has a potential maximum protein gain of 200-240 g/day (Rao and McCracken, 1990, 1991; Van Lunen and Cole, 1998) and this equates to a growth potential of 1.2-1.3 kg/day. In commercial

practice, the growth rate is often well below 0.80 kg/day (MLC, 1999). Thus, actual performance may be only 50-60% of the animal's true genetic potential. Similarly, in terms of reproduction, the modern hyper-prolific sow should be capable of rearing 30 piglets per year (2.5 litters of 12 piglets), yet in many countries 20-22 piglets per sow per year is the norm. This is a 25-30% loss in reproductive potential. Indeed, one interesting observation from Table 1 is the minor improvement that has been achieved in actual litter size over the past 35 years: from 9.8 to 10.9 piglets born per litter. Most advances have been achieved through improved management and husbandry practices, resulting in more litters per sow per year.

Table 1. Changes in pig performance in the UK since 1960.

	1960	1965	1970	1975	1980	1985	1990	1995	1999
Piglets born/litter	9.8	10.0	10.3	10.3	10.3	10.5	10.7	10.8	10.9
Litters/sow/year	1.74	1.85	1.95	2.00	2.14	2.26	2.23	2.25	2.24
Pigs reared per sow/year	13.3	14.9	16.3	16.9	18.3	20.2	21.1	21.6	21.9
FCR (kg/kg)	3.96	3.79	3.85	3.63	3.25	2.90	2.69	2.58	2.61
Overall herd feed requirements (kg/kg carcass weight)	6.18	5.67	5.58	5.27	4.81	4.22	4.10	3.80	3.81

Sources: Ridgeon (1993); MLC (1995-1999).

Demands are made on the pig industry from all sides: the consumer wants a high quality, nutritious, healthy product at a competitive price. Production has to be transparent, traceable, under a high welfare system, with minimal impact on the environment and of course, still remain profitable for the producer. Can research and development help to satisfy these needs? Can it be directed to enhance performance, reduce costs, improve meat quality and ensure the highest welfare and environmental standards? Where is it most successfully applied and who is to administer and fund it and of course, what needs to be done? This paper attempts to provide information on these areas that are of importance to practical pig production. However, it is not the intention to cover all aspects of research and development needs, but to limit the discussion to those areas of most importance and where most potential benefits are likely, including legislative requirements.

What should be done?

Growth rate and lean tissue gain: Effects of health status

For an animal to achieve its maximum rate of growth under commercial conditions it is essential that protein or lean tissue gain is optimized. Thus, factors that limit lean tissue gain have a major effect on both the rate and efficiency of growth. Although the effects of nutrition, housing and management are well documented, a major current limitation to the high growth potential of the modern pig genotypes is poor health status or disease (Carr *et al.*, 1998). The identification of these limiting factors has resulted in the development of systems of disease management, such as 'all in – all out' production systems, split-site weaning and partial depopulation, in order to enhance the health status of the herd and hence improve growth rate and feed efficiency (Muirhead and Alexander, 1997).

Raising the health status of pigs on commercial farms not only unlocks the high growth potential and improves the efficiency of production, but also enhances pig welfare and since nutrient utilisation is improved, less nutrients are excreted and there is less risk of environmental pollution. The major targets for improvements in pig health should therefore be the conditions that enhance both gastrointestinal and respiratory health. A better understanding of the mechanism of disease at both a metabolic and a physiological

level and its effects on production are required, as well as a more effective way to assess health status on farm, so that the most appropriate dietary regime may be adopted.

It is now known that when the immune system of the animal is challenged there is a cascade of events, which invokes a series of metabolic and endocrine adjustments that have consequences for growth and development. Initially cytokines are released, which activate both cellular and hormonal responses and alter several endocrine and metabolic pathways (Klasing, 1988). Catabolic hormones are elevated, whereas anabolic hormones are inhibited. Body protein synthesis is reduced and protein degradation is increased during periods of antigen exposure. This results in a reduced rate of protein utilisation and a lower voluntary feed intake (Stahly, 1996). Consequently, protein gain and growth rate is reduced, feed is less efficiently utilized and carcass fatness is increased (Williams *et al.*, 1997a and 1997b).

Another important consequence of the inflammatory response is the release of acute phase proteins by the liver. Acute phase protein responses are typically associated with more profound immunological insults, such as acute infection or trauma, and it is therefore interesting that they increase during such processes as weaning in the piglet (Gaskins, 1998). Some of these acute phase proteins may increase several-fold and Reeds *et al.* (1994) have suggested that this may represent a major pathway of amino acid use which partially accounts for the increase in muscle protein turnover and even loss of nitrogen that accompanies immunological response.

Since the enhancement of lean tissue growth is fundamental to the success of pig production, it may be that new methodologies and technologies need to be developed to regulate and improve the efficiency of growth in pigs. This may involve exogenous modifiers and currently growth enhancers, such as somatotropin, β-agonists and insulin-like growth factor I (IGF-I) are available (Dunshea and Walton, 1995). Alternative methods may involve genetic manipulation using DNA technology to alter the genome of the animal to specifically manipulate its endocrine system (transgenics), or immunomodulation in which immunity is enhanced to produce long-term stimulation of the animal's own hormone production (McCauley *et al.*, 1995). Although these are emerging techniques that offer considerable potential, it may well be that in many countries their use may be restricted because of ethical, welfare and socio-economic circumstances.

Improving reproductive efficiency

Efficient sow performance on many commercial farms means a sow producing 2.0-2.4 litters and rearing 20-24 piglet per year. This is some way short of the known biological potential. However, any improvement must be sought through increasing litter size, since much of the improvements in reproductive efficiency have been brought about by increases in the number of litters produced per sow per year, which is nearing the maximum possible (Table 1). Litter size is the product of ovulation rate and perinatal survival. Hence, rapid improvement in the number of piglets born alive per litter could be made if both ovulation rate and embryo/foetal survival could be improved simultaneously. Although ovulation rate is highly heritable (45%), selection for that trait results in only a small improvement in the number of piglets born per litter since perinatal survival is lowly heritable (about 10%) (Johnson *et al.*, 1985, 1999). A major limitation is therefore the high level of embryo mortality that occurs, with 30-40% of fertilised eggs failing to develop into embryos and survive to term.

Several reports suggest that the development of the pig conceptus to day 11 of gestation contributes to embryo mortality by creating divergence of embryo development within litters (Gordon, 1997). Indeed, embryo diversity and embryo mortality may be the result of follicular heterogeneity. Variation in the endocrine environment may cause such diversity and Blair *et al.*, (1994) found that animals with high embryo survival on day 11 of gestation tended to exhibit less variation in embryo development than pigs with low embryo survival. Thus, if litter size is to be improved, it may well be that greater attention needs to be paid to the endocrine environment in early pregnancy and the inter-relationship among embryos. There is evidence that slow-developing embryos may be

eliminated as a consequence of the changes in the uterine environment provoked by the fastest-growing embryos and this needs to be further elucidated (Hyttel *et al.*, 1994).

The study of Chinese breeds of pigs, with their known superior reproductive efficiency, may possibly help our understanding of the mechanism for hyperprolificacy. The difference in litter size between the Chinese and the traditional breeds of sow ranges from higher ovulation rate and lower embryonic mortality to greater uterine capacity and enhanced placental efficiency (Webb, 1994). More recently, a genetic marker for this increased performance with favourable alleles for oestrogen receptors, has been detected in Chinese x USA cross sows, and when selected, resulted in two extra piglets born alive per litter (Rothschild *et al.*, 1996). The markers for such quantitative traits loci (QTLs) may therefore be used in future to improve the reproductive efficiency of sows.

Although optimum biological or economic efficiency is not realized until the sow reaches her sixth parity, few animals actually achieve their sixth parity; the norm is 3-4 litters per sow lifetime. Premature reproductive failure, that is failure to conceive and re-breed, is therefore a major cause for disposal on many commercial units and accounts for over 50% of the total animal losses. The economic loss from premature culling represents a major cost burden to the pig industry. There is evidence from several studies of a relationship between reproductive performance, nutrition and the balance of the lean and fat in the animal (King and Williams, 1984; Mullan and Williams, 1989; Gaughan *et al.*, 1995; Whittemore, 1996). Genetic selection for increased leanness conflicts with the requirements for adequate fat reserves in the young sow. There is a need to examine the extent to which the manipulation of body lean and fat in the young gilt at first mating influences long-term reproductive performance.

Environmental constraints: Improving nutrient utilisation

In many countries, there is concern about the effects of pig production on water, soil and air quality. This has arisen from the concentration and intensification of pig production in particular areas and the dangers of excessive quantities of elements, such as nitrogen and phosphorus, entering the soil and aquifers. Large quantities of carbon dioxide, ammonia, methane and other volatile gases are lost to the atmosphere. This has led to restrictions in some countries on the quantities of animal waste being produced, as well as regulations regarding the concentration of gases to which stockmen and animals may be exposed. Indeed, one way to improve the image and value of pig production is to produce meat in ways that benefit the environment.

A pig consumes 8 - 9 kg of nitrogen between weaning and slaughter at 100 kg body weight. Less than 3 kg is absorbed and deposited as lean tissue within the body and the remaining 5 - 6 kg is excreted, of which 33% is in faeces and 67% in urine. Tamminga and Verstegen (1992) have calculated that, for example, in the Netherlands, some 207×10^3 tonnes of N were consumed annually by all classes of pigs. Of this 159×10^3 tonnes were excreted in faeces and urine and the remaining 48×10^3 tonnes were therefore deposited in the body. This represents an efficiency of nitrogen retention of 23%. Some 20.7×10^3 tonnes were associated with ammonia in the storage of the excreta and a further 25.9×10^3 tonnes were lost as N during spreading on the soil. Thus, 46.6×10^3 tonnes or 22.5% of the total nitrogen consumed by the animal disappeared directly into the atmosphere with obvious environmental impact. It would therefore benefit both the livestock producer and the environment if nitrogen and phosphorus excretion from animals could be kept to a minimum.

A number of strategies can be used to minimize surplus nitrogen consumption by the animal. These include:

- More careful balancing of the nutrients in the diet to meet the needs of the animal.
- The adoption of phase feeding techniques.
- The improvement in the quality of dietary protein, so that the balance of amino acids more closely matches the 'ideal protein'.
- Reducing the content of crude protein, but maintaining the content of digestible amino acids in the feed.

- The use of performance enhancers to improve the utilization of dietary protein and amino acids, and the use of other products to regulate intestinal flora and limit the bacterial degradation of protein.
- The modification of raw materials by processing techniques, such as expansion and other means, so that metabolic wastage is reduced, including the reduction of anti-nutritional factors.

One of the major developments in recent years has been the use of enzymes to improve the digestibility of energy, protein and phosphorus in the feed. Enzymes can be targeted at the non-starch polysaccharide (NSP), protein and phosphorus fractions of the diet. Indeed, enzymes such as xylanase, arabinase, cellulase and alpha-galactosidase, which are aimed at breaking down fibrous materials, will indirectly lead to improved efficiency of protein and amino acid utilization. A higher proportion of the proteins and amino acids are released and used for growth and production purposes, rather than being voided by the animal. The main effects of the simple addition of enzymes to pig diets are shown in Table 2. Similarly, the addition of phytase enzymes has not only shown significant improvements in phosphorus digestibility, but significantly higher growth rates and efficiency of feed utilisation have also been reported (Jongbloed *et al.*, 1991; Dierick and Decuypere, 1994).

Enzymes may also be used to 'detoxify' plant material, or reduce the content of harmful substances within them; e.g., phytase in rapeseed, alpha-galactosides in soya beans, gossypol in cotton seed meal. Similarly, as the wet feeding of pigs becomes popular, enzyme treatment, water soaking and fermentation could become more important treatments in the future.

Table 2. Magnitude of the effect of adding specific enzymes to pig diets.

	Growth rate	Feed efficiency
Protease	small	small
Amylase	1.04[1]	0.96[1]
Pentosanase	1.05	0.95
β-glucanase	1.01	0.98
Cellulase/hemicellulase	1.03	0.91
Blend of amylase, proteinase and β-glucanase	1.09	0.93

[1]Values are ratios of treatment:control (Dierick and Decuypere, 1994).

Optimizing product quality

Meat quality has assumed greater importance in recent years. People are more conscious of health matters, are more demanding and critical of product quality and, since pigs have become progressively leaner, more complaints have been received that the eating quality of pig meat has declined. Pig producers need to be aware that quality assessment will become increasingly important.

The objective must therefore be to produce pig meat of consistently good eating quality, with desirable flavour and good keeping quality. Although breed, sex, rate of growth, as well as environmental factors all influence meat eating quality, the diet fed to the animal, as well as the conditions under which it has been kept, have a very significant effect.

The aspects of meat quality that are of most interest are colour and water-holding capacity of the muscle, fat firmness and eating quality, all of which can be manipulated by nutrition. For example, several studies have shown that the fatty acid component of dietary fats affects the flavour and composition of carcass fat (Wood and Enser, 1989). The composition and quality of intramuscular fat (marbling fat) is influenced in a similar manner. *Ad libitum*, rather than restricted feeding, tended to produce more tender, juicier meat (MLC, 1989). This was further enhanced by the feeding of diets with high energy and low protein content just prior to slaughter (Blanchard *et al.*, 1999).

The importance of skatole and indole in odour and flavour perception in pig meat has been reported by Madsen *et al.*, (1990). Skatole is produced by microbial degradation of tryptophan in the hindgut and dietary fibre has been suggested as a contributory factor. However, the type of fibre may be important and the feeding of sugar beet pulp, a material which contains high levels of soluble fibre or NSP monomers, actually reduced the concentration of skatole and increased the overall liking and acceptability of pig meat (Wood *et al.*, 1994). It is not clear how this occurred, but contributory factors may be: reduced breakdown of tryptophan by bacteria, reduced absorption of skatole from the gut and the lowering of the pH in the large intestine to a value that suppressed both the growth and activity of the microflora (Jensen *et al.*, 1995).

Specific dietary ingredients and feeding strategies must therefore be developed on a 'designer diet principle' to ensure not only optimum animal growth and feed efficiency, but also to promote the organoleptic and eating qualities of pig meat for a more discerning and health-conscious consumer. The choice of dietary ingredients and their quality will become of increasing importance to assure the consumer that adequate food safety measures are in place.

Animal welfare

In future, more consideration must be given to the effects of nutrition and management on the welfare of the animal. This is especially relevant to sows during pregnancy, which are normally fed well below their voluntary feed intake or appetite potential during pregnancy. Such regimes may meet production objectives, but they may not provide good welfare, since the animal does not feel satiated and abnormal behaviour patterns, like bar chewing and polydipsia, may develop. It has been known for some time that the occurrence of abnormal behaviour in confined systems is related to feed intake (Appleby and Lawrence, 1987) and stereotypic behaviour is considerably reduced by high feed allowances (Meunier-Salaün, 1999). Recent legislation in some European countries banning the use of systems of confinement has necessitated the development of group-housing systems and this has created additional problems for the feeding of the sow. Group-feeding during pregnancy can cause considerable aggression at feeding time and may result in the unequal distribution of feed among sows. Dominant sows consume more feed and achieve higher than expected weight gains, whereas subordinate sows do not eat sufficient feed to maintain body condition. Brouns and Edwards (1994) demonstrated that low-ranking animals achieved only 50% of the gain of high-ranking animals when floor-fed in a group-housing situation. However, when a low density, bulky diet was provided *ad libitum*, then all the animals' weight gain was similar and on target.

It may be possible to use different types of materials and feeding schedules that can induce feelings of satiation at acceptable nutrient intakes and improve sow welfare. One such material is sugar beet pulp, which appears to have unique properties. When included at high inclusion rates (60%) in the diet, abnormal behaviour was considerably reduced and aggressiveness was minimal, yet the intake of the animal was sufficient to meet its daily nutrient needs (Edwards, 1993). Interestingly, when fed *ad libitum*, the intake of the animal increased with duration of gestation and in keeping with its increased metabolic requirements (Sadler *et al.*, 1994). The use of such materials and the development of *ad libitum* systems of feeding sows during pregnancy will therefore become of increasing importance in the future. Such systems should induce satiation, remove competition and improve welfare of the sows, yet be simple and cheap to operate.

Emerging technologies

The future competitiveness of the pig industry will be influenced by the speed of implementation of new and emerging technologies, especially biotechnology. This is a rapidly evolving field of science and, although many applications have been speculated, many difficulties have to be overcome before they can be commercially applied. Nevertheless, as the molecular and physiological bases of animal functions are

unravelled, new approaches to advance animal performance can be explored and developed.

Examples of these up-and-coming technologies and their potential application in commercial practice are presented in Table 3. All of these will have an enormous impact on the way the pigs are nurtured and managed at all stages of production and on the efficiency of pig production *per se*. It is therefore important that the pig industry keeps abreast of these new developments and applies them at the earliest opportunity. By responding to the challenge in this positive way it can compete successfully with the other meat industries.

Table 3. Examples of existing and emerging technologies and their potential application in practice.

Technology	Application
DNA technology	pST, β-agonists to enhance performance
Fermentation technology	Enzymes, peptides, sugars to improve digestive efficiency
Dietetic developments	Probiotics, yeasts, acids to enhance health of gut
BLUP and development of molecular probes	Estimation of genetic progress and selection for superior traits
Gene-mapping/genetic manipulation	Transgenic pigs, gene transfer
Marker assisted selection	Candidate genes to improve litter size (ESR), meat quality, health status etc.
Sexing of semen	Selection of all male/female litters
Immune modulation	Enhancing the endocrine and immune status; better performance, disease resistance and health status

Who should pay and who should do the work?

In the past, research has been broadly classified and organised into applied, strategic and basic studies and it is likely that it will continue in this format in the future. The funding for such programs was the responsibility of central or provincial governments and/or levy-based support systems. However, in the past 10 years this has changed in several European countries, partly as a result of an overall reduction in funding from public funds, as well as a change in the priorities and philosophy for research and development. A larger proportion of the reduced funding is being directed to more strategic and basic studies. The public funding of applied or production research was dramatically reduced, with the onus on self-funding from industry which was seen as the end-user and beneficiary of such studies. However, in some countries, industry was slow to respond and did so only after the facilities and staffs involved in near-market research projects were drastically reduced. As a consequence, many large private organisations and companies now carry out their own production-type research programs. They may conduct these at their own research facility or, increasingly, at a university or research institute because of the considerable costs in establishing and maintaining their own in-house facilities.

This trend to 'privatisation' and self-financing of research is set to continue as the number of producers decrease and the size of individual herds increase. In addition, many companies now operate on a global basis and are sufficiently large to establish their own research programmes. Most production-type or near-market research will therefore become privately funded and carried out on commercial farms where the results can be applied immediately to the exact conditions under which they have been generated.

Campbell (1995) has drawn similar conclusions. In addition, the results are not published and therefore not available to competitors or the industry at large. However, the costs of such commitments will have to be included in the price of the end-product.

There is therefore a need to initiate and develop strategic and near-market research programmes that ensure the economic competitiveness of the industry as a whole. The most effective way to achieve this is to develop collaborative programs of work between industry and government that are pre-competitive, with agreed aims and objectives. Such an approach involves a stakeholder or link-type commitment and should involve all sectors of the industry, including the end-user (British Society of Animal Science, 1999). All sectors of the industry would be partners and equally involved to ensure that the research programmes are appropriate to the needs of the industry and to allow effective technology transfer and application of the results with minimal lag time. Such an arrangement would not only include partnerships between government and producers through the payment of levy fees, as is the case with the Pig Research Development Corporation in Australia, but partnerships among producer groups, commercial companies and government. This strategy will also allow a more effective understanding and communication between scientists and industry, so vital to the success and competitiveness of the industry. The funding for such projects would come from both government and industry, and for the latter could come from a variety of sources, such as individual companies, producer organisations, levy payments and other sponsoring agencies. The proportion of funding contributed by each will vary depending upon the nature of the project and a committee drawn from academia, industry, producer and consumer communities would be responsible for monitoring progress and ensuring that target objectives are met. Both commercial and research facilities could be used and participating companies might be given some lead time for commercial application of any results before widespread publication.

Research is also needed to provide information on the basic biology of the pig, that is, basic or fundamental research. This provides a basis for further advances in animal growth and reproduction, as well as the health and welfare of the animal. Such studies usually involve physiological, endocrine, immunological and molecular techniques that are outside the scope of production and strategic research and are designed to provide a greater understanding of the complex interactions of the biological systems in a range of environmental and ecological systems. It is usually at a cellular level and considerably removed from 'on-farm' application, since it provides research that will give rise to new products or technology in the medium to long-term (5-20 years). Such work needs to be funded from the public purse and carried out at universities and research establishments.

Greater demands are being placed on pig production because of new legislation. Changes in the way animals have to be kept, new codes of practice for welfare, hygiene and environmental control, new procedures to control the safety of pig feed and pig meat products are currently being considered. However, these need to be thoroughly researched, so that the most effective and easily manageable procedures can be adopted and developed. Since these are required by law, the cost of these developments must be met from public funds.

A balance is therefore required of near-market, strategic and basic research with short, medium and long-term objectives. To achieve the various research objectives and to have successful research programs, there must be adequate resources and appropriately staffed facilities. Strategic and basic research cannot be 'turned off and on' and sufficient resources must be maintained at both Universities and Research Institutes to ensure that the immediate and long-term needs of the industry are met. They are also needed to meet acute needs in times of crises, such as have been experienced with Salmonella in the 1980's and Bovine Spongiform Encephalitis (BSE) in the 1990's, as well as in the training of young scientists, so vital for the future success of the industry. In addition, the requirement for the registration and authorization of newly developed products in many countries stipulate that the work be carried out at independent centres. Although industry may contribute, overall responsibility for the maintenance of such facilities lies in the public domain.

The success of research and development is dependent on the transfer of knowledge and information among scientists, that is scientific communication, and the transfer of technology to practitioners, that is down to production level. This has not always been achieved and although improving, the time taken for the application of new knowledge emanating from research papers into practice is often too long. Mechanisms and opportunities must therefore be put in place that allow a greater interaction and more effective communication among scientists, advisors and consultants for the good of the pig industry, as is suggested in the stakeholder type approach.

Key to a successful pig industry

The key to a successful pig industry therefore is to adopt new methods, technologies and information from research. This will allow it to become more competitive, not only against other meats, but also against imports from other countries. To achieve this, the costs of production must be minimal, the products offered must be perceived as good value in terms of price, quality and safety and it must be possible to differentiate between these products.

In terms of cost of production, research and development can play a major role in developing strategies that allow the genetic potential for both growth and reproduction to be achieved on farm. Similarly, since feed is the largest cost involved in pig production, accounting for 65-70% of the total costs, this is where the most savings can be made. In this respect, the feed provided in the growing and finishing period (20-100 kg body weight) is of most concern, as it represents two-thirds of the total costs of feeding the pig, including that required for the sow and boar. Indeed, the aim should be to produce a pig to 100 kg body weight with an overall herd feed efficiency of less than 2.75 kg of feed per 1 kg of growth (Table 4). Information is available on the nutrient requirements of the animal at each stage of growth and reproduction. It is important that advice is given and taken, and that feeding strategies are developed and customized to meet the needs on each farm, to ensure optimum utilisation of feed, minimal nutrient loss to the environment and lowest feed costs. Obviously, the health status of the animals has a major effect on overall efficiency, as discussed earlier, and needs to be taken into account when developing the appropriate strategy.

Table 4. Suggested nutrient and feed requirements at the different stages of production.

	Gestation	Lactation	Starter	Weaner-1	Weaner-2	Grower	Finisher
			7-10 kg	10-17 kg	17-25 kg	25-60 kg	60-100 kg
DE (MJ/kg)	13.0	14.0	16.5	15.5	15.0	14.0	13.4
Lysine (g/kg)	6.0	10.0	16.5	15.3	14.0	12.0	10.5
Lysine (g/MJ DE)	0.46	0.71	1.00	0.98	0.92	0.86	0.78
Target feed (kg/pig)	35	18	3	9	15	70	125

The total feed requirement is 275 kg/pig; giving an overall feed conversion efficiency of 2.75:1.

In the consumer-led market, the industry must be adaptable enough to provide for the different needs of all its customers. Greater public awareness of systems of production, of their ecological consequences, as well as an ever more discerning and demanding customer means that a wider range of products needs to be considered. Creating consumer choice is the order of the day, but just as important is consumer information; from products available to sources of origin and methods of production, from inherent health benefits of pork to possible ways of preparing it to maintain and enhance its flavour, taste and texture. Only then can the consumer make an informed choice. This is not just an exercise in advertising a product to make a profit for the

producer, but offering a product and a service that benefits the public at large, improving general human health and well-being.

Pig meat must be identifiable as a healthy, safe, nutritious and good value food. In this respect, quality assurance schemes or codes of practice are critical; they guarantee that specific standards are met and maintained at farm level and throughout the production line. These schemes are a detailed and timely response to the demands of the consumer and will secure his confidence by promoting high standards of quality assurance, traceability and animal welfare.

Conclusions

Progress in pig science is dependent upon basic, strategic and applied research. Basic research must provide information on the physiological and molecular principles fundamental to growth, development and reproduction, with the long-term objective of developing technologies for commercial application in 5-10 years time. Strategic research, on the other hand, tests the importance of the results of basic research and develops their potential to improve some aspect of pig production. Strategic research also provides information that allows interpretation, on a biological level, of differences in response among animals. Applied research takes the solutions generated from strategic research and applies them in commercial practice.

In the future it is likely that applied or near-market research (response research) will be minimally funded from the public purse, but will increasingly become the charge of the industry *per se*, since it is the immediate end-user of the results and has most to benefit. However, in terms of strategic research, an interesting innovation will be the development of a stakeholder type approach that will involve both public and private funding and will include all sectors of the industry, including the retailer and consumer. Public funding may become further limited and will be increasingly directed towards funding innovative and basic research to deepen our understanding of the basic biology of the pig and for research relating to proposed or newly-implemented legislation.

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A REVIEW - VARIATION IN PIG PRODUCTION AND PERFORMANCE

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Abstract

The extent, causes, and management implications of the variation commonly experienced in pig production, particularly with regard to all-in/all-out management and low-cost deep-litter housing systems, are examined. Topics reviewed include: genotype and crossing system, disease, hygiene, the effect of feed additives on variation in growth, nutritional factors, weight and age of pigs at weaning, management of lightweight pigs pre- and post-weaning, group size, stocking rate, sorting and mixing of pigs, pig behaviour, and the effect of season. Variation in the number of pigs weaned, and the disparity in number of males and female pigs produced per week and the problems that these factors pose, are considered. The economic impact of within-group variation is estimated using the AUSPIG simulation model. Management strategies to reduce variation in performance and marketing strategies to minimise the effect of variation on profit are discussed.

Introduction

The Australian pig industry is going through a period of rapid change as it attempts to position itself in a global market place. A consistent supply of pork in the quantity and quality required by processors, and ultimately the consumer, is crucial to the expansion of both domestic and export markets and to the long-term viability of the industry.

Minimising and managing the variation that occurs at all stages of the production chain is perhaps one of the biggest challenges currently facing the Australian pig industry as it restructures and adopts new technologies and housing systems. Batch farrowing, segregated early weaning, multi-site production, all-in/all-out management (AIAO), split-sex, and phase feeding are rapidly gaining acceptance in both conventional and alternative housing systems, and have enabled the use of far larger group sizes than previously considered acceptable. Groups of 100 to 1000 weaners or grower/finishers are not uncommon, but present new challenges in managing variation in growth and carcass quality. The adoption of new technologies has also reduced health costs and increased whole of life growth rates to 700-750 g/d compared with 600-650 g/d for above-average traditional continuous flow operations. However, some of the financial benefits derived from improved performance may be offset by decreased revenue because of sub-optimal carcass values and reduced facility throughput caused by variation in weight at the end of the grow-out period. The impact of variation is most apparent at the end of the grow-out period when critical marketing decisions are made, but the sources of variation originate much earlier in the production chain.

Reported values for variation in the final weight of market pigs

The advent of specialised grow-out facilities has stimulated interest in performance monitoring of the grower herd, but there are surprisingly few reports of the variation in growth rate that occurs in these facilities. Cook (1991) discussed the variation in growth rate and carcass quality experienced on a group of farms in Queensland. However, these data are derived from relatively small herds managed on a continuous flow basis. More recently in North America, the distributions of final weights in AIAO facilities have been studied (Patrick *et al.*, 1993; Deen, 1995; Roberts and Deen, 1995; Deen 1997). In a study of grower/finisher records obtained from a commercial records bureau, Rademacher *et al.* (1997) reported a coefficient of variation (CV) of 9% for 307 groups of pigs with an

average final weight of 113 kg. Within-group CVs were not reported. As a rule of thumb, based on collective experience in research and commercial facilities in the USA, Brumm (pers. comm.) suggested a CV of 15 to 18% for groups of pigs entering an AIAO facility at 20 to 25 kg live weight, and 10% when some or all of a group are first marketed, assuming pigs enter the group within one week of age.

Inherent variation

Some variation in final weights of groups of pigs is unavoidable because of the age range of pigs entering a facility and the growth rate differences between males and females as modelled in Table 1. The simple spread sheet model used assumes an average growth rate of 650 g/d and that entire males grow 50 g/d (7.7%) faster than females, in the absence of other sources of variation. A seven-day age range produces a difference of over 12 kg between the weight of the youngest females and the oldest males within a group of pigs with an average final weight of 105 kg at 23 weeks of age. This equates to about 60% of one standard deviation (SD), assuming a normal distribution of weights.

Table 1. How age, growth rate (GR) and sex affect the final weight of female and male pigs.

	Female (F)			M+F	Male (M)		
	Youngest	Average	Oldest	Average	Youngest	Average	Oldest
Age (d)	157.5	161.0	164.5	161.0	157.5	161.0	164.5
GR (g/d)	625	625	625	650	675	675	675
Weight (kg)	98.4	100.6	102.8	105.0	106.3	108.6	111.0

The effect of even small (7 d) variations in age and differences in male and female growth rates should be taken into account when developing throughput and marketing plans. Additional accommodation may be required to house females for a longer period to achieve market weights similar to males. Alternatively females may be marketed at a lower live weight, a practice often associated with more acceptable carcass backfat thickness.

Other sources of variation

Frey (1998) listed the following factors that are reported to increase variation in grower/finisher pigs:

- Genotype and crossing system
- Disease
- Management system
- Access to feed and water
- Feed delivery
- Separation of feed ingredients
- Weight at entry
- Group size
- Space allocation
- Sorting and co-mingling
- Dominant or submissive behaviour
- Stockmanship
- Season

Genotype and crossing system

Differences in growth and carcass traits of individual pigs contribute to variation observed within groups of pigs produced for market. The phenotypic performance of an animal is determined by its genotype and environmental factors. Some indication of the extent of within-breed variation in phenotypic performance can be gleaned from published performance test data. For example, Hall *et al.* (1999) reported CVs for backfat of 21%, food conversion ratio 11%, average daily gain 13%, and daily feed intake

13%, calculated from the records of 1,832 pigs of a Large White sire line selected for lean tissue growth. The pigs were tested in groups of twelve and fed *ad libitum* from a single-space feeder with electronic food intake recording equipment. Variation of at least this order of magnitude may be expected for these production traits in other genotypes. The distribution of estimated breeding values and genetic trends for daily gain and backfat of Large White, Landrace and Duroc boars performance tested at different locations in Australia are reported in National Pig Improvement Program Reports (Macbeth, 1999). Differences of 2 to 3 mm in backfat and 50 to 60 g/d in daily gain can occur between the top and bottom tenth percentiles of all boars tested for each breed.

Variation between and within groups of pigs is also influenced by the crossing system adopted (Long, pers. comm., 1999). Maintaining multiple breeds and lines of purebred sows within herds either for rotational breeding programs or for in-herd production of terminal parents creates variation in market pigs. In traditional two- or three-breed rotational breeding programs, breed composition alters with each rotation of the breeding cycle, which may increase variation in performance and carcass quality if there are wide differences between breeds for these traits. Maintaining populations of purebred animals within commercial herds to produce terminal parents also can be a potential source of variation in pigs marketed. Not all pigs produced from matings to raise terminal parents are retained for breeding purposes. A proportion will be marketed as finisher pigs that may have distinctly different carcass characteristics to the majority of pigs produced requiring separate market outlets in some instances.

Theoretically, the potential for variation in performance and carcass characteristics is minimised from a genetic standpoint when market pigs are derived from terminal parents in a single specific cross. There is a growing trend towards buying in all replacement females and males, although cost, limited availability of breeding stock, and difficulty in obtaining replacements from a single source may limit this practice to small and medium size units.

The use of artificial insemination may also reduce variation by distributing semen from single boars over a greater number of females, and by the more rapid introduction of superior genetic material into herds. However, some degree of genetic variation within groups of grower/finisher pigs is inevitable, regardless of genotype and crossing system selected.

Disease and the use of additives

The effect of disease on variation is not well documented. There are numerous reports of the impact of disease on average performance parameters but its impact on variability is less frequently reported. Patrick *et al.* (1993), in a controlled study, found that pigs exhibiting clinical disease and then treated, took an additional 15.3 days to reach slaughter weight. Skirrow (1993) reported the mean and SD for growth rate of groups of pneumonia-free and pneumonia-affected pigs in two Western Australian studies, making it possible to calculate the CV and the number of days to reach 100 kg. Using data from the first study, the CV for growth rate and the number of days required to reach 100 kg were calculated to be 7.5 and 5.9%, and 187 and 175 days, respectively, for groups with and without pneumonia. Similarly, in the second study the CV for growth rate and the number of days required to reach 100 kg were calculated to be 9.0 and 5.0%, and 193 and 178 days, respectively, for groups with and without pneumonia. The variation in growth in the pneumonia-affected groups was 27% and 80% greater than that in the non-infected groups in studies 1 and 2, respectively.

Deen *et al.* (1998), in a trial using 3,411 pigs housed in five grow/finish sheds, found that tylosin not only increased the average weight of treated pigs compared to controls, but also influenced the distribution of weights. Tylosin increased the growth of the smaller pigs more than that of the larger pigs and reduced the variation in final weight. The lightest 20% of the pigs fed tylosin gained 4.1 kg ($P \leq 0.1$) more than the lightest 20% of the controls. However, there was no significant difference in the weights of the heaviest 20% of pigs on each treatment. Similarly, Tillman (1997) found that the response to bacitracin methylene disalicylate was greater in lightweight pigs, and decreased the CV in final weight from 13.1% for the controls to 11.1 % for treated pigs. Frey (1998) reported

no significant difference in treatment means for pigs fed a growth promoter compared to unmedicated controls. However, the SD for growth rate was reduced by 15 g/day and for final weight by 1.5 kg. Although there was a trend to increased backfat in the medicated group, the mortality rate was lower and there were considerably fewer tail-end pigs.

Management system

In an experiment where pigs were removed from a low-health-status farm with a high level of respiratory disease to a more hygienic environment operated on an AIAO basis, Straw (1991) reported less severe respiratory disease and faster growth than in the control group maintained on the low-health-status farm. The mean growth rate and CV were 639 g/d and 20.5%, and 765 g/d and 16.5%, for the pigs reared on the low health status farm and in the improved environment, respectively. Patrick *et al.* (1993) reported decreased variation in growth rate of pigs under AIAO management compared to continuous flow management. The CV for the number of days to reach 105 kg was 6.55% under AIAO management compared to 8.62% for continuous flow management. In both of these experiments, improvements in pig performance appeared to be a result of improved respiratory health brought about by a combination of AIAO management and the higher standard of hygiene practised. All-in/all-out productions systems without thorough cleaning of facilities between batches of pigs are unlikely to improve significantly the performance or reduce the prevalence and severity of respiratory disease compared with continuous flow systems (Cargill and Banhazi, 1998), and therefore may do little to reduce variation attributable to this cause.

Nutritional factors

Feed quality

The choice of feedstuffs, feed preparation methods, and feeding management strategies have a major influence on efficiency of nutrient extraction and utilisation by pigs (Edwards, 1999). Failure of the diet supplied to meet nutrient specifications; abrupt changes in ingredients; inappropriate particle size; inadequate mixing; and deterioration of feed in storage, will contribute to variation in pig performance and carcass quality. Separation of feed during conveying may also cause differences in the quality of feed offered that may result in production differences within a building. On farms, there is always the risk of incorrect feed being supplied which, if undetected, may adversely affect performance. Effective quality control measures at all stages of the feed supply chain are essential to minimise variation caused by inconsistent feed quality.

Feeding management

The benefits of phase and split-sex feeding are widely accepted within the pig industry as a means of reducing the cost of production (Edwards, 1999) and also the nitrogen and phosphorus content of effluent (Pluske *et al.*, 1997). Both phase and split-sex feeding require dietary specifications to meet closely the nutrient requirements of target populations of pigs. This is difficult to achieve if wide weight and age variation exists within populations. Variation may increase in proportions of pigs that have dietary requirements substantially different to the average for the population. This dilemma can be resolved by raising dietary specifications by one standard deviation above the mean requirements for a population, in which case about 83% of a population will receive adequate diets compared to only 50% if specifications for population means are used (Edwards, 1999). However, the cost effectiveness of this practice requires determination case by case using a simulation model.

Fluctuation in daily feed intake

Daily fluctuations in feed intake of individual animals decreases protein deposition and increases fat deposition, creating an increasing disparity in live weights which renders setting dietary change points more difficult, and resulting in greater variation in carcass weight and backfat thickness (Edwards, 1999). Fluctuations in daily feed intake may occur for many reasons. Individual pigs may exhibit a pattern of feed restriction followed by engorgement. Disease, trauma, social factors, temperature, changes in feed quality, and interruptions to the feed and water supply may also affect appetite and feed intake.

Weight at entry

Variation in weight at entry is generally considered to be a significant source of final-weight variation in AIAO production systems. Patrick *et al.* (1993) found a strong relationship between the initial weight on entry to a grow-out facility and the number of days to reach 105 kg. For each 0.454 kg heavier a pig was on entry to the grower/finisher facility, 0.76 fewer days were needed to reach 105 kg.

The proportion of lightweight pigs entering either nurseries or wean-to-sale facilities is also of concern to producers. It is necessary to distinguish between excessive variation in entry weight and the problem of managing lightweight pigs, although in practice the two are often associated. It is possible to have reasonable uniformity but a low average entry weight, which may result in a higher proportion of lightweight pigs in a group (Tokach *et al.*, 1998).

Variation in weight-for-age at weaning is inevitable if farrowing facilities are operated on an AIAO basis. Tokach *et al.* (1998) examined several large groups of pigs and found the SD of individual pig weight at weaning at 21 days to be consistently close to 0.9 kg with approximately normal distributions for pigs weaned within a 7 day age range. Although techniques such as split weaning and split nursing have been shown to reduce weaning weight variation (Donovan and Dritz, 1997), the most effective way of reducing the number of lightweight pigs at weaning is to inrease the average weaning weight (Tokach *et al.* 1998). Increasing the average weaning weight from 4.5 kg to 5.5 kg would theoretically decrease the number of pigs weighing less than 4.5 kg from 50 to less than 16%. However, this strategy does not necessarily reduce variation in gain during the grow-out period or final-weight variation.

Several studies have demonstrated the effect of weaning weight on days to market. Mahan and Lepine (1991) showed that pigs weighing from 7.3 kg to 8.6 kg at weaning (25.3 days) reached the sale weight of 105 kg about 15 days sooner than pigs that weighed from 4.1 kg to 5.0 kg at weaning at 23.8 days, regardless of the lighter group being fed a higher quality starter diet than that fed to the heavier treatment. The heavier pigs grew almost 10% faster from weaning to 105 kg than the lighter group. They concluded that the heavier pigs at weaning maintained their weaning weight advantage through to market weight. Rademacher *et al.* (1997), after examining post-weaning performance in weaner facilities, noted that growth rate, feed intake and mortality rates were poorer for groups of pigs entering weaner facilities at 3.5-4.0 kg than for those entering at 4.5-6.5 kg average weight, and recommended that pigs should weigh at least 4.5 kg before entering a weaner facility.

Minimum weaning weight and maximum weaning age targets are often mutually exclusive for a proportion of pigs because of the variation in birth date, birth weight, and pre-weaning growth rate. Although back-suckling and cross-fostering of piglets to delay weaning of lightweight pigs may reduce variation in weight at weaning, these techniques conflict with fixed weaning ages imposed for disease control purposes. There is also considerable doubt about the benefit of reducing variation in weaning weight by cross-fostering late in the nursing period. Straw *et al.* (1998) found that continuous cross-fostering throughout the nursing period reduced the variation in weight at weaning by 41% but depressed pre-weaning growth rate by 20% compared with restricting cross-fostering to the first two days of life. Other management options to meet these seemingly opposing objectives include separately rearing pigs that fail to meet the required minimum weaning weight, either off-site or in parallel to the main weaner facility, or by relaxing the weaning

weight target if the number of lightweight pigs is small (Sornsen, 1998). Some producers elect to establish a communal off-site weaner facility to cater for lightweight weaners. Such a facility can also be used to house weaners that are surplus to the capacity of the receiving facility in the event of production exceeding target levels.

Group size, space allowance and feeding space.

The effect of group size, space allowance and feeding space on pig performance were discussed by Chapple (1993) and, more recently, by Nielsen *et al.* (1995) and Gonyou and Stricklin (1998). Although there is a lack of agreement among studies on the effect of group size on performance, it seems that increasing group size alters the behaviour, social interaction, and activity of pigs. However, most studies have been restricted to traditional penning arrangements and to group sizes considerably smaller than those now being used in both conventional and alternative housing systems. It is possible that in the presence of adequate feeding and drinking arrangements, any adverse effects of increasing group size on performance may plateau at some threshold yet to be determined. There are few reports in the literature of the effect of very large group sizes on pig performance, and an urgent need exists for much of the early work to be repeated on considerably larger groups.

The move to larger group sizes has been driven in part by the lower capital cost of weaner and grow-out facilities utilising large pens compared to conventional penning arrangements. Bent and Coleman (1997) stated that passageways and partitioning account for 23% of the internal floor area of conventional buildings. Peet (1997) estimated the number of pigs that can be housed in any given building area can be increased by 30% for nurseries and 20% for grower/finisher facilities by the removal of the majority of passage space and pen partitioning. Peet (1997) also suggested that because space is used more effectively in properly designed large pens, space allowances at the lower end of accepted standards can be used, further reducing the capital cost compared with conventional layouts.

Large groups of weaners

English *et al.* (1994) investigated the performance of a group of 200 piglets weaned at 21-28 d at an average weight of 7.4 kg (range 4.8 kg to 10.8 kg) and housed in a straw-based rearing system at a stocking rate of 0.65 m^2 per pig. Growth rate was analysed according to weight category at weaning. Relative to the lightest weight category at weaning, the growth rate of the medium and heaviest categories was 5 and 14% higher, respectively. In a follow-up study, pigs at weaning were penned separately into light (6.6 ± 0.05 kg), medium (7.5 ± 0.04 kg) and heavy (8.5 ± 0.05 kg) groups of 40 per pen. The growth rate increase relative to the light group was 4.1 and 11.7% in the medium and heavy groups, respectively. All weight categories grew around 11 to 13% faster in the second study than in the first, perhaps because of the smaller group size. However, the growth of the light group in both studies was considered satisfactory. It was concluded that good performance was possible for large groups of weaners on straw-bedding provided that adequate feeding, watering and floor space was available. While light pigs had a slightly poorer growth rate post-weaning, this appeared to be more a function of their weaning weight rather than any disadvantage in terms of digestive or competitive capability.

Large groups of grower/finishers

Penny *et al.* (1997) examined the behaviour of low (L) and high (H) performing pigs identified within groups of 66, 124 and 208 pigs housed on deep-bedded straw. The growth rate of L and H pigs within each group remained significantly different during the behavioural recording period but the growth rate between the different sized groups was not significantly different. Although feeding observations for the L and H pigs were not different, L pigs were more active and spent more time performing behaviours indicative of foraging motivation and possible feeding frustration.

Data derived from a Western Australian study to evaluate the use of straw-based shelters for grower/finisher pigs (Payne, 1997) have been re-analysed to determine the effect of group size on the variability in weight when the first pigs in a group were removed for marketing (first draw) and in the final weight of groups of pigs. The original study was conducted over three years, using two shelters each of 200 m^2 that were initially partitioned longways into two pens. Six batches (n=1950) of single-sex pigs were raised in each shelter. The partition was removed from one shelter for batch 3 onwards and from the other shelter for batch 6. Each shelter was stocked with 150 pigs for batches 1 - 4, with 175 pigs for batch 5, and 200 pigs for batch 6. Thus group sizes of 75, 100, 150, 175 and 200 pigs were achieved during the study. Stocking rate, which ranged from 1.0 m^2 to 1.3 m^2 per pig, was not standardised but the number of pigs per feeding space and per drinker was similar for all groups.

Most pigs were marketed as baconers to a single processor at weights which maximised returns according to the prevailing contract price schedule. Carcasses with P2 backfat measurements of less than 14 mm at hot standard carcass weights from 66.0 kg to 88.0 kg attracted the highest price. Some lightweight pigs that were judged incapable of reaching bacon weight by 24 weeks were sold as porkers at 45 kg to 65 kg live weight at the time of first-draw.

The pigs were obtained from a single 500-sow breeding unit which meant that litters from two weeks production were required to make up the required batch sizes. There was a significant (P=0.042) linear relationship between the within-group SD of entry weight and group size.

A model was constructed to account for variability in entry weight to examine the effect of group size on the within-group variability of first-draw and market weights, using group SD as the measure of variability. A multiple regression analysis between within-group SD for market weights and group size, sex, and within-group SD for entry weights accounted for only 2.5% of the total variance, with all the independent variables being non-significant (P=0.966, P=0.556, P=0.824, respectively). When the same independent variables were used with first-draw weight as the dependent variable, group size and all the two-way interactions were also non-significant (P=0.642 for group size). The remaining independent variables accounted for 69.7% of the total variance with effects of both sex and within-group SD of entry weights significant (P=0.006 and P<0.001, respectively). The within-group SD of first-draw weights for males was significantly higher for males than for females (9.2 vs 7.8 kg).

Finally, a multiple regression analysis with live weight at first-draw as the dependent variable, and group size, sex, entry weight, age, and age at first draw as independent variables, together with all the two-way interactions, accounted for 57.4% of the total variance with all the independent variables significant (P<0.001). When separate multiple regressions were fitted for males and females, and interactions between the independent variables used above, but excluding sex, were added to the model, 61.5% of the variance between live weight at first-draw for males and 59.2% for females was accounted for. The main effect of group size was significant (P<0.001) for both males and females, with the average weight for males at first draw decreasing by 0.41 kg for every increase of 10 in group size above 75 pigs and increasing by 0.53 kg for every increase of 10 in group size for the females. Within-group SD of entry weight also significantly affected SD at first draw.

The different effect of group size on weight at first-draw for males and females reflected the greater variation present in the males. The wider spread of weights necessitated marketing a proportion of light and heavy males earlier in the grow-out period compared to females, when the average live weights of the groups were lower, to minimise penalties imposed for under- and over-weight pigs.

Sorting and mixing of pigs

Studies have shown that sorting pigs by weight when forming groups does not necessarily reduce final weight variation, and that sorting by sex in the case of castrates and females does not appear to have an adverse effect on performance (Tindsley and Lean, 1984; Gonyou *et al.*, 1986, Gonyou, 1998; Mikesell and Kephart, 1999). However

group sizes and the range of weights used by these authors were small and it cannot be assumed that similar results would be achieved in much larger groups. Similarly, studies on the effect of regrouping pigs on performance and behaviour have used small groups in conventional pens (Sherritt *et al.*, 1974; Stookey and Gonyou, 1994). Anecdotal evidence suggests that the deleterious effect of mixing large groups of pigs in deep litter systems is considerably less than might be expected, but it remains for this casual observation to be scientifically substantiated.

Behaviour and social order

Recent research indicates that individual pigs differ in their ability to cope with a variety of stressors and suggests the existence of active or passive coping styles, each associated with distinctive behavioural and physiological responses (Hessing *et al.*, 1994; Giles and Kilgour, 1999). It appears that coping style is continuously distributed between the extremes of active or passive behavioural responses to stressors and may have a genetic component. Hessing *et al.* (1994) found that pigs classified as resistant (active coping style) or non-resistant (passive coping style) in response to a 'backtest' (restraint in a supine position for sixty seconds) conducted in the first and second weeks after birth performed similarly from nine weeks of age until slaughter when grouped according to coping style. However, mixed groups of resistant and non-resistant pigs grew faster and were less variable (801 g/d, CV = 7.1%) than resistant groups (761 g/g, CV = 11.8%) and non-resistant groups (773 g/d, CV = 10.5%). Carcass parameters and health status were also superior for the mixed pens. Giles and Kilgour (1999)compared the performance of grower pigs classified according to vocalisation in response to nose-roping. Pigs with low vocalisation scores (passive coping style) grew faster from 57 to 87 kg live weight than those with high vocalisation pigs (active coping style) whether housed in single pens (1,196 vs 1,137 g/d) or in groups of six pigs per pen (1,057 vs 990 g/d).

Individual behavioural characteristics appear to influence the social stability of groups (Hessing *et al.*, 1994). It has been suggested that a certain amount of variation is necessary for the development and maintenance of a social order within a group and, that in the absence of variation in weight when the group is formed, it will develop over time (Tindsley and Lean, 1984; Gonyou, 1998). Tindsley and Lean (1984) found that the CVs for groups of eight pigs selected on the basis of similarity of age and weight (18 ± 0.5 kg) increased from 2.0 to 13.4% over a 10-week trial period during, while those selected on the basis of similarity of age but mixed weights (18 ± 4.5 kg) remained relatively stable at 17.1 and 17.8 %. Weight range at allocation had no effect on final weight range and pen occupancy time, but even-weight groups were 2.8 kg heavier ($P<0.01$) at the end of the trial because of superior weight gain in the first week after mixing, attributed to a shorter settling period.

However, little is known about social order and behaviour of pigs in very large groups in deep-litter systems. The social consequences for pigs reared in these systems compared to conventional systems have not yet been quantified.

Seasonal variation

Superimposed on within-group variation is the influence of season. There is a marked seasonal pattern in both carcass weight and backfat thickness throughout Australia, although all piggeries are not equally effected. Pigs slaughtered in late summer and early autumn tend to be lighter and leaner than those slaughtered in winter and early spring (Trezona *et al.*, 1999). However, examination of individual carcass data from a large commercial piggery showed that although carcass weight and carcass quality follow the same seasonal pattern (Figure 1), regression analysis showed that only about 15% of the variation in P2 backfat was explained by carcass weight. On the same farm, the CV for carcass weight was 11.3 % and that for P2 backfat was 21.7 %.

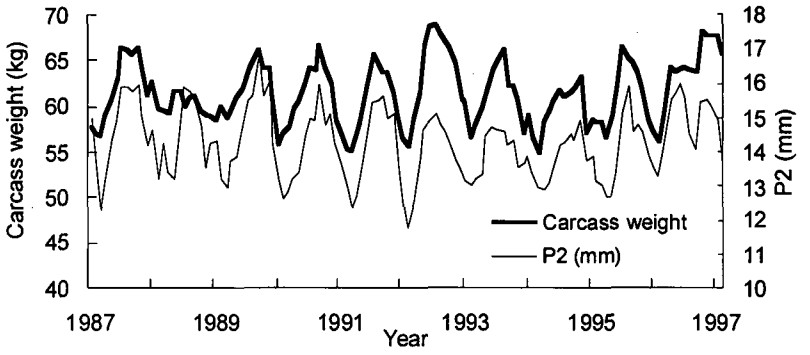

Figure 1. *Average monthly carcass weights and P2 measurements from a Western Australian piggery.*

Additional sources of variation

Variation in the number of weaners produced per week

Although most pig units are designed to produce a set number of pigs per week, in practice there is often considerable divergence between target production and that achieved. An indication of the variation that exists within the Australian pig industry is presented in Table 2 which also contains some important production parameters recorded from a sample of six herds in Western Australia, average size 360 sows, and representing over 2100 sows in total (Frey, unpublished).

Table 2. Coefficients of variation (%) for production parameters calculated from weekly performance data recorded on six Western Australian pig units for 52 weeks.

Parameter	Herd No.					
	1	2	3	4	5	6
Total number of services	22	13	14	18	27	12
Number of sows farrowed	27	19	23	34	31	18
Total pigs born alive	30	19	23	34	32	18
Number of litters weaned	25	16	15	34	25	9
Total number of pigs weaned	28	16	17	28	26	11
Pigs weaned per litter	8	6	7	11	13	6
Pre-weaning mortality	54	23	83	40	39	27
Average age at weaning	8	9	11	11	14	3

The total number of pigs weaned and the average age at weaning potentially have the greatest impact on variation in post-weaning performance. Both affect facility throughput and stocking rate. Under-production reduces potential returns, but in most cases will not adversely affect pig performance. However, over-production may lead to increased stocking rates and lighter and more variable sale weights. Producers may choose to over-mate to be certain of meeting farrowing targets, but this practice needs to be properly managed to avoid over-loading facilities. Alternatively producers may set a target for weaner production that ensures the required number of pigs for the grow-out facility is always met, and diverting the surplus to capacity to another outlet.

Disparity in numbers of males and females produced

It is generally assumed for planning purposes that equal numbers of male and female pigs are produced on a weekly basis. This may indeed be valid for large herds, but analysis of data from a 650-sow herd in Western Australia demonstrates that this is not always the case for small- and medium-size herds. Weekly numbers of males and females born alive over a six-month period are shown in Figure 2. On average males outnumbered females by 6% but, more importantly, there was a greater than 10% disparity between sexes in 40% of the weeks, and an extreme case when it was 24%. This may create a dilemma if accommodation has been designed for specific numbers of pigs of each sex, creating the potential for under- or over-stocking for 40% of the time unless some mixed-sex pens are formed.

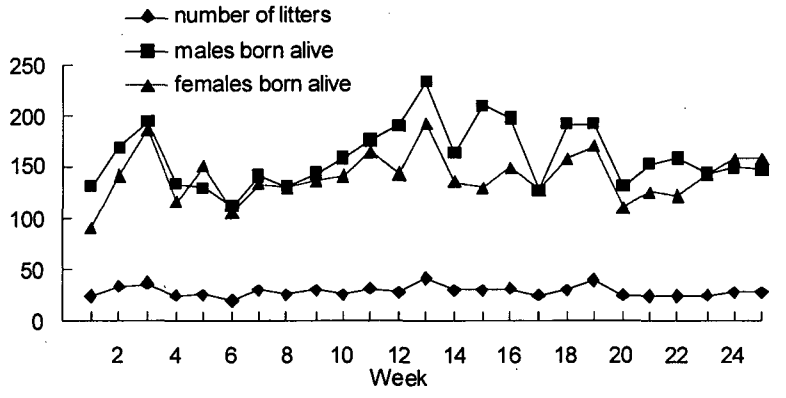

Figure 2. *Weekly production of male and female pigs from a 650-sow herd in Western Australia.*

Unexplained variation

A large proportion of variation observed in controlled experiments and in the field cannot be explained by the factors discussed above. Patrick *et al.* (1993) found that 71% of the variability observed in their controlled study was not accounted for by initial weight, sex, continous-flow or AIAO management, lung lesion score or clinical signs of disease. Similarly, Payne (unpublished) showed that about 43% of the variation in weight at first draw was not accounted for by group size, age and weight at entry. It is likely that genetic variation within and between groups, and individual behavioural characteristics of pigs were major sources of the outstanding variability in both studies. In the field, management errors, equipment failures and stockmanship differences are also probable causes of variation.

Economic impact of variation

Most producers are well aware that variation in pig production and performance has a major impact on the profitability of their enterprises. However, the cost of performance variation is difficult to quantify, as are the benefits and costs of control measures. Cook *et al.* (1993) applied the concepts of Total Quality Management Statistical Process Control methodology in the development of the PIGPULSE computer software for the analysis of throughput and efficiency traits for breeding and finishing herds. The software transforms data collected on farm and at abattoirs into useful managerial information by using statistical techniques to distinguish between normal and abnormal trait variation. Other methodologies are being developed to evaluate cost of

variability in sow output (Deen and Davies, 1998) and for optimising profit and pig flow in AIAO grow-out facilities (Dritz and Tokach, 1998).

The AUSPIG simulation model was used to simulate the effect of within-group variation on pig performance and profitability. Performance data for pigs considered to represent low, medium and high levels of performance in a commercial piggery were incorporated into the model (Table 3). The simulation was conducted on the basis that pigs were transferred each week at 56 days of age into two single sex pens designed to hold 120 finisher pigs (100 kg LW), and subsequently sold at 166 days of age with a dressing percentage of 76%. If 100% of pigs had a level of performance classified as medium, then profitability was $12.16 per pig sold (Table 4). However, if the distribution of pigs was such that 20, 60 and 20% of pigs were classified as low, medium and high, respectively, then overall profitability was reduced by $0.74 per pig sold.

Table 3. Base data incorporated into the AUSPIG simulation model representing low, medium and high levels of performance of males and females.

Parameter	Performance level					
	Low		Medium		High	
	Males	Females	Males	Females	Males	Females
Weaning weight at 21 d (kg)	4.5	4.5	5.5	5.5	6.5	6.5
Growth rate (g/d)	631	617	674	659	715	697
Sale live weight (kg)	96.0	94.0	103.0	101.0	110.0	108.0
P2 (mm)	14.2	16.5	12.6	14.7	11.4	13.5
FCR (kg/kg)	2.46	2.51	2.33	2.43	2.30	2.36

The same model was used to predict the effect of 20% more M than F pigs being produced in a week. In this scenario, the management options were either to maintain different sized, single-sex groups (i.e. one pen of 144 M and one of 96 F), in which case the stocking rate of the male pen was increased by 20%, or to maintain design group size but with mixed sexes in one group (120 M or 96 F + 24 M). When pigs were overstocked, it was assumed that feed intake was reduced by 10% during the last two weeks and feed conversion worsened by 7%. The model predicted that maintaining single-sex groups, and hence increasing the stocking rate of the males, would reduce profit by $1.77 per pig. However, maintaining equal sized groups increased profit per pig by $0.65 due to the greater number of more profitable entire males being reared in total. However this assumed that there was no affect on performance from having males and females mixed together, which in practice might not be the case.

Table 4. Predicted effect on profitability of variation in growth rate, increased stocking rate, and ratio of males (M) to females (F) in a group of grower/finisher pigs.

		Profit $/pig
Simulation 1	100% medium growth rate pigs, 50% M and 50% F pigs	12.16
Simulation 2	20% low, 60% medium, 20% high performance, M & F pigs	11.42
Simulation 3	Stocking rate of male group increased by 20%	10.39
Simulation 4	50% M and 40% F + 10% M pigs	12.81

Marketing strategies to minimise the effect of variation on returns

Most processors will accept a wide variation in carcass weights and backfat but usually impose price penalties on carcasses outside preferred specifications. If the difference between the average price received for consignments of pigs and the highest

price on a grading schedule is relatively small, the marketing strategy is approaching optimum. A large differential, as may be the case with large numbers of light, heavy and overfat pigs in consignments of pigs marketed on a strict all-out basis, requires a different approach. It may still be possible to market pigs on an all-out basis by selling pigs to the most appropriate buyer if there is access to processors with different market requirements. This approach may incur additional sorting and transport costs, but is likely to be a cost-effective option that meets market demands. Many producers do not have access to a variety of markets and must address the problem of variation by selectively marketing pigs over time. This can be achieved most accurately by weighing and sorting pigs. It is now common practice for producers with multiple shelters to construct a pre-sale shelter in which pigs from several shelters can be weighed and sorted into consignment lots. Alternatively, a pre-determined proportion of pigs from a group may be sold according to a fixed schedule. For example, the heaviest 25% of a group might be sold at 22 weeks, the next heaviest 50% at 23 weeks and the remaining 25% at 24 weeks. Bent and Coleman (1997) determined that such a strategy not only reduced the number of out of range carcasses, but also enabled the average carcass weight of the group to be increased. There is, however, a cost involved in selectively marketing pigs regardless of method used, which must be considered in relation to the rewards received or the penalties incurred from marketing unsorted pigs.

Summary

Variation in production and performance is a very real but often hidden cost to the pig industry that is difficult to quantify. A coefficient of variation of 10% is suggested as a benchmark for live weight when first marketing occurs from groups formed from pigs within one week of age. Some variation in final weight is unavoidable because of small differences in age at entry to a facility, and growth rate differences between males and females. Although crossing system influences herd variation, differences in individual genetic potential create variation in performance within groups of grower/finisher pigs. Disease is a major source of variation. Feed additives can be effective in reducing variation by reducing the proportion of lightweight pigs within groups. Disease control using all/in, all/out management with stringent cleaning of facilities between groups also reduces variation. Variation in weight at entry to weaner facilities is a major source of final weight variation. Decreasing the proportion of lightweight pigs entering a facility by increasing average weaning weight improves the performance of grower/finisher pigs and reduces variation in final weight. The effect of very large group size on variation in performance is not yet fully understood, although evidence suggests that good performance is achievable in large groups. It appears that a certain amount of variation is necessary for maintenance of social order within groups, and that sorting pigs by weight when forming groups does not necessarily reduce variation in final weight. Superimposed on within-group variation is the influence of season which affects performance and carcass quality. Other sources of variation seen on-farm include fluctuations in the production of pigs and disparity in the numbers of males and females born on a weekly basis. Variation in production results in uneven throughput which in turn may increase variation in performance of the grower/finisher herd. The economic impact of variation is difficult to quantify but can be significant because of lost revenue due to sub-optimal carcass values and reduced facility throughput. Profit per pig is also affected by a within-group variation in performance. Few recording systems adequately identify the extent of variation and the associated problems. Producers should adopt more sophisticated analytical techniques which facilitate the review and interpretation of production data, and which can quantify the cost effectiveness of interventions aimed at reducing variation.

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THE EFFECTS OF OIL SPRAYING ON AIR QUALITY IN TRADITIONAL WEANER ACCOMMODATION

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The negative effects of airborne particles on human and animal health, welfare and productivity are well documented (Cargill *et al.*, 1998). Suspended particles can also absorb toxic and odorous gases as well as components of decaying bacteria and act as vectors for these and other pollutants. Reducing the concentration of airborne particles in piggery buildings is therefore important and can improve production efficiency and animal welfare, and reduce the potentially harmful effects of particle inhalation to humans (Donham *et al.*, 1989). The objective of this research was to evaluate the effects of spraying a mixture of oil and water directly onto pen floors inside weaner accommodation on the concentration of airborne particles.

Air quality parameters were recorded for two days (control period) in a partially-slatted, mechanically-ventilated weaner room housing approximately 85 pigs (mean live weight 17 kg) at the stocking rate of 0.3 m^2/pig. The floor of the same room was then sprayed daily for two days with a mixture of canola oil and water (50:50) at the rate of 3 g/pig (6.3 g/m^2), using a hand-held sprayer. The air quality parameters were compared between the two treatments. The experiment was repeated three times at fortnightly intervals and the data were pooled for analyses.

Airborne viable bacteria and respirable and total particles were measured as previously described (Banhazi and Cargill, 1997). Dust pumps were operated from 0900 to 1700 hours, corresponding with highest pig activity. Carbon dioxide was monitored throughout the experiment, using a Masterman Gas Monitoring Machine, to confirm that ventilation rates were similar on all days.

The concentration of carbon dioxide did not vary significantly throughout the experiment but there was a statistically significant reduction in the concentration of airborne viable bacteria and total and respirable airborne particles (Table 1).

Table 1. Concentrations of respirable and total airborne particles, viable bacteria and carbon dioxide for the control and treatment periods.

Treatment	Respirable dust (mg/m^3)	Total dust (mg/m^3)	Viable bacteria (CFU/m^3)	Carbon dioxide (ppm)
Control	0.46[a]	2.98[a]	138,000[a]	1120[a]
Treatment	0.32[b]	2.01[b]	103,000[b]	1090[a]
Reduction (%)	30	33	25	N/A

[a,b]Values in the same column with different superscripts are significantly different ($P \leq 0.05$).

The experiment achieved its aim of demonstrating a reduction in the concentrations of both airborne particles and airborne viable bacteria in the airspace following the direct spraying of an oil and water mixture onto the floor. However, further studies are needed to determine the duration of the positive effects gained, the frequency of spraying, the minimum amount of oil necessary and the possible negative effects of spraying an oil and water mixture on subsequent surface hygiene.

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THE EFFECTS OF OIL SPRAYING ON AIR QUALITY IN A STRAW BASED SHELTER

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The concentration of respirable particles reported in straw based shelters is significantly higher than in other forms of pig buildings (Banhazi and Cargill, 1999). Although straw based shelters are popular due to their low capital requirement and good production potential, it is hypothesised that reduced dust levels in these shelters can result in further improvements in animal health, welfare and productivity. An experiment was designed to evaluate the effects of spraying a mixture of oil and water directly onto the straw, using a hand-held chemical sprayer, to control viable and non-viable airborne particles inside straw-based shelters to reduce airborne particle loads.

Air quality parameters were recorded for two days (control period) in a straw-based shelter housing approximately 105 pigs (mean live weight 70 kg) at the stocking rate of 1.35 m^2/pig. The straw on the floor was then sprayed daily for two days with a 50:50 ratio mixture of canola oil and water at the rate of 6 g/pig. The air quality parameters were then compared between the two periods. The experiment was repeated three times at weekly intervals with a new layer of straw added before each experiment and the data were pooled for analysis.

Ammonia, carbon dioxide, airborne viable bacteria and respirable and total particles were measured as previously described (Cargill and Banhazi, 1997).

The concentrations of ammonia and carbon dioxide were unaffected by the treatment, but there was a statistically significant reduction in the concentrations of total and respirable airborne particles and airborne bacteria (Table 1).

Table 1. Concentrations of respirable and total particles, viable bacteria, ammonia and carbon dioxide for the control and treatment periods.

Treatment	Respirable dust (mg/m^3)	Total dust (mg/m^3)	Viable bacteria (CFU/m^3)	Ammonia (ppm)	Carbon dioxide (ppm)
Control	0.35^a	3.99^a	$350{,}000^a$	1.8^a	479^a
Treatment	0.22^b	3.11^b	$176{,}000^b$	1.9^a	465^a
Reduction %	37	22	50	N/A	N/A

^{a,b}Values in the same column with different superscripts are significantly different ($P \leq 0.05$).

Although spraying a mixture of oil and water onto the straw significantly reduced the concentration of respirable particles to below the recommended maximum target level for pig health and welfare, the concentrations of airborne viable bacteria and the total particle load remained above the recommended maximum value. Further studies are needed to determine if spraying over a longer period, or more frequent spraying of the straw, would suppress the generation of airborne particles and bioaerosols more effectively. The effect of long-term spraying on the quality and absorbent characteristics of straw also requires investigation.

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PIG RESEARCH AND DEVELOPMENT CORPORATION

THE FEEDING BEHAVIOUR OF MALE GROWING PIGS HOUSED IN DEEP-LITTER AND CONVENTIONAL HOUSING SYSTEMS

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Housing growing pigs in large sheds using a floor base of deep litter has been developed as an alternative housing system for pigs. This system is cheaper to establish and is perceived as being more "welfare-friendly" for pigs, compared to conventional intensive systems. Recent industry records have shown that pigs in deep-litter systems are about 10% less efficient in converting feed to live weight and are 1-2 mm fatter (P2 back fat) compared to conventionally housed pigs.

In a preliminary experiment ethograms of behaviour for growing pigs in deep-litter (rice hulls; 0.7 kg/pig/d) and conventional housing systems were developed (R. Sargent, unpublished). The aim of the present experiment was to compare the feeding behaviour of male growing pigs housed in these two systems. Eight hundred and eighty Large White x Landrace entire male pigs were used in the experiment. There were 200 pigs/pen (1 m^2/pig - 17 pigs/feeder) in the deep-litter system and 15 pigs/pen (0.69 m^2/pig - 15 pigs/feeder) in the conventional system. The deep litter pens were 10 x 20 m (width x length) and the conventional pens were 2.7 x 3.6 m. Double spaced, wet-dry feeders were used in both treatments and feed was offered *ad libitum*. Four replicates were used, with 10 animals per treatment per replicate randomly selected as focal animals for observation. Time in the feeding area (i.e., within about 1 m of the feeder) and time with head in the feeder (termed "feeding") were observed during daylight hours using time-lapse video from 20-22 weeks of age. Analysis of variance was used to analyze treatment effects with the group used as the experimental unit.

Table 1. Feeding behaviour of male growing pigs housed in deep-litter and conventional housing systems (data for focal animals observed).

	Deep-litter system	Conventional system	SED	P
Total time within 1 m of feeder (s)[1]	3776	6483	759.9	0.012
Total time feeding (s)	1554	1724	198.5	0.425
Number of feeding bouts	30.5	52.9	4.32	0.002
Average duration of feeding bouts (s)	52.4	33.3	6.96	0.034
Number of social interactions within 1 m of the feeder	17	37.8	8.19	0.044

[1]Seconds.

Observations on feeding behaviour indicated that conventionally-housed pigs spent more time within 1 m of the feeder, had more feeding bouts and a shorter duration of feeding compared to pigs on deep-litter. Pigs in the conventional system also had a higher frequency of social interactions around the feeder than those in the deep-litter system, which may have caused more interruptions during feeding for pigs in the conventional system. The difficulties in gaining and maintaining feeder access in the conventional pens may be responsible for the shorter but more frequent feeding bouts observed. It is possible that the relatively unrestricted feeding that occurs in the deep-litter treatment may result in increased fat deposition and poorer feed conversion efficiency because of changes in feeding patterns that affect feed utilization.

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EFFECT OF TRIPHOSPHOR AND PASCAL RED LIGHTING ON PRODUCTION AND BEHAVIOUR OF WEANERS

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Despite considerable research effort to improve poor post weaning feed intake the problem persists, especially in the first week after weaning. Groups of newly weaned pigs from different litters are aggressive (McGlone *et al.*, 1987) and can take up to a week (Graves *et al.*, 1978) to establish their dominance orders. There has been limited success with lighting strategies to reduce aggression in pigs and improve feed intake. This study examined whether providing the lighting that pigs were accustomed to in the pre-weaning period (red lighting at night from infrared lamps) could improve production and behaviour. The experiment was conducted in a weaner facility comprising four rooms each with six pens (three pens/sex; stocking rate 0.32 m^2/pig) of 15 Large White pigs maintained at 28-30°C in the first week. Two of the rooms (control lighting) had four single cool-white fluorescent tubes (lights on 0730 h; lights off 1630 h) with natural light entering the room via small louvred windows. The other two rooms (treatment lighting) were each fitted with one triphosphor daylight tube (lights on 0730 h; lights off at sunset) and one pascal red tube which provided night lighting after sunset. At weaning the treatment group were provided 24 hours of light with a 20 min daily reduction in pascal red light. There were two replicates per treatment with live weight, feed usage and feed conversion determined in the first week. Video cameras recorded behaviour for 3 h over four periods; at mixing, on day 3 and from sunset on the first and third day after weaning. All social, harmful social (nosing other pigs, being nosed, tail biting, tail bitten, tail sucking, tail sucked) and aggressive behaviours were recorded. The randomised block experiment was repeated and data pooled. General linear modelling procedures were used to analyse the data for the main effects of treatment, sex, period of weaning and interactions.

Table 1. Effect of triphosphor (TP) and pascal red (R) lighting on production and incidence of pig behaviours (events/pig/h) for weaners from 17-24 days of age.

Treatment	LW[2] 17 d (kg)	LW 24 d (kg)	FI[3] 17-24 d (kg/pig)	FCR[4] 17-24 d	Stood on	Chew ears	Nose abdomen
TP/Pascal R	6.169	6.561	1.133	0.38	1.89	1.46	0.09
Control	6.373	6.826	0.865	0.53	2.39	1.12	0.16
lsd[1]	NS	NS	0.23	NS	0.26	0.22	0.06

[1]lsd, least significant difference ($P \leq 0.05$); NS, not significant in analysis of variance ($P > 0.05$). [2]LW, live weight. [3]FI, feed intake. [4]FCR, feed conversion ratio.

Pigs weaned at 17 days and provided alternative lighting showed no significant improvement in live weight (LW) or feed conversion ratio (FCR) in the first week, despite an improvement in feed intake (FI) (Table 1). Weaners provided alternative lighting exhibited an increase in the incidence of ear chewing but a reduced incidence of both nosing the abdomen and being stood on by other pigs (Table 1). There were, however, some important treatment x period of weaning interactions. Pigs provided pascal red lighting on the first night of weaning engaged in significantly higher incidences (events/pig/h) of nosing (3.58 vs 2.55), tail sucking (0.11 vs 0.02), head thrusting (0.76 vs 0.27), fighting (1.36 vs 0.45) and ear chewing (1.65 vs 0.64) compared to control pigs. The improvement in feed intake of weaners presumably occurs because the extra red light provided at night stimulated feeding behaviour but caused an increase in the incidence of aggression. The trend for poorer FCR in the control group might be explained by the higher level of aggressive behaviours in the treatment group.

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VARIABILITY OF WATER INTAKE IN STALLED SOWS

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Water medication is regarded as an efficient mechanism for delivering a range of products used to control and treat bacterial diseases in pigs (Pointon *et al.*, 1995). Although a number of authors provide recommended intakes and flow rates of water for both non-lactating and lactating sows, there appears to be little documented evidence of the extent of variation in water consumption among sows in a herd (Henry and Upson, 1992).

Water consumption was measured over a 24-hour period for 6 consecutive days in two experiments. A group of 20 pregnant sows, with a mean body live weight of 223 kg (174 to 320 kg) was used in the first experiment and a similar group weighing 205 kg (176 to 236 kg) was used in second experiment. In two further experiments, using the heavier group of sows, water intake was measured over two 8-hour periods for 4 days, with and without water deprivation for 16 hours overnight. The mean intake was recorded for each sow, and the mean ± standard deviation (SD) for water consumption in sows are presented in Table 1.

The sows were fed once daily with 2.7 kg of a standard commercial diet containing 16% protein. Each sow was supplied with water, from a nipple drinker attached to an individual overhead tank, delivered at a flow rate of 1.2 l/min. A curved metal plate placed below the nipple collected any water that was spilt into a container. The amount of spilt water was measured and deducted from the total water used to obtain the daily consumption for each sow.

Table 1. The mean (± SD) daily (24 hour) water consumption for sows over 6 days and the mean (± SD) daily 8-hour water consumption for sows over 4 days with and without overnight water deprivation.

Experiment	Daily water intake over a 24 h period (Experiment 1)	Daily water intake over a 24 h period (Experiment 2)	Daily 8-h water intake with 16 h overnight deprivation	Daily 8-h water intake without overnight deprivation
Absolute range (l)	0.5 – 42.5	2.0 – 40.0	2.0 – 26.25	0.0 – 18.5
All sows (mean ± SD)	10.95 ± 9.93	12.78 ± 10.32	9.18 ± 6.33	5.13 ± 3.68

The mean water consumption of sows was less than recorded by Madec *et al.* (1986) but the range for individual sows was similar. Although the daily 24-hour water intake for individual sows varied from ± 24% to ± 126% around the mean, the variation among individual sows was considerably greater. In both 24-hour experiments, there was a significant difference ($P<0.001$) in water intake levels between the 10 sows with the lowest intake (means 4.2; 5.6 l) and the 10 sows with the highest intake (means 17.4 l; 19.9 l). Depriving sows of water overnight for 16 hours reduced the variation between the highest intake group (mean 13.2 l) and lowest intake group (mean 5.1 l), but the difference was still significant ($P<0.001$). In the 24-hour experiments, the mean quantity of water wasted was 4.48 ±4.87 l/day and 4.9 ±4.23 l/day, respectively.

The results indicate that caution needs to be exercised to avoid over and under dosing animals, when medicating sows via water.

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NUMERICAL MODELLING OF AIR TEMPERATURE AND VELOCITY IN A FORCED VENTILATION PIGGERY

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Pigs are reared under more intensive conditions than other farm animals because of their unique nature. Growth varies with the environment and pigs are very responsive to climatic variation (Kilgour and Dalton, 1984; Turner *et al.*, 1997). Therefore, they are subjected to intensive environment control and management for higher productivity.

The aim of the current work was to numerically model air speed and temperature in a forced ventilation piggery as an aid to achieving optimum environmental control. This approach can also help to identify problems in the design of piggeries and offer suggestions for improvements. A steady two-dimensional numerical model, which allowed for the effects of buoyancy, turbulence and heat generated by the pigs was chosen. Computational fluid dynamics software (Fluent) was used in the modelling exercise. This software solves the continuity, momentum, and energy equations in differential form using the integral volume method. The equations were solved using a semi-implicit algorithm with an iterative line-by-line matrix solver.

As an example, the effect of varying the ventilation rate for a specific design is presented in Figure 1. This piggery is occupied with weaner pigs (5-20 kg live weight) at a density of 0.30 m^2/pig. The ventilation system is a forced extraction fan type with four stages. The floor is fully slotted and open to an air space below the floor, from where the air is removed by extraction fans. The air inlet, which is a fixed size opening, is located in the ceiling at the opposite side of the extraction fans.

The fans provide airflows of 0.72-2.0 m^3/s. The effect of ventilation rate has been investigated for a winter day where the outside temperature was assumed to be 5°C. Pigs were modelled as cylinders 0.15 m in diameter and generating 20 w/pig of heat. The floor was set at 5°C while the roof was set at 15°C taking into account the effect of solar radiation gained by the steel roof during daylight hours. The walls and the ceiling were assumed to be perfectly insulated.

Table 1. Effect of ventilation on the temperature range at the pigs' level.

Inlet velocity (m/s)	Ventilation rate (m^3/s)	Temperature range (°C)
0.16	0.717	16.70 – 22.00
0.23	1.030	14.65 – 20.00
0.30	1.344	13.10 – 18.30
0.37	1.658	12.20 – 17.00
0.45	2.016	12.20 – 15.50

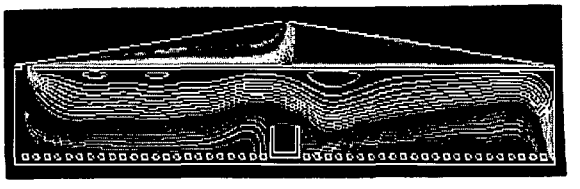

Figure 1. *Air streamlines in the piggery with ventilation rate of 0.717 m^3/s.*

The temperature range at the pigs' level decreased as the air ventilation rate is increased (Table 1). The lower temperatures were at the inlet side while the higher temperatures were at the exit side due to the heat generated by the pigs. To achieve smaller temperature variations, a central positioning of the air inlet and two exhaust fans, one on each side would seem more ideal for this size shed. The predicted air movement, (Figure 1) shows how a solid passageway barrier should be avoided since it obstructs the airflow. Knowledge of the airflows and temperatures can also help in choosing the location of a thermostat so that optimum control of the climate can be achieved. Computational fluid dynamics software such as Fluent can be of great assistance in the design and analysis of forced ventilated piggeries.

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VARIATION IN BIOLOGICAL TRAITS OF OUTDOOR SOWS

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While the modern, intensive outdoor unit is considered to be the most cost-effective production system in some climates (McGlone, 1997), a recent report on welfare aspects of outdoor pig production listed 48 recommendations for either allowed practices or for the need for more research (Farm Animal Welfare Council, 1996). The following study compared the welfare of sows in indoor and outdoor.

Risks to welfare were examined on the basis of the variation that occurs in so-called 'fitness traits' such as growth, survival and reproduction. The rationale is that animals will be at greater risk in situations where the variation in these traits is greatest. For example, a large variation may be due to increases in competition for food resulting in lighter pigs and lower backfat, and social and physical factors affecting reproductive performance.

The experiment involved four indoor and four outdoor farms in Victoria with measurements made on groups of mated sows 8-9 weeks post-mating during each season. Indoor sows were all group housed at the time of measurement but one of the farms had a period of stall housing post-mating. The unit of measurement was the farm. Measurements made in February, April, June and September included body weight, backfat, the number and length of lesions on the head and shoulders, rectal temperature, claw length and gait and lameness, determined over a 10 m route after leaving the measuring crate, using a 5-point scale. The farms provided reproductive records. Data were analysed by a 2-way analysis of variance for treatment x season effects and by the chi-square test. Treatment but not seasonal effects are presented below.

Table 1. Coefficient of variation (%) for morphological and reproductive traits in dry sows housed indoors and outdoors

Trait	Indoors	Outdoors	SED
Backfat	24.1^{a}	28.3^{b}	1.69
Number of lesions	84.0^{p}	272.0^{q}	52.9
Length of lesions	52.0^{x}	258.0^{y}	52.0
Farrowing rate	23.5^{x}	50.4^{y}	7.36

$^{ab, pq, xy}$Values in the same row with different superscripts are significantly different at P≤0.05, P≤0.01 and P≤0.001, respectively.

A total of 868 indoor housed sows and 372 outdoor housed sows were examined. Effects of treatment on the coefficients of variation were only evident for backfat, number and length of lesions and farrowing rate (Table 1). There were no effects of treatment on the coefficients of variation for body weight (13.7 vs 14.0 %), rectal temperature (1.6 vs 1.8 %), claw length score (159 vs 40 %), lameness score (32 vs 24 %), gait score (27 vs 35 %), piglets born alive (26 vs 24 %) and piglets weaned (19 vs 16 %). While 93 % of indoor sows had a normal claw length score of 0, only 3 % of outdoor sows were in this category ($X^2_1 = 352$; P<0.001). The greater variation in length and number of lesions was an artefact due to the low number but variable length of lesions in outdoor sows.

The data from this experiment suggest there were no large differences in risks to welfare, on the basis that variation in a number of traits was similar between sows housed indoors and outdoors. However, the greater variations in backfat and claw length suggest areas that warrant further research.

We are grateful to the producers who allowed this work to be conducted on their properties.

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AN ON-FARM COMPARISON OF THE WERRIBEE FARROWING PEN AND CONVENTIONAL FARROWING CRATES

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The Werribee Farrowing Pen (WFP) was developed from research on sow and piglet behaviour. In the WFP the sow is not restrained and is provided with rice-hull bedding at farrowing to stimulate pre-partum nesting behaviour. In addition, the piglets' requirements for a small, warm, safe area in the pen have been incorporated. Information about the WFP is available at:

http://www.animal-welfare.org.au/farrow_pen/werribee_pens1.html

This trial compared the productivity of sows and litters in WFPs and conventional farrowing crates (CFCs) over 1.5 years on a pig farm in Victoria with 330 commercial PIC® breeding sows at a ratio of 4:1 Camborough 15 and 22. Four WFPs (2.33 m wide x 3.5 m deep, with sloping concrete floor) and 5 CFCs (Proctor® crates 1.6 m x 2.3 m, with full-mesh woven wire floor, raised 0.2 m above shed floor level) were constructed side-by-side in a row in an uninsulated grower shed. A heated creep area was provided in both treatments. Weaning occurred on day 22 ± 3.6 (mean ± SD) of lactation. The trial involved 146 sows and their litters (66 in WFPs and 80 in CFCs) over 17 farrowing batches. Sow parity number averaged 2.8 ± 1.8 (range 1 - 8). After farrowing, sows were offered an increasing quantity of feed daily to day 7 of lactation, and thereafter were fed to appetite. Water was available *ad libitum* from nipple drinkers. Sows were fed twice daily and in 6 of the 17 batches, the amount of feed provided was recorded daily. Quantitative data were analysed using one-way ANOVA blocked on farrowing batch (Genstat 5 for Windows, Release 4.1). Differences between the housing treatments in piglet mortality variates were analysed using the Chi-squared test on counts of litters.

There were no treatment differences in the number of piglets born per litter (total born (TB) 11.5 ± 3.0), number born alive (10.8 ± 2.8), the proportion of stillbirths (6.5% of TB) or the number weaned (9.4 ± 1.2). Cross fostering to standardize litter size to 10 piglets occurred in about 95% of litters in both treatments, with an average net increase of 0.3 and 0.7 piglets per litter fostered in the WFP and CFC treatments. There was no difference in pre-weaning mortality in the WFP (15.5% of live born) and the CFC (17.5%). The main cause of death in the WFPs was overlaying by the sow (45% of deaths compared to 20% for CFCs; χ^2_3=7.6, P=0.06), while the main cause of death in the CFCs was the combination of small/weak/non-viable (31% of deaths compared to 14% in WFPs; χ^2_3=7.1, P=0.08). Piglet deaths attributed to chilling were similar between the treatments (14 vs 16% of deaths in the WFP and CFC treatments), but chilling may have contributed to other causes of mortality such as overlay. As expected, approximately 70% of deaths occurred in the first three days post-partum. Over the first 21 days of lactation, sows in the WFPs compared to CFCs were fed 0.7 kg more feed per day (average values for six farrowing batches 6.7 ± 1.0 vs 6.0 ± 0.7 kg/sow/day, respectively; P<0.02). The difference between treatments was greatest in the third week of lactation (8.0 ± 1.4 vs 6.8 ± 1.3 kg/sow/day, respectively, P<0.02).

In conclusion, the results suggest that sows and litters perform at least as well in the WFP as in farrowing crates. The increased feed intake by sows in the WFPs may have positive consequences for milk production, piglet growth and long-term prolificacy, although these were not measured. These benefits may help offset the requirement for increased floor area for pen compared to crate farrowing systems. Piglet mortality in this trial was higher than expected, particularly overlay deaths in the WFP and small/weak/non-viable piglets in the CFC treatment. However, it was probably exacerbated by the absence of insulation in the building, with cold or draughty conditions predisposing piglets to increased risk of chilling as either the primary or secondary causes of death.

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PIG RESEARCH AND DEVELOPMENT CORPORATION

THE EFFECTS OF RIDGE VENT SIZE ON AIR QUALITY

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Elevated concentrations of ammonia gas and bioaerosols in the air space of naturally-ventilated pig sheds adversely affect the health and growth rate of growing pigs (Cargill and Skirrow, 1997). Controlling ventilation rates in naturally-ventilated sheds is achieved mainly by manipulating ridge-vent and wall-vent openings. Although ventilation rates influence pollutant concentration (Cargill and Skirrow, 1997), the effect of ridge-vent size on air quality has not been reported.

To evaluate the effect of ridge-vent size on air quality, an existing shed with a narrow ridge-vent (0.3 m wide with a cap 0.1 m above) was divided into two sections with a solid wall. A wide ridge-vent (1.1 m wide with a cap 0.55 m above) was installed in one section. The dimensions of the shed were 11 m x 55 m x 2.9 m (average height). The wall height at the eaves was 2.6 m. The side-shutters were hinged from the top, providing an opening of 1.6 m. Effluent disposal was via a pit, flushed daily, beneath slats down the centre of the shed. The mean stocking rate and the mean stocking density in each section were 0.56 m^2/pig and 2.78 m^3/pig, respectively.

Ammonia and carbon dioxide were measured using Kitagawa® gas tubes. Airborne viable bacteria and respirable and total particles were measured as described by Banhazi and Cargill (1997). Concentrations of gas and bacteria were measured at 0600, 1200 and 1800 hours and airborne particles were measured over an 8-hour period from 0800 hours. Sections were monitored on two consecutive days on six occasions at 2-weekly intervals during spring. The side-shutters were either opened or closed 12 hours prior to monitoring, and ridge vents remained half-open throughout the experiment. Each of the observations was used as a replicate in the data analysis and the concentrations of pollutants are reported in Table 1.

Table 1. Mean concentrations of ammonia, carbon dioxide, airborne bacteria and airborne particles recorded in each section of the shed for six monitoring periods.

Ridge size	Shutters	Ammonia (ppm)	Carbon dioxide (ppm)	Viable bacteria (cfu/m^3)	Total particles (mg/m^3)	Respirable particles (mg/m^3)
Narrow	Open	5.5[a]	700[a]	119,000[a]	2.9[a]	0.30[a]
Wide	Open	3.5[a]	525[b]	68,000[b]	2.2[b]	0.23[b]
Narrow	Closed	12.0[c]	1,500[c]	146,000[c]	2.8[a]	0.28[a]
Wide	Closed	4.5[a]	800[d]	89,000[d]	2.1[b]	0.21[b]
Maximum acceptable[1]		7.0	1,500	100,000	2.4	0.23

[a,b,c,d]Values in columns with different superscripts are significantly different ($P \leq 0.01$).
[1]Cargill and Skirrow (1997).

Increasing the size of the ridge vent had a positive effect on air quality when side-shutters were both open or closed, significantly reducing all parameters to within recommended concentrations. The most positive benefit occurred when side-shutters were closed. In this setting there was a significant reduction in all pollutants ($P<0.01$) in the section with a wide ridge-vent. The build up in gases that is usually recorded when sheds are closed up was also avoided, indicating that ventilation was significantly improved.

The results confirm the value of installing wide ridge-vents in naturally-ventilated sheds as an aid to ventilation and maintaining good air quality.

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PIG RESEARCH AND DEVELOPMENT CORPORATION

THE EFFECTS OF AGE SEGREGATED REARING ON AIR QUALITY AND PRODUCTION EFFICIENCY – A CASE STUDY

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Age Segregated Rearing (ASR), which incorporates all-in/all-out management and cleaning between batches, is a practical way of improving air quality in pig sheds, reducing the incidence and severity of respiratory disease and improving production efficiency and animal welfare (Cargill *et al.*, 1997; Clark *et al.*, 1994). It also has the potential to reduce Occupational Health and Safety risks associated with human exposure to airborne pollutants in piggery buildings. However, most of the Australian data is based on experiments conducted on farms comparing both continuous flow (CF) and ASR management in the same sheds. Little data is available on the productivity in ASR systems when fully implemented on a commercial farm.

A herd with 180 Large White x Landrace sows was selected to evaluate the implementation of ASR under fully commercial conditions. Air quality parameters, respiratory health data and production figures were recorded on the farm for the 6-month period prior to the implementation of an ASR management system (CF period), and for the same 6-month period towards the end of the first year of ASR management (ASR period). The air quality parameters, production efficiency and respiratory health of the pigs were then compared between the two periods. No significant genetic changes were made during the monitoring period. Ammonia and carbon dioxide, airborne viable bacteria and respirable and total particles were measured as previously described (Banhazi and Cargill, 1997).

Table 1. Average growth rate, feed:gain ratio, pleurisy prevalence, and the concentrations of airborne particles, viable bacteria, and ammonia for the continuous flow management (CF) and Age Segregated Rearing (ASR) periods.

Treatment	Ammonia (ppm)	Respirable particles (mg/m^3)	Total Particles (mg/m^3)	Viable bacteria (CFU/m^3)	Pleurisy prevalence (%)	Growth rate[1] (g/day)	Feed: Gain Ratio
CF	5.4[a]	0.307[a]	2.09[a]	147,000[a]	45.0[a]	550	3.11
ASR	3.9[b]	0.201[b]	1.79[a]	83,000[b]	29.7[b]	630	2.88
Reduction (%)	27.7	34.5	14.0	43.5	34.0	14.5	7.4

[a,b]Values in the same column with different superscripts are significantly different ($P \leq 0.05$). [1]From birth to slaughter.

Following the implementation of ASR management, the concentration of airborne pollutants decreased significantly, while production parameters improved markedly (Table 1). While the percentage of pigs with lung lesions decreased marginally, there was a 33% reduction in lung scores and pleurisy prevalence, associated with an improvement of 14.5% in growth rate and 7.4% in feed:gain ratio. The reduction in pleurisy prevalence was associated with a reduction in the percentage of pigs with pleuropneumonia lesions from 8.2% to 0.2%.

The health and production improvements achieved on the farm were greater than previously described in experimental data, suggesting that experimental data represents the minimum improvement that can be expected. Whole farm conversion to ASR resulted in much greater respiratory disease control and was associated with a reduction in the usage of antibiotics.

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PIG RESEARCH AND DEVELOPMENT CORPORATION

INFLUENCE OF XYLANASE ADDITION TO DIETS CONTAINING WHEAT CO-PRODUCTS AND NUTRITIONALLY-DEFINED WHEAT ON GROWING PIG PERFORMANCE

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Wheat and wheat co-products vary in their available energy and amino acid content (Batterham *et al.*, 1980) leading to unpredictable growth rate and feed:gain. Carbohydrase enzymes, targeting the non-starch polysaccharides in these raw materials, may improve their nutrient availability. This experiment examined the response of pigs to the use of a fungal xylanase, produced by *Trichoderma longibrachiatum*, in diets containing maize/wheat co-products or wheat/wheat co-products. The feeding value of the wheat was established in an earlier experiment, and was designated as 'high', 'medium' or 'low' quality.

There were eight dietary treatments. A positive control diet based on maize and soya bean meal was formulated on a digestible energy (DE) basis to contain 14.4 MJ DE/kg and 0.7 g available lysine/MJ DE. Two additional treatments were compared following substitution of 200 g/kg of the maize with wheat co-products (millrun, a bran/pollard mixture) with or without addition of xylanase (4000 U/kg feed). A second positive control diet (14.0 MJ DE/kg and 0.7 g available lysine/MJ DE) containing 'high' quality wheat was formulated for comparison with four additional treatments where the 'high' quality wheat was substituted with 'medium' or 'low' quality wheat (650 g/kg) and millrun (50 g/kg), with or without addition of xylanase (4000 U/kg feed). Fifty-nine individually penned boars were used (Bunge synthetic genotype), with 7-8 boars per dietary treatment. Growth and feed conversion were measured over a 5-week period from 28 kg live weight.

Table 1. Effect of xylanase on performance of pigs fed the experimental diets.

Treatment	Xylanase (-/+)	Finish weight (kg)	Daily gain (g)	Daily feed intake (kg)	Feed:gain
Maize	-	61.2	944	1.812[a]	1.92
Maize + millrun	-	59.1	886	1.651[b]	1.88
Maize + millrun	+	61.4	938	1.705[ab]	1.82
SEM		0.971 (NS)	19.2 (NS)	0.040*	0.037 (NS)
Wheat- high	-	61.9	960[a]	1.772[a]	1.84
Wheat- medium	-	60.2	918[ab]	1.622[ab]	1.80
Wheat- medium	+	61.2	945[a]	1.710[ab]	1.81
Wheat- low	-	58.3	878[b]	1.576[b]	1.76
Wheat- low	+	61.5	952[a]	1.728[a]	1.81
SEM		0.651 (NS)	12.4*	0.033*	0.021 (NS)

[a,b]Values within columns and diet types (maize or wheat based) with different superscripts are significantly different. *P≤0.05 (one way analysis of variance), NS not significant.

Addition of millrun to the maize diet significantly reduced feed intake, with numerical reductions in growth rate. The xylanase group was intermediate. Feeding medium and low quality wheat sequentially reduced feed intake and daily gain. Xylanase addition significantly improved performance in low quality wheat to a level equivalent to that of the high quality wheat control.

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THE EFFECT OF STEEPING AND ENZYME SUPPLEMENTATION ON THE PERFORMANCE OF LIQUID-FED WEANER PIGS

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Providing newly weaned piglets with a liquid diet may aid the transition from a highly digestible milk-based diet to a dry pellet or mash diet. Steeping the diet leads to the proliferation of *Lactobacillus* bacteria, which occur naturally in the grain. This increases the acidity of the mixture, which aids in the digestive process. Steeping a liquid diet has also been associated with the activation of endogenous enzymes in the grain, therefore improving the availability of nutrients (Brooks *et al.*, 1996). Enzymes can enhance the digestibility of dry diets, and it has been shown that they are more effective in a liquid medium if adequate steeping time is allowed (Brooks *et al.*, 1996). The current study examined the effects of steeping time and enzyme supplementation on the performance of weaner pigs fed liquid diets.

Sixty male Large White x Landrace pigs (weaned at 27 d) were randomly allocated to a 2 x 2 factorial experiment including steeping time (1 h vs 15 h) and enzyme addition (+ or – enzyme). Diet was mixed in a ratio of 2.5 l/kg at 25°C. A wheat-based diet (3.2 mm screen) was prepared. A long chain alkane ($C_{36}H_{74}$) was added (50 g/t) to the feed as a digestibility marker. A commercial xylanase was added at the point of mixing the liquid diets (0.4 g/kg).

Steeping the diet for 15 h improved feed intake (P=0.001), growth rate (P=0.03) and 21-d weight (P=0.031). There were also significant steeping x enzyme interactions on growth rate (P=0.045) and 21-d weight (P=0.045) with the enzyme improving both parameters in pigs offered the diet steeped for 1 h but not in those fed the diet steeped for 15 h. Steeping time and enzyme supplementation did not affect FCR.

Table 1. The effects of steeping time and NSP enzyme supplementation of liquid, wheat based diets fed to male pigs between 8 and 15 kg live weight (0 - 21 days).

Steeping Time (h)	Enzyme	Starting wt kg	21 day wt kg	Daily gain (g/d)	FCR[1]	Feed intake[1] (g/d)
15	-	8.28	15.42[a]	340[a]	1.07	364[a]
15	+	8.28	15.08[a]	324[a]	1.21	388[a]
1	-	8.32	14.19[b]	279[b]	1.16	310[b]
1	+	8.28	15.02[a]	321[a]	1.14	359[a]
SED		0.047	45.40	21.4	0.06	18.34
Significance						
Steeping (s)		NS	*	*	NS	**
Enzyme (e)		NS	NS	NS	NS	**
s x e interaction		NS	*	*	NS	NS

[a,b,c]Treatment means within a column sharing the same superscripts do not differ significantly (P >0.05). [1]100% DM basis; *P≤0.05; **P≤0.01; NS P>0.05.

The effect of steeping time on feed intake and growth performance is probably due to activation of the endogenous glycanases and phytase, which changed the cell wall structure of the grains. Enzyme supplementation tended to improve feed:gain (P=0.06) of pigs offered feed steeped for 1 h, but not that steeped for 15 h, suggesting that a longer steeping time may allow the endogenous enzymes to act fully on the substrate, thus reducing nutritive value.

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ENZYMES CAN ELIMINATE THE DIFFERENCE IN THE NUTRITIVE VALUE OF WHEATS FOR PIGS

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The feed intake and daily gain of weaner pigs varies widely due to the type of wheat included in their diets (Cadogan *et al.*, 1999). The difference is thought to be related to the cell wall structure and/or the quality and quantity of the non-starch polysaccharides (NSPs) present in the grain. This study investigated the effect of including in the diet a commercial enzyme on the performance of young male pigs fed wheats that had caused very low (Currawong), intermediate (Cocumba) and normal (Lawson) feed intakes in previous studies (Cadogan *et al.*, 1999).

The wheats were included at 650 g/kg in a conventional weaner diet. A commercial enzyme (Roxazyme G2) containing activities of xylanase, β-glucanase and cellulase was added at 120 g/tonne. The diets were offered *ad libitum* to weaners (Large White x Landrace) weighing approximately 7 kg. After a 3-d adaptation period, 12 pigs were allocated to each treatment in individual pens for a 21-d experiment.

Feed intake and daily gain were markedly ($P<0.001$) improved (Table 1). There was a significant wheat x enzyme interaction ($P<0.001$) on both measurements. Enzyme increased daily gain and feed intake by 50.6% and 42.8%, respectively, in pigs fed Currawong, but it had only marginal effects on feed intake and daily gain in pigs fed the other two wheats. Feed conversion ratio (FCR) was not significantly affected.

Table 1. Effects of enzyme supplementation on feed intake, daily gain and feed conversion ratio of male weaner pigs fed three different wheats for a 21-d period.

Wheat	Enzyme	Daily gain (g)	Feed intake (g)	FCR[2]
Currawong	-	230[a]	318[a]	1.38
Currawong	+	466[b]	556[b]	1.23
Cocamba	-	425[b]	540[b]	1.27
Cocamba	+	445[b]	521[b]	1.20
Lawson	-	460[b]	525[b]	1.14
Lawson	+	479[b]	570[b]	1.20
SE		13.9	13.4	0.02
Statistics[1]				
Wheat (W)		***	***	NS
Enzyme (E)		***	***	NS
W x E		***	***	NS

[a,b]Values in the same column followed by different superscripts are significantly different ($P\leq0.05$). [1]NS, not significant; ***$P\leq0.001$. [2]FCR, feed conversion ratio.

The feed intake of pigs fed Currawong and Lawson wheats varied by 50%, leading to a significant difference in weight gain. A feed enzyme product, which had affinities for both soluble and insoluble NSPs, totally eliminated this difference. This indicates that the large differences in feed intake and daily gain in weaner pigs fed different wheats are related to the quality and/or quantity of the NSP components.

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EFFECTS OF NEW SEASON'S WHEAT ON THE GROWTH PERFORMANCE OF YOUNG MALE PIGS

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The new season wheat phenomenon in poultry and the subsequent detrimental effect on apparent metabolizable energy and growth have explained the poor growth and feed efficiency of broilers fed diets containing these wheats (Choct and Hughes, 1997). New seasons wheat phenomenon in pig production has not been reported nor has it been researched.

One hundred and twenty male pigs between 6.5 and 7.5 kg live weight (LW) were selected, placed in individual crates, and offered a common nursery diet for 3 days. Animals were then reweighed and randomly allocated, at an average of 7.5 kg LW, to 10 experimental diets containing 10 new season's wheats collected from Southern NSW and Victoria from the 1996/97 harvest. Pigs were offered diets and water *ad libitum* for 21 days. The basal diet used in the study was formulated to contain 650 g/kg of the 10 different experimental wheat types, and highly digestible raw ingredients. The diets were formulated to contain 14.5 MJ DE/kg and an available lysine content of 0.8 g/MJ DE. Celite was added to the diets as an acid-insoluble marker. The entire experiment was repeated 10 months later. Growth performance and dry matter (DM) digestibilities are presented in Table 1.

Table 1. Effects of new seasons wheat type on the performance of young male pigs offered diets for 21 days, commencing at 7.5 kg live weight

Wheat Cultivar	Final weight (kg)	Daily gain (g)	Feed intake (g/d)	Feed:gain	DM digestibility (%)
Currawong	12.41	233^{a}	271^{a}	1.18	86.4^{abc}
Dollarbird	14.63	341^{b}	388^{b}	1.15	85.0^{cde}
Finley Rosella	15.48	376^{bc}	432^{bc}	1.14	84.7^{cde}
Wimmera Rosella	16.66	433^{c}	476^{c}	1.10	84.2^{cde}
Cocamba	15.78	396^{bc}	438^{bc}	1.11	85.8^{abcd}
Parsons Rosella	15.95	399^{bc}	445^{bc}	1.13	83.3^{e}
Matong	16.15	419^{c}	486^{c}	1.16	84.2^{de}
Triller	16.39	438^{c}	502^{c}	1.15	85.3^{abcd}
Janz	15.73	394^{bc}	432^{bc}	1.09	86.4^{ab}
Lawson	16.45	447^{c}	514^{c}	1.14	86.8^{a}
Significance[1]	***	***	***	NS	***

[1]NS, not significant; ***P≤0.001. ^{a,b,c,d,e}Values in each column with different superscripts are significantly different (P≤0.05).

Wheat variety had a significant influence on voluntary feed intake and daily gain, but no effect on feed:gain. Dry matter digestibility was significantly different among wheats, although, there was no correlation between diet DM digestibility and pig growth performance. The marked differences remained among the test wheats after 10 months, although, feed intake and daily gain were an average of 11.3% (P<0.001) and 9.8% (P<0.001) higher respectively for the aged wheats.

Storage did not reduce the differences among the 10 wheats, indicating that the new season's wheat phenomenon, commonly observed in poultry, was not producing the negative effect. The results demonstrate the extreme variability wheat based diets cause on young pig growth performance, and the need for further research on the components of wheat that effect voluntary feed intake.

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EFFECT OF OIL EXTRACTION PROCESS ON THE TRUE ILEAL DIGESTIBLE REACTIVE LYSINE CONTENT OF CANOLA MEAL

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When ingredients such as canola meal undergo processing, the ε-amino group of lysine can react with other compounds. Heating can result in xenobiotic crosslinks occurring within existing protein chains, such as when the ε-amino group of lysine reacts with a cystine residue to form lysinoalanine. In this instance, the new compound is resistant to degradation during subsequent analysis and decreases in lysine and cystine concentration can be detected (van Barneveld, 1993). However, if processing results in early Maillard reactions and the formation of compounds such as the fructoselysine moiety, these reacted lysine derivatives are acid labile and can revert back to lysine during the acid hydrolysis step of conventional amino acid analysis (Rutherfurd *et al.*, 1997). This leads to an overestimation of lysine content and the digestible lysine content of the processed feed ingredient. This experiment aimed to compare the effects of three oil extraction methods for canola seed on the total, reactive, digestible and digestible reactive lysine content of the resulting meals.

Cold-pressed, expeller extracted and solvent extracted canola meal was prepared commercially from a single source of canola seed. Reactive lysine and digestible reactive lysine content were determined using methods described by Rutherfurd *et al.* (1997). Diets used to determine ileal digestibility were sugar/starch based and contained the canola meals at 300g/kg, respectively. Celite® was added to the diets as an acid-insoluble ash marker. An additional diet containing 126 g/kg of enzymically-hydrolysed casein was included to determine endogenous amino acid losses for true ileal digestibility calculations. Soya bean meal was used as a control. Large White male pigs (35-40 kg body weight) fitted with simple T-piece ileal cannulas were provided diets based on a 5 x 5 Latin square design. Diets were fed for 7 d (3 x maintenance) prior to 8 h digesta collections over 2 consecutive days.

Table 1. Assessment of various methods for determining lysine content (with digestibility coefficients) in cold-pressed, expeller extracted and solvent extracted canola meals.

	Canola meal lysine content (g/kg, air-dry)			Statistics[1]	
Methods	Cold-pressed	Expeller	Solvent	Diet	SEM
Total	17.41	17.25	18.70	-	-
Reactive	13.00	10.88	11.38	-	-
Apparent ID	14.66 (0.84^{a})	13.20 (0.77^{b})	14.74 (0.79^{b})	***	0.012
True ID	16.02 (0.92^{a})	14.49 (0.84^{b})	16.08 (0.86^{b})	***	0.012
Apparent ID reactive	11.15 (0.86^{a})	8.53 (0.78^{b})	9.23 (0.81^{c})	***	0.007
True ID reactive	12.35 (0.95^{a})	9.79 (0.90^{b})	10.35 (0.91^{b})	***	0.007

ID, ileal digestible; SEM, standard error of mean; ***P≤0.001. [1]Analysis of coefficients only. [a,b,c]Values in a row with different superscripts are significantly different (P≤0.05).

Despite similar total lysine concentrations in the canola meals, true ileal reactive lysine digestibility was significantly higher (P<0.001) in the cold-pressed meal (Table 1). Gross reactive lysine content was a good indicator of true ileal digestible reactive lysine content (and thus the degree of heat damage) and may have a role in the routine assessment of canola meal quality. *Supported by the Australian Oilseeds Federation.*

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PREDICTING ENERGY AVAILABILITY IN BARLEY FOR PIGS AND POULTRY USING RAPIDLY DETERMINED FIBRE CONTENT

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The digestible energy (DE) content of barley for pigs ranges from 11.7 to 16.0 MJ/kg, dry matter (DM) (van Barneveld, 1999). Similarly, there is variation in the apparent metabolizable energy (AME) content of barley for poultry ranging from 10.4 to 12.2 MJ/kg DM (Hughes and Choct, 1999). Several factors are thought to be responsible for these variations in energy content including influences exerted by non-starch polysaccharides (NSP). Grain NSP content has been suggested as a predictor of available energy content, however, NSP analysis is tedious and expensive. With this in mind, this study examined the potential for a rapidly determined fibre assay (RDF) as an indirect measurement of available energy in grain.

The content of NSP in 19 barley samples from different regions of Australia was determined using the standard alditol acetate method and a newly developed RDF method. The latter involved milling a 50 g barley subsample through a 1 mm screen. Following this, starch and protein were removed from approximately 1 g of sample using α-amylase/amyloglucosidase and protease, respectively. Ethanol was used to precipitate soluble NSP and remove protein and glucose. The residue was filtered and washed with ethanol and acetone in sintered crucibles. The residue was then dried at 105°C overnight and collected as RDF. *In vivo* DE in pigs and AME in poultry were determined on these samples using total faecal collection methods. Analysis revealed RDF and NSP were correlated with DE and AME and RDF was correlated with NSP (Figures 1 and 2).

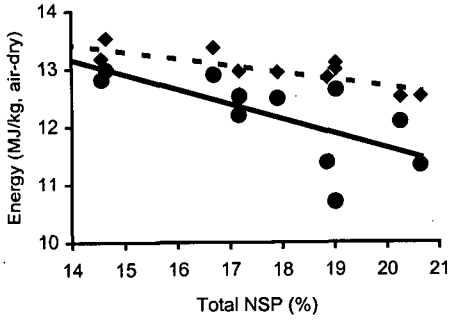

Figure 1. *Correlation between total NSP content and digestible energy (r^2=0.47; ●-●) and apparent metabolizable energy content (r^2=0.52; ♦-♦) of barley.*

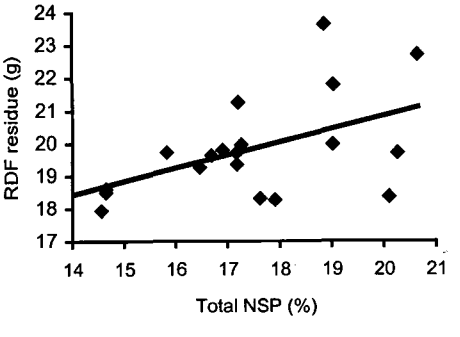

Figure 2. *Correlation between rapidly determined fibre (RDF) residue (g) and total non-starch polysaccharide (NSP) content of barley (r^2=0.32).*

Both DE and AME were significantly correlated (P<0.05) with total NSP (Figure 1) but not with RDF (P>0.05). This suggests total NSP has potential as a predictor of energy availability in barley for monogastric animals. The relationship between RDF and total NSP is weak (Figure 2) and hence further refinements of this assay are required before it can substitute standard NSP analysis.

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PIG RESEARCH AND DEVELOPMENT CORPORATION

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A SYMPOSIUM - DEVELOPMENT AND IMPLEMENTATION OF GENETIC IMPROVEMENT TECHNOLOGIES IN PIG BREEDING

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Introduction

The main aim of genetic improvement is to increase profitability. In the introduction to the genetics symposium at the APSA conference in 1989 Mike Goddard described the pig farmers' objective as "select boars and sows with the highest breeding value for profit". Ten years later, this objective has not changed. However, pig breeders today are able to use better tools to achieve this objective. In 1989, the genetics symposium focussed on the use of Best Linear Unbiased Prediction (BLUP) technology for genetic improvement (Long, 1989; Fyfe, 1989). During the last decade, this technology has been adopted by pig breeders through the provision of "black boxes" of specialised computer programs, which aid breeders in their selection decisions (Nicholas, 1997). These selection decisions have been based only on phenotypic performance records so far. However, developments in molecular genetics will provide information about the underlying genes influencing economically important traits.

Information from molecular genetics is expected to be especially useful for genetic improvement of disease resistance. Overall, genetic improvement so far has focussed mainly on efficient lean meat growth, reproduction and meat quality and presentations at APSA have focused on these topics (McPhee, 1989; Treacy, 1989; Webb, 1991; Hermesch, 1997). Selection for improved performance in these traits may have had undesired effects on the disease resistance of pigs contributing indirectly to the "Growth Gap" between pigs in production systems of high and low health status.

The objective of this symposium is to explore the use of information derived from DNA technologies for genetic improvement of pig production and to investigate possibilities to breed for increased disease resistance in pigs.

The first paper by Goddard (1999) provides background information and principles of genetic improvement to set the scene for this symposium. The paper firstly describes principles of breeding objectives and selection methods currently in use. The paper then explains how economically important traits are influenced by individual genes with a small or a major effect. Finally, scenarios when DNA technology is most useful for genetic improvement are discussed.

The first applications of DNA technology in pig breeding are presented by Moran (1999). The paper provides information about hyperpolymorphic DNA markers and their use for breed relationships and parentage testing. Next, the paper describes how linkage maps allow detection of quantitative trait loci (QTL) which might be useful for pig breeding through marker assisted selection (MAS).

Kerr *et al.* (1999) describe statistical methods which cope with the challenge of incorporating information about genes of moderate to large effect in breeding decisions. The paper also introduces the "new logistics and language" necessary to incorporate DNA technology into practical pig breeding.

In the final paper by Crump (1999) options for genetic improvement of disease resistance are explored. The two alternatives include genetic control of susceptibility to infection and genetic control of immune response. Opportunities for genetic improvement of disease resistance with and without molecular information are discussed.

NEW TECHNOLOGY TO ENHANCE GENETIC IMPROVEMENT OF PIGS

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Abstract

The rapid development of DNA technology is providing increased knowledge of the genetics of economically important traits and new tools with which pig breeders can improve them. Experiments mapping genes for quantitative traits show that there are many genes affecting such traits and their effects vary from very small to large. However there are no fundamental differences between what have previously been called polygenes, oligogenes, major genes and quantitative trait loci (QTL). Properties of quantitative traits such as selection response, heritability and genetic correlations are determined by frequencies of these genes, which in turn are controlled by mutation, migration, selection and drift. Some genes with a large effect on a quantitative trait are likely to be at a very low frequency prior to selection because they are recent mutations and/or because natural selection acts against them. These genes will contribute to the response to traditional selection, but might not be detected in experiments designed to find QTL. Many genes that play a role in the physiology of a trait under selection, will not contribute to the selection response because there are no significant mutants segregating in the population.

The results of DNA tests can be combined with phenotypic information to calculate more accurate estimated breeding values (EBVs). The increase in accuracy depends on the accuracy of the conventional EBVs based on phenotypes only, the amount of variation in profit explained by the DNA tests, and the overlap between the traits measured and the traits affected by the DNA tests. Pig breeders can decide to use a combination of measurement and DNA tests based on a single principle – maximize the accuracy of the EBV for profit while minimizing the cost of measurement and DNA testing.

In the future it is expected that there will be a change from markers in linkage equilibrium with QTL, to markers in linkage disequilibrium with the QTL and tests for the QTL itself. This will make DNA tests much easier to use. There will be a decrease in the cost of DNA tests and an increase in the number of tests, so that the benefit gained from using DNA tests increases and they are used as a screening tool before selecting pigs for conventional measurement.

Introduction

The basic steps in the design of a pig-breeding program include:

- defining the breeding objectives
- choosing a crossbreeding system and selecting the breeds or lines to be used within it
- selecting within these lines the best pigs as parents of the next generation.

It is the selection within lines that is the basis of on-going genetic improvement of pig production and the subject of this paper. New technology, which directly detects differences between animals in DNA sequence (the genetic code), is becoming available and will improve our ability to select the best pigs as parents. This paper describes the benefits to be had from the new technology and provides a background to the two following papers, which describe the technology in more detail. However, new technology will not change the breeding objectives and will add to, not replace, existing methods. Therefore, before discussing new methods for selection this paper will first consider the definition of breeding objectives and the selection methods already in use. Most traits of economic importance are quantitative traits so a brief description will be given of how knowledge gained from new DNA technology is improving understanding of the genetics of quantitative traits.

Breeding objectives

It will be assumed that the overall objective is to increase profitability in the commercial pig population that utilizes the lines under selection. However, profitability is a property of the whole production system not of individual pigs, yet it is individual pigs that must be selected or culled. Therefore, breeding objective must be defined in terms of traits that are properties of individual pigs. Breeding objectives commonly include traits such as growth rate, food conversion ratio, meat yield %, litter size and meat quality. They are the traits that directly determine income and costs. The breeding objective can be defined by a profit function, which takes the genetic value of these traits as input and predicts changes in profit as an output. The profit function does not attempt to predict actual profit, which depends on daily changes in market prices and management skill. Rather it predicts, for a fixed set of prices and management, the change in profit that would occur due to a genetic change in each trait (See Goddard, 1998 for a review of breeding objectives and profit functions). Often the profit function can be well approximated by a simple linear formula of the form:

$$\text{Profit} = a_1 * \text{trait}_1 + a_2 * \text{trait}_2 + \ldots\ldots + a_n * \text{trait}_n$$

In this formula a_1 (called the economic weight of trait one) is the amount by which profit increases if trait one is increased by one unit while all other traits in the equation are held constant.

The objective when selecting boars and gilts as parents is to select those whose progeny and later descendents will have the highest profit as defined in the equation above. That is, breeders wish to select animals with the highest breeding value for profit. They cannot directly observe an animal's breeding value so must estimate it from observable information. The best way to estimate breeding values has become an industry in its own right.

Estimation of breeding values

The most obvious source of information about an animal's breeding value is its own performance. For instance an animal's growth rate is a guide to its breeding value for growth rate, but it is an imperfect guide because it also depends on environmental effects. Programs that calculate EBVs attempt to allow for these environmental effects as much as possible, for instance by comparing the performance of a pig with that of his or her contemporaries. However, even when this is done, typically less than half the remaining variation in performance is due to variation in breeding value. An animal's performance or phenotype is still used to predict breeding value but the prediction is not 100% accurate. If the trait has a heritability $h^2 = 0.25$ or 25%, then the accuracy of the EBV, based on his own phenotype, is $h = 0.5$ or 50%.

The accuracy of a pig's EBV can be improved by using information on the performance of its relatives, because they have some of the same genes as it has. Progeny provide the most direct information on its breeding value and, with a very large number of progeny, the accuracy of the EBV approaches 100%. In some cases there is no observation of the pigs own performance and so prediction of his breeding value must rely on information from relatives. For instance, a boar does not express female traits such as litter size and his carcase characteristics cannot be observed while he is still alive.

Further information about a pig's breeding value can be obtained from its own performance and that of its relatives on correlated traits. For instance, food conversion ratio (FCR) is genetically correlated with the concentration in the blood of insulin-like growth factor 1 (IGF-1) (Bunter personal communication; Wuensch *et al.*, 1998). Thus measurement of IGF-1 concentration can increase the accuracy of EBVs for FCR, especially if FCR has not been measured itself.

A statistical analysis called a multi-trait BLUP combines all these sources of information to calculate an EBV which is as close to the true breeding value as possible.

Depending on the data that is available on a pig and its relatives, the accuracy of its EBV for a given trait will vary from near 0 to near 100%.

Once multi-trait EBVs for all the traits in the breeding objective have been calculated, an EBV for profit can be simply calculated from the formula:

$$EBV\ (profit) = a_1 * EBV_1 + a_2 * EBV_2 + \ldots\ldots + a_n * EBV_n$$

where EBV_n is the EBV for the n^{th} trait. Since the individual EBVs are not 100% accurate, neither is the EBV for profit, but it is the most accurate estimate that can be calculated from the available information. The EBV for profit is the logical criterion to use when selecting boars and sows.

When a breeder defines his breeding objective he is making a strategic decision based on his prediction of future market conditions. However, he should not ignore any traits that have an important effect on his herd's profitability. For instance, he is fooling only himself if he leaves FCR out of his objective because it is hard to measure. Whether it is measured or not, FCR has an important effect on profit. However, the breeder does not necessarily have to measure all traits in the breeding objective. In deciding which traits to measure he should aim to maximize the accuracy of the EBV for profit while minimizing the cost of measurement. Thus he might decide not to measure FCR (although it is in the objective), but to measure IGF-1 (although it is not in the objective) because IGF-1 is cheaper to measure and genetically correlated with FCR and hence with profit. Alternatively, he might decide to measure IGF-1 on all pigs, select the best and measure FCR only on them. The important principle is that definition of breeding objectives is based on predicted future market prices, while choice of traits to record is based on whether or not recording an extra trait will increase the accuracy of the EBV for profit enough to justify the costs.

A new source of information about a pig's genetic value

It seems obvious that when predicting a pig's genetic value one should consider information about the genes he carries. Unfortunately, until recently, the genes for most economically important traits could not be examined directly. The reasons for this are explained in the next section. However, the world is in the midst of a revolution in biotechnology, which is daily, or at least yearly, providing new tools with which to examine the genes an animal carries. These tools are based on detecting differences between animals in DNA sequence, which is the cause of all genetic differences. The challenge is to combine this new source of information with existing information so as to increase the accuracy with which genetic value can be predicted. Before tackling this challenge, it is necessary to describe how continuously variable or quantitative traits, such as growth rate, are controlled by individual genes.

Major genes and polygenes are all just genes

Many economically important traits vary continuously from low to high values and do not obviously follow Mendel's laws of inheritance. However, Fisher (1918) showed how many genes, each having a small effect on the trait, combined with environmental effects, could explain the continuous variation seen in quantitative traits. This is still the accepted explanation. The genes have been called polygenes or more recently quantitative trait loci (QTL). The characteristic of a polygene is that its effect is small relative to the remaining variation, so the genotype of an animal cannot be determined from its phenotype. Fortunately it is not necessary to know an animal's genotype at each gene affecting an important trait – it is sufficient to know the combined effect of all these genes on its breeding value. Therefore genetic improvement of quantitative traits has been carried out with great success despite almost complete ignorance of the genes responsible.

The distinct methodology of quantitative genetics, based on estimating breeding values from phenotypic data, has encouraged an artificial distinction between major genes and polygenes. Major genes are genes that have a big enough effect, relative to other

sources of variation, for the genotype of an animal to be determined from its phenotype and that of its relatives using a technique called segregation analysis. In reality there is a continuous variation in the size of effect of genes. Some genes have a big effect on a given trait, some genes a small effect and other genes a medium-sized effect. The same gene can have a big effect on one trait but a small effect on another trait. For instance, the ryanodine receptor or halothane gene has a big effect on meat quality, a medium sized effect on lean percentage and a very small effect on growth rate (Gueblez *et al.*, 1995)

Basically there is no difference between major genes and polygenes. In each case, a mutation causes a change in the DNA sequence that alters the function of the gene. This change in function may cause a large effect on a trait being studied or a small effect. Subsequently, there are two alleles of this gene present in the population – the original allele and the mutant – and the gene is said to be segregating in this population.

Quantitative genetics is just genetics

As new technology allows us to identify these mutations it becomes apparent that the genes for quantitative traits are just genes. It has long been known or believed that the phenomena of quantitative genetics are due to the properties of individual genes, but now this can be observed directly. For instance, the double muscling gene is one of the genes affecting eye muscle area in cattle. A mutation occurred in breeds such as the Belgium Blue in the myostatin gene whose normal function is to inhibit muscle growth (Georges *et al.*, 1998). The mutation inactivated the gene, leading to an increase in muscle growth. In nature this mutation is selected against because it causes an increase in calving difficulty and other problems. However, when selection for muscularity was applied in the Belgium Blue the frequency of this gene increased until it almost totally replaced the normal allele in this breed. All Belgium Blue cattle examined have the same mutation indicating that all the double muscle alleles trace back to a single original mutation. The same allele is also found in the Asturiana breed indicating that the double muscle gene arrived in the Asturiana by crossing with Belgium Blue or vice versa. However, some breeds have a different mutation at the myostatin gene indicating that it occurred independently of the mutation in Belgium Blue. The myostatin gene is not the only gene affecting muscularity in cattle. Even in breeds with only the normal version of the gene there is genetic variation in eye muscle area, presumably caused by variation at other genes.

In the pig there is almost certainly a gene for myostatin but we do not observe double muscling because the mutations seen in cattle either have not occurred or not been detected. This illustrates an important point: there must be appropriate variation in a gene before it can cause variation in the population. There may be hundreds of genes that play a physiological role in muscle growth, but in many cases none of the pigs in the population or even the species may carry a mutation which affects muscle growth. That is, the gene is not segregating in this population or breed.

A gene that does segregate in pigs is the ryanodine receptor gene and it also illustrates the phenomena of quantitative genetics. The normal N gene mutated only once to n so all the breeds except one which contain n genes acquired them by crossbreeding. The n gene causes an increase in pale, soft and exudative (PSE) meat and a decrease in survival under stress. Consequently, when this gene is segregating it will cause a genetic correlation between mortality under stress and PSE. Other genes, when segregating, may or may not cause both mortality under stress, and PSE. However, not all genes affect both traits: if they did the genetic correlation between mortality and PSE would be 1.0. Therefore, there must be genes with a different pattern of effects on these two traits. Consequently, when the n gene is not segregating in a line it is expected that the genetic correlation between PSE and mortality under stress will decrease.

These two examples have been chosen because the difference in DNA sequence between alleles is known. However, they are atypical examples in some regards. Most genes affecting quantitative traits do not have such big effects on the trait or on fitness. For instance, in Holstein cattle there is a gene close to the M blood group gene that affects milk yield. Selection for milk yield has been steadily decreasing the frequency of the M_2M' allele, which decreases yield, for the last 20 years at least, but the allele is still

present in the breed (Rocha *et al.*, 1998). In fact, gene-mapping experiments have found at least 10 chromosome regions containing genes that affect milk yield and composition. Of course there are more than 10 genes affecting milk yield, but many may have effects too small to be significant using current techniques. There is no evidence that all of these genes have large effects on fitness as the halothane and double muscling genes do. Nevertheless, it seems likely that some genes with a desirable effect on one trait will have an undesirable effect on fitness and this is most likely for alleles that have a large effect and that are initially rare.

Contemplation of these results leads to a paradox. If genes with moderate to large effects were segregating for traits such as milk yield, one would expect selection to have increased the frequency of the favourable allele until it approached 1.0. This would cause a reduction in genetic variance and heritability, but no reduction in heritability has been observed in practice. A possible explanation is that as selection is decreasing variation at some genes, it is increasing variation at other genes by increasing the frequency of initially rare alleles. If this is true, it must be anticipated that selection response in the medium term will come from genes that are initially rare and therefore are not identified in experiments designed to find genes for economic traits.

These examples illustrate how the frequency of genes for quantitative traits are controlled by the same forces as other genes – mutation, migration (e.g. by crossbreeding), selection (natural and artificial) and random drift. The experimental evidence is starting to provide a picture of the genetic architecture behind quantitative traits in terms of the number of genes, their effects, their allele frequencies and the selection coefficients to which they are exposed.

Finding genes for quantitative traits

Two broad approaches are used to find genes for quantitative traits. In the candidate gene approach, knowledge of the physiology of the trait is used to suggest (candidate) genes that might affect the trait. The strategy is then to look for differences between alleles in DNA sequence and then to determine if these differences are associated with differences in the trait. This approach has proved successful in some cases such as the effect of the oestrogen receptor gene on litter size (Rothschild *et al.*, 1996). However it is often unsuccessful, perhaps because many genes, although involved in the physiology of the trait, show no variation which greatly affects their function. When the candidate gene approach is successful it is very useful because it should identify the actual gene that affects the quantitative trait. This is not the case in the gene mapping approach.

In the gene mapping approach, genes for quantitative traits (i.e. QTL) are detected by linkage to genetic markers. If markers covering all the chromosomes are used, called a genome screen, genes for quantitative traits throughout the genome can be detected. Thus this method is less likely to be a complete failure than the candidate gene approach but it has two disadvantages. Firstly, large and costly experiments are necessary. Secondly, the outcome is that a gene for a trait of interest is known to map to a particular region of one chromosome but the identity of this gene is still not known. It is known that the gene is linked to one or more markers and these markers can be used for marker assisted selection, but this is not a simple matter if the marker(s) and the gene are in linkage equilibrium.

Linkage equilibrium and disequilibrium

Linkage equilibrium means that chromosomes, which carry the favourable allele at the QTL, do not all carry the same allele at the marker. In fact, across the population, the association between marker alleles and QTL alleles is random. Within the offspring of one boar, one marker allele will be consistently associated with the favourable QTL allele. This fact can be exploited by marker-assisted selection but only after determining for each family which marker allele is associated with the favourable QTL allele.

Linkage equilibrium is likely unless the marker and the QTL are very close together on the chromosome, i.e. very closely linked. Linkage disequilibrium means that the association between marker alleles and QTL alleles across the population is non-random.

If the linkage disequilibrium is complete, one marker allele is always on the same chromosome as the favourable QTL allele. In that case marker assisted selection is easy to apply and in practice it is almost as good as having identified the QTL itself.

Linkage disequilibrium is expected in livestock populations for three reasons. Firstly, effective populations' sizes are small. For instance, the effective population size in the Holstein breed is about 50. Secondly, some of the QTL mutations will have occurred within the last 100 generations and so will still be linked to the marker alleles that were on the chromosome in which the mutation occurred. Thirdly, crossbreeding causes linkage disequilibrium, but only to a small extent in crosses between outbred breeds, which do not differ greatly in gene frequency. Experiments in cattle are in fact finding linkage disequilibrium between markers and QTL up to 10 cM apart.

A logical strategy for mapping, using and identifying QTL is as follows:

- map QTL using a genome screen
- find markers in linkage disequilibrium with the QTL. This provides more useful markers for marker assisted selection and maps the QTL more precisely, which is beneficial in the next step
- search the pig gene map in the region to which the QTL has been mapped, and the homologous region of the human gene map, for possible candidate genes
- test these (positional) candidate genes to determine if they affect the trait

Using identified genes and markers

Long term selection response is based on the additive or average effect of genes. The additive effect of a gene is the average effect it has when combined randomly with other genes in the gene pool of the population. The breeding value of a pig for a certain trait is simply the sum of the additive effects of all the genes it contains. Thus selection is still based on finding the pigs with the highest breeding value for profit even if pig breeders possess knowledge about individual genes and can test for them. Non-additive effects of genes arise from dominance and epistasis. Utilisation of additive effects will be considered first and then utilisation of non-additive effects.

Utilizing additive effects of identified or mapped QTL

Consider the situation where one or more genes which affect profit have been identified and DNA based test for them exist. How useful will these DNA tests be? This question can be answered approximately by using conventional selection index methods. There are two sources of information – the conventional EBV that could have been calculated in the absence of the DNA tests and the prediction based on the DNA tests alone. The two most important parameters are the accuracy of the conventional EBV (r) and the proportion of genetic variance for profit explained by the DNA tests (d). The covariance between the two sources of information is also relevant. If the conventional EBV is based on phenotypic measurements of profit, this covariance is just r d. In this case the use of the DNA tests increases the reliability of the combined EBV by:

$$\frac{d}{1 - d\,r^2}\,(1 - r^2)^2$$

For instance, if the accuracy of the conventional EBV is 0.5, and the DNA tests explain 0.2 of the genetic variance, the reliability of the EBV is increased from 0.25 to 0.37. Therefore the accuracy of the EBV is increased from 0.5 to 0.61.

In some situations the conventional EBV will be based on measuring only a subset of the traits in the breeding objective. For instance, meat quality traits may not be measured. Assume that the DNA tests are for genes affecting traits that have not been measured (e.g. meat quality) and that the covariance between the DNA test and the measured traits is zero. Then the use of the DNA tests increases the reliability of the EBV for profit based on both measurements and DNA tests by d. Thus in the previous

example with r^2=0.25, d=0.2, the reliability increases from 0.25 to 0.45 and the accuracy increases from 0.5 to 0.67.

Other assumptions about the covariance between the DNA test and the conventional EBV lead to slightly different formulae, but many cases will lie somewhere in between the two situations given above (i.e., between the cases where the covariance is r d and where it is zero). These situations show how r and d determine the usefulness of the DNA tests.

The usefulness of the DNA tests is increased if r is low. This occurs if important traits cannot be measured on the pigs prior to selection. For instance, conventional EBVs of young pigs for litter size and carcase traits have a low accuracy and therefore, if these traits are important in the overall objective, the EBV for profit will have a low accuracy. The EBVs of poorly heritable traits also tend to have low accuracy, thus increasing the value of the DNA tests. If a recessive gene is at low frequency, phenotypes cannot distinguish carriers from normal homozygotes so the accuracy of assessing breeding value is low and a DNA test for carriers is beneficial. (Meuwissen and Goddard (1996) demonstrate these conclusions regarding traits with an EBV of low accuracy). It is the accuracy of the EBV at the time selection decisions could be taken which is important. For instance, after a progeny test for carcase traits, the EBV of a boar is highly accurate, but before the progeny test it is of low accuracy. Thus DNA tests which increase the accuracy before progeny testing would allow selection to take place earlier and hence reduce the generation interval. Similarly, the accuracy of a pig's EBV is low prior to performance testing, so DNA tests could be used to screen the pigs at this stage and decide which ones to performance test. In general, DNA tests are most useful when they can create a new opportunity for effective selection at a time when the conventional EBV has low accuracy.

The difference between the two situations given above (i.e., covariance between conventional EBVs and DNA tests equals r d or zero) shows that DNA tests will be more useful if they predict breeding value for traits which are uncorrelated with the traits measured.

Both situations show that the proportion of the variance explained by the DNA tests is a critical parameter. Note that this is the proportion of the genetic variance in profit not just one trait. This emphasizes the importance of the QTL effect on all traits in the breeding objective. In the formulae, it does not matter whether d is due to one gene and one DNA test or many genes and many DNA tests. The proportion of variance explained by the DNA tests (d) depends on the amount of variance explained by the QTL and the accuracy with which the effects of these QTL can be estimated. This accuracy depends mainly on the amount of data from which the QTL effects are estimated. For a marker, which is in linkage equilibrium with the QTL, the effects must be estimated on a within-family basis and therefore they will usually be estimated with limited accuracy and this, in turn, will limit d. Thus selection based on markers in linkage equilibrium with the QTL will be less useful than selection based on markers in linkage disequilibrium with the QTL or selection based on the QTL themselves (Henshall and Goddard, 1997).

Combining the phenotypic data and the DNA tests into a single overall EBV is the logical way to use both sources of information. Selection on this EBV will maximize gain in breeding value in the next generation. However it may not maximize long term gain in breeding value. This is because as selection increases the frequency of the favourable allele, it will eventually decrease the genetic variance caused by this gene. This occurs with conventional selection but it will occur more quickly when DNA tests are used that increase the selection pressure on some genes at the expense of others. Consequently, long term response can be increased by putting less emphasis on the marked QTL than indicated by the EBV (Dekkers and van Arendonk, 1998). However, the advantage gained by optimizing long term response may not be great. In many simulated selection programs, conventional selection never caught up with marker assisted selection (Henshall, personal communication). Also there are practical disadvantages of the optimum long-term strategy. By slowing down the rate at which the unfavourable allele is eliminated from the population, the time over which DNA testing must be continued is increased, and with it the cost. Also long-term response has less economic value than short-term response if the value of future income is discounted.

If a favourable allele is rare, selection for it will initially increase the genetic variance. Therefore the medium term response might be maximized by placing more weight on such a gene than indicated by the EBV. This is done in marker assisted introgression (Haley and Visscher, 1998) when a desirable gene is introduced into a line, which lacks it, by crossing with an otherwise poorer line that carries the gene. The carriers of the gene in each generation are backcrossed to the better line so that eventually the favourable allele is obtained on the genetic background of the better line. Unfortunately, considerable selection response can be sacrificed during the introgression process. Perhaps a method of maximizing the long-term response would be to view the problem as one of selection from the two lines and apply the method of Dekkers and van Arendonk (1998).

Utilizing non-additive effects of QTL

Non-additive gene action such as dominance and epistasis may be common, but it is hard to use. Currently, the main tools for using non-additive variance are crossbreeding and minimization of inbreeding. DNA tests could improve the use of non-additive effects. For instance, if genes show dominance so that genotype AA = Aa>aa and BB = Bb > bb, one parent of a cross could be selected for AA and the other for BB.

The callipyge gene in sheep shows a most unusual pattern of inheritance called polar overdominance (Crockett *et al.*, 1998). If a sheep inherits the callipyge allele from its sire and a normal allele from its dam, it displays increased muscling of the hind legs. However, if it inherits the callipyge allele from both parents, the sheep appears normal. It is hard to see how the callipyge gene could be fixed in a line without a DNA test for it. Recently, it has been found that the IGF-2 gene in pigs affects muscling, but only the allele inherited from the sire is active, while the maternally derived allele is turned off (Jeon *et al.*, 1999; Nezer *et al.*, 1999). This is called imprinting. Again a DNA test could be used to select for the high muscling allele in a sire line but against it in a dam line.

The cost of DNA tests

There will be a cost for obtaining a tissue sample from each pig, carrying out the DNA test in the laboratory and incorporating the information into EBVs. However, another major cost in some cases will be the cost of measuring additional traits and pigs. If the DNA test is a test for the QTL itself and if the pig breeder believes that the effects of the gene estimated in other pigs will apply in his herd, then he need do no additional recording. This has been the case with the test for the halothane gene: Most pig breeders simply used the test without checking that it worked in their own herd. However, to use a test for a marker in linkage equilibrium with the QTL, it is necessary to estimate the effects of the marker alleles within families so the pig breeder must measure the traits and DNA test pigs in his own herd. This could involve measuring traits not routinely recorded, or DNA testing and measuring extra pigs so that the marker effects can be estimated with sufficient accuracy. A test for a marker in linkage disequilibrium with the QTL presents an intermediate situation. The pig breeder might do at least some checking that the marker allele effects found elsewhere apply in his own herd.

Benefit-cost approach to DNA testing

Tests based on DNA typing represent an additional source of information about a pig's breeding value. Decisions about using DNA tests should be based on the same principle as decisions about which traits to record. That is, the increase in the accuracy of the EBV for profit should be compared against the increase in costs. Additional benefits, if any, from long term selection response or utilization of non-additive gene effects can be added to the benefits from a more accurate EBV.

As more DNA tests become available the benefits are almost cumulative because each test adds to the proportion of genetic variance in profit which is explained (d). However, the cost of multiple DNA tests should not be additive because many of the costs are the same no matter how many genes are tested. Therefore the benefit – cost balance

should become more positive. In the next few years the cost of DNA testing should drop, which will also make DNA testing more profitable. If the cost of DNA testing becomes low enough, it could be used to screen a large number of pigs and select which pigs to measure, especially for expensive traits such as FCR.

The author is currently carrying out a research project, funded by the PRDC, to determine how DNA tests can be used within pig breeding programs to maximize the benefits-costs from this new technology.

Alternative methods to use DNA tests

In some cases it may not be necessary to combine the DNA test results and phenotypic data into an EBV. For instance, most pig breeders used the halothane gene test to cull carriers and then selected within the non-carriers on conventional EBVs. This method is satisfactory if the gene has a large effect on profit but not on the traits for which EBVs are available. However, in all cases a benefit-cost analysis should be carried out. For instance, nearly all pigs are carriers of some deleterious recessive gene. However, if all these carriers were culled, nearly all conventional selection response would be sacrificed and the frequency of abnormal pigs, which is already low, would only drop slightly.

Conclusions

Tests based on DNA typing for genes affecting profitability are a new tool that is becoming available to pig breeders. They will be able to choose a combination of traits to record and DNA tests to use according to a common principle – increasing the accuracy of the EBV for profit while minimizing the recording and DNA testing costs. All sources of information can be combined into one EBV, which is a logical selection criterion. The benefit of adding DNA tests to phenotypic measurement depends mainly on two parameters, the accuracy of the existing EBV for profit and the proportion of genetic variance in profit explained by the DNA tests. DNA markers are most useful if they explain a lot of the genetic variance in profit especially through traits whose conventional EBV are lowly accurate, for instance, because the trait cannot be measured before selection.

It is likely that many genes and tests for them are necessary to explain a significant fraction of the variance in any trait let alone in profit. Hopefully the genes of largest effect will be discovered first, and as more genes are found the total proportion of variance explained will increase and so will the benefit from DNA tests.

Some useful genes will be at very low frequency in the population and we will sacrifice medium term response if we do not select effectively for them. In fact the most useful gene for which to have a DNA test, would be one that has a large effect and is rare. The ideal selection criterion would select pigs carrying such a gene somewhat more often than selection on EBV would do. This bias in favour of a 'rare' gene is also used during marker assisted introgression of a gene from another breed. However, rare genes with a large effect are the most likely category of gene to have a detrimental effect on fitness, so their effect on total profit must be estimated.

The DNA tests for markers in linkage equilibrium with a QTL are less useful than a test for the QTL itself because they are more costly to use (due to the need to DNA test and measure phenotypes on many pigs) and less accurate (because the marker effects have to be estimated within family).

In the future it is likely that the cost of DNA tests will decrease and may become cheaper than phenotypic measurement and recording, especially expensive measurements such as FCR. In that case the DNA tests could be used to screen a large population from which the better pigs are selected for a conventional phenotypic test. This could reduce testing costs and/or increase the effective size of the nucleus herd thus increasing rate of genetic gain.

Symposium continued on next page

MARKING THE WAY TO BETTER PIG BREEDING

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Abstract

This review follows the trail from hypervariable DNA markers, through applications like parentage testing, analysis of breed relationships and gene mapping, towards the eventual goal of better pig breeding. A central focus of the review is the mapping of quantitative trait loci (QTL), but with a prospective emphasis on the identification and exploitation of the underlying genes ultimately responsible for the QTL effects. The review concludes with a brief look at developments in the automated technology for rapidly genotyping large numbers of animals for large numbers of genes responsible for inherited variation in performance and productivity.

Introduction

Currently DNA marker technology is a very valuable adjunct to conventional animal breeding, enabling parentage verification and the routine identification of carriers for some genetic disorders. However, the scope for this molecular technology is much broader and of far greater economic consequence than these current applications. This paper will review current and potential uses of DNA marker technology in pig improvement. The background research and knowledge base necessary for these applications will also be reviewed.

The fundamental premise of this work is that all genetically determined variation in productivity or economic value of animals is ultimately encoded in their DNA. If all the DNA sequence variants responsible for these differences in performance could only be identified, then superior animals to use as parents for the next generation could be chosen simply by doing DNA tests. Alternatively DNA markers tightly linked to and associated with these causal variants could be used as proxies, if the causal genes had not been identified. In either case, if the DNA variation has previously been correlated with variation in an economically important trait, such as growth or meat quality or susceptibility to disease, it can be used to assist or accelerate the choice or rejection of animals as parents. This might merely slightly speed up the rate of improvement for traits like backfat thickness, where measurement of performance is cheap and easy, and conventional selection is efficient. More usefully it might also enable early decisions on the choice of parents for intractable but important traits, only measurable post-slaughter, like meat quality, or only expressed in one sex, like ovulation rate. The use of DNA markers in this way has been termed marker-assisted selection (MAS). The potential of MAS has provided the economic and scientific justification for gene mapping and genomic studies in pigs and other domestic animals, as detailed maps are prerequisites for the identification of the chromosomal regions or the very genes responsible for variation in the traits. Of course all this presupposes that DNA samples can be obtained from candidate breeding stock and marker tests can be applied at a reasonable cost. New automated and miniaturised technologies are now being developed, especially the so-called gene chip technology, which will enable routine and very cheap testing of thousands of genes simultaneously, providing optimism that this will be so.

Figure 1 summarises the purposes and structure of this review. The central objective addressed is development of "better breeding programs". The review focuses on the DNA marker technology enabling the construction of genetic maps and the identification of genes and chromosomal regions responsible for variation in economic traits and ultimately to their application in better breeding programs. The highly polymorphic DNA markers shown at the top of the figure provide an historical starting point, as the markers have been fundamental to map development and have other immediate applications important to pig breeding. Single nucleotide polymorphisms (SNPs) (see bottom of Figure 1), which are much less variable but very much more

common (estimated to be found 0.5 to 10 times per 1000 base pairs), are an alternative to the highly polymorphic markers. The main advantage of SNPs is that they can be detected using extremely rapid and automated assay systems (Gilles *et al.*, 1999), like gene chips, which are currently undergoing a rapid phase of development and improvement. The majority of causal DNA sequence variants, which are the ultimate tool of MAS, are nucleotide substitutions with some small deletions or insertions as well. Importantly, all will be detectable using the gene chip technology developed for SNPs. Few causal DNA sequence variants are currently available to pig breeders but many more will become available in the future, critically depending on maps developed with highly polymorphic markers.

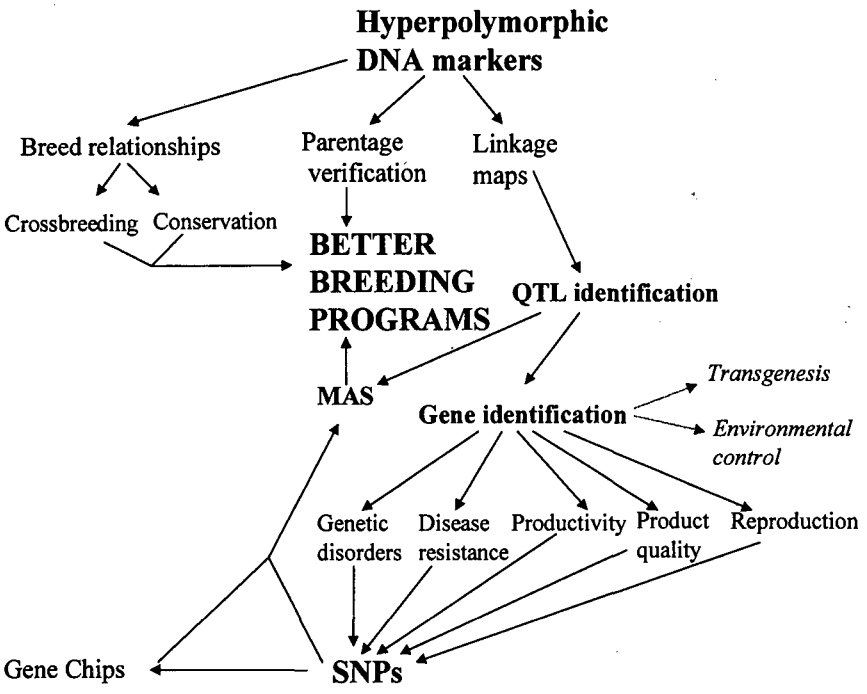

Figure 1. *Overview of applications of DNA marker technology in pig breeding (QTL, quantitative trait locus; MAS, marker-assisted selection; SNP, single nucleotide polymorphism). Better transgenesis and environmental control of gene expression are other outcomes potentially arising from identification and characterisation of genes influencing economically important traits but are not discussed in this review.*

Hyperpolymorphic DNA markers

Microsatellites, which are tandem repeats of short DNA sequences, have become the most widely used, highly variable DNA marker in mammalian genetics in the 10 years since their first use (Weber and May, 1989; Tautz, 1989). The repeats range from single nucleotide e.g., $(A)_n$ up to four or even more nucleotides in length, but the most commonly exploited microsatellite in mammalian genetics is the dinucleotide $(AC)_n$ repeat. In the pig, there are estimated to be 60-100,000 locations of the AC repeat randomly distributed throughout the genome (Wintero *et al.*, 1992), so this class of repeat alone provides a surfeit of genetic markers. Importantly, microsatellites are highly variable in repeat length, but obey the normal laws of Mendelian segregation. At any particular site in the genome, animals will have different repeat numbers, with many different repeat-length alleles likely to be present in a population. As a result, there is a greater than 60-70% chance that for a particular locus any animal in a population will be

heterozygous. The heterozygosity enables geneticists to track the transmission of alternative chromosomal segments from parents to progeny. If one of those segments is associated with a positive effect on a trait and the other with a negative effect, one can infer the existence of a quantitative trait locus within the segment. Heterozygosity is equivalent to informativeness for gene mappers. The more variable a marker, the more informative it is. Because of this high informativeness, microsatellites have been a boon to human and animal genetics, being critical to the charting of detailed genetic maps and to the identification of genes and regions responsible for quantitative variation. Most microsatellite markers are classified as anonymous in that their location in relation to specific genes is unknown and they simply serve as flags for particular regions within maps. However a proportion of microsatellites have been found closely adjacent to or even within genes of known function (see for example Moran, 1993).

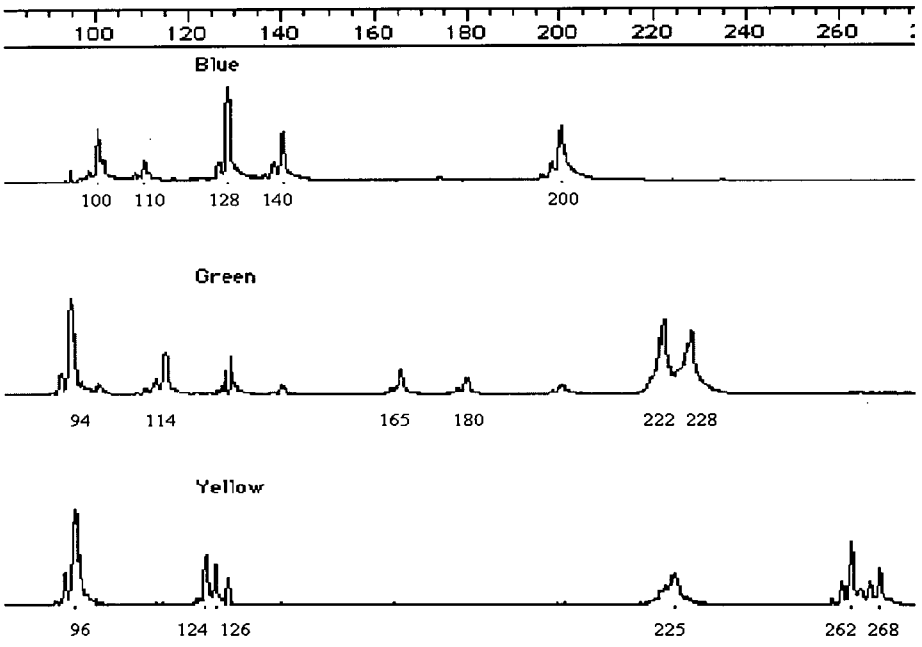

Figure 2. *Scan of typical dye-label gel for a pig genotyped for 10 microsatellites. The blue, green and yellow parts of the scan are shown separately for convenience of presentation in black and white. The scale in base pairs is calibrated with respect to in-lane molecular weight standards labelled with red fluorescent dye. The genotypes of this boar from the Bunge resource family are (Blue): Sw72 100/110, Sw905 128/140, S0206 200/200; (Green): Sw240 94/114, S0087 165/180, S0228 222/228; (Yellow): Sw707 96, Sw951 124/126, S0141 225/225, S0010 262/268. Sw707 is X-linked. The highest blue peaks can be seen as shadows in the green scan.*

After amplification in the polymerase chain reaction (PCR), microsatellite variants are identified as bands of different sizes separated in high-resolution gel electrophoresis. While originally visualised by autoradiography, microsatellites are now detected by dye labelling on automated DNA sequencers. In the Applied Biosystems Incorporated (ABI) system, four dye colours are available. The red colour is reserved for the size standard, which permits the accurate measurement of the size of microsatellite alleles down to single nucleotide resolution. Three other colours; blue, green and yellow, are available for labelling the microsatellites during PCR amplification. Figure 2 illustrates a typical gel trace for ten microsatellite loci simultaneously analysed in one animal. Depending on the apparatus, 36 to more than 90 animals can be analysed on a single gel. Software is

available for automatically interpreting the output from such machines and storing the data for subsequent analysis. In gene mapping work, automatic systems are used to log and assist in interpreting data from 100 or more loci from 500 to 1,000 or more animals, as manually keeping track of the information becomes almost impossible.

Breed relationships

The conservation of genetic resources is an increasingly important issue to animal breeders and the general community alike. Concentration on a narrow spectrum of high performance breeds has led to the abandonment of lower performing breeds at an alarming rate. Molecular marker technology is now allowing measurement of the diversity within and among breeds. This permits the development of action plans hopefully assisting in the conservation of the greatest level of diversity for the least cost.

The Measurement of Domestic Animal Diversity (MoDAD) program of the Food and Agriculture Organisation (FAO) has the objective of determining breed relationships as well as assessing intra-breed variability for all major species of domestic animals. For the pig, both the International Society of Animal Genetics (ISAG) and the European Pig Gene Mapping Program (PiGMaP) consortium have assisted by developing a standard panel of microsatellite markers to be genotyped on samples of all breeds. There are currently 27 microsatellites in this panel but at the ISAG meeting in Auckland in 1998, it was decided to increase this number to 55. Ideally a sample of 25 unrelated males and 25 unrelated females should be genotyped for each breed, although for rare breeds this is impossible.

During 1997, Professor Kui Li, from Huazhong Agricultural University in China, genotyped these 27 microsatellites on samples of eight Chinese breeds while working at the University of Sydney (Li *et al.*, 1998). The samples from some of the breeds were very small and the results are accordingly unreliable for those breeds, so only four breeds are included in the comparison with Australian commercial pigs shown in Figure 3. Chinese pigs are traditionally classified into six types, mainly based on geographical origin with some recognisable elements of body conformation and colour. Three of the Chinese breeds in Figure 3 are classified as Central China type but are split in this dendrogram by the Erhualian breed, a River Sea type. Testing the validity of the traditional breed type classification and resolving the relationships among breeds will be aided by additional microsatellite markers and possibly by mitochondrial DNA sequence. Professor Kyu Il Kim, an Australian Research Council Visiting Professor at the University of Sydney, is currently using mitochondrial D-loop sequence to resolve the relationships of these breeds and other Asian breeds of pigs.

Some studies have already used microsatellites for analysing breed relationships in pigs outside of the MoDAD program. Paszek *et al.* (1998) have estimated genetic distances and times of breed divergence for one Chinese and four European breeds, using allele frequency data for nine microsatellites linked on chromosome six. The European breeds, as expected, clustered in a separate branch of the phylogeny from the Chinese Meishan. The Yorkshire and Hampshire breeds were found to be the most closely related of the European breeds analysed, with a calculated divergence time of 391 years. Meishan, representing Asian stocks, were estimated to have separated from the European breeds about 2,200 years ago. These estimates of breed divergence are consistent with historical records. However, it should be stressed that estimates of breed separation times depend on assumptions that no mixing or interbreeding have occurred and this cannot be confidently assumed even between European and Chinese breeds, since there are records of introduction of Asian pigs into Europe during the 17th and 18th centuries.

Breed relationship (phylogeny) information can be used in various ways. For example, if a rare and endangered breed is closely related to a common breed, then it may be logical to abandon the rare breed or merge it with the common breed. If there are several rare breeds shown to be closely related, it may be most efficient to merge them and manage them as a single population. On the other hand, if there is a rare breed very different from any other breed, then conservation resources should target that breed. Breed relationship data also have potential value in crossbreeding. Pig producers are frequently searching for novel breeds to cross with established breeds like Large White

and Landrace. The more distantly related such breeds are, the better will be the outcome in terms of the expected heterosis for production and reproduction-related traits.

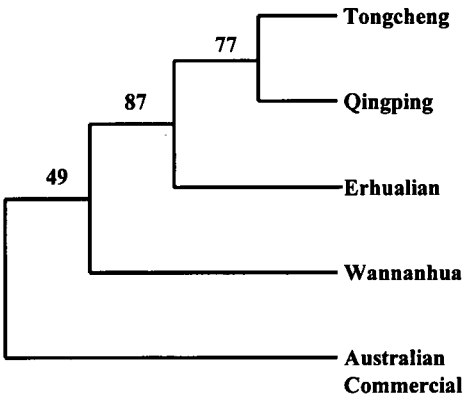

Figure 3. *Consensus dendrogram derived from a matrix of Nei's genetic distance among breeds calculated from allele frequency data for 27 microsatellite loci. The consensus is the result of 5000 bootstrap re-samplings of the data with the percent bootstrap support for nodes in the phylogeny shown to the left of the relevant node. The Erhualian breed is a River-Sea (Lower Changjiang Basin) type, whereas the other three Chinese breeds are Central China types. The Australian commercial pigs are of mixed Large White and Landrace origin.*

Parentage testing

The ability to verify the parentage of animals is vitally important in animal breeding. Now at last, DNA technology has provided a system to do this with great accuracy. With costs of parentage testing decreasing and the efficiency of detection of errors now very close to 100%, there may be a greater place for routine parentage testing in pig breeding. It is important to detect and correct parentage errors to get the full benefit of packages like PigBLUP, where performance information from relatives is vitally important in estimating breeding values.

Even in the best run breeding system (and in humans), a pedigree error rate of about 10% or more is likely. This usually involves incorrect identification of sires. However, recording errors can result in misidentification of dams and in some cases even of both parents. For example, if a piglet were to change pens before it was earmarked, then the identity of both parents would be incorrectly recorded. In the past, blood group variation was used as a rather inefficient tool to detect pedigree inconsistencies. Microsatellites allow virtually certain recognition of pedigree errors and, in many cases, actually allow retrospective identification of the correct parents. With as few as 10 highly variable microsatellite markers, it is possible to recognise all pedigree errors. The cost of this type of pedigree checking is decreasing. In the future, it is probable that it will be routinely applied even in pig breeding, in addition to sheep and beef cattle breeding, where there is strong demand due to the difficulty of supervising or otherwise monitoring matings.

Porcine microsatellites have been evaluated for parentage testing in projects in the Department of Animal Science at the University of Sydney. They have been useful in detecting pedigree errors, both in gene mapping resource families and in a collaborative project with a transplantation research group at Westmead Hospital to make an inbred line of pigs. For example, at one stage in the inbreeding program, a gilt was kept in a pen with two of her supposedly sexually immature littermate brothers. A deliberate mating was set up with one of these males, but it was suspected that the other male had also mated with her. Microsatellites proved that this was so and each of the piglets from her first litter was assigned unambiguously to the respective sires based on inheritance of microsatellite alleles.

Linkage mapping

Pigs have 19 pairs of chromosomes. With the development of better staining techniques for mammalian chromosomes in the 1970s, each chromosome could be reliably identified, although the standard karyotype for the pig was defined by Gustavsson only as recently as 1988. The standardised karyotype can be converted into an outline physical map, namely an ideogram, showing landmark bands. Useful features can be added to this outline map in several quite different ways, namely physical mapping and linkage mapping. The end result of both will be ordered positions of markers on chromosome maps. However, the relative spacings between markers will differ between the two types of maps, since in linkage maps, probability of recombination rather than physical distance determines the spacing.

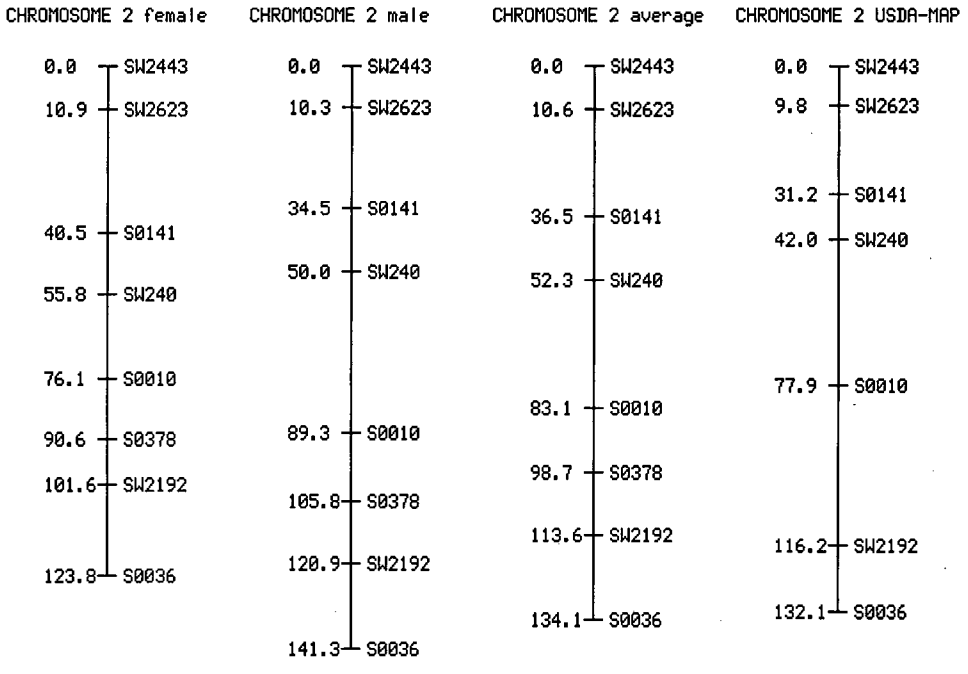

Figure 4. *Linkage maps of chromosome 2, derived at the University of Sydney from a two-generation commercial pedigree bred at Bunge Meat Industries. These agree well in order and spacing with the United States Department of Agriculture (USDA) map. Since recombination frequency between markers differs between males and females, separate University of Sydney maps are shown for females, then males. The average of these male and female maps can be compared with the USDA map, an average map but with some bias towards the male map. All markers are PiGMaP (S0... series) or USDA (SW... series) microsatellites. Map distances are in centiMorgans (cM), an adjusted measure of recombination frequency, from the endmost marker SW2443, arbitrarily scaled to position 0 in these maps.*

To make a linkage map, it is necessary to have families (two or preferably three-generation pedigrees), in which to detect the products of crossing-over during meiosis (the process leading to formation of eggs or sperm). Crossing-over generates recombinant chromosomes detectable in the progeny. Genes close together on a chromosome are unlikely to have a crossover between them. This means that alleles at two close loci will stay together (co-segregate). Loci far apart are very likely to have a crossover between them and the alleles at the two loci will be mixed into new combinations as well as the original combinations. By working out the proportion of recombinants in a group of progeny, the recombination frequency can be determined and this will then allow ordering of the genes along the chromosome.

By systematically looking at pairwise combinations of genes, an unmapped gene will be found tightly linked to some markers and unlinked to others and thus can be assigned to a linkage group on a chromosome. Using multipoint mapping, the order of genes within a linkage group can be determined. In some cases, there is insufficient information available to unambiguously order all genes on a chromosome and it would be uneconomic to breed pedigrees large enough to obtain the necessary information. Only the best guess order is available for genes close together. Very close genes and markers will be boxed together in maps to indicate that their order cannot be determined. Figure 4 shows a map for chromosome two produced from a two-generation pedigree of commercial pigs; bred at Bunge Meat Industries as part of PRDC project US36. For comparison, the position of the same markers on the USDA map is also shown. Since the map reflects the probability of crossing-over between the markers, there is not an exact correspondence between the maps, due to sampling and also to some biological differences in recombination frequency between animals. However, there is quite a good correspondence overall. This map also illustrates the substantial differences in recombination rate and position typically found between males and females, reflecting the differences in the distribution of crossovers between the sexes.

Comprehensive linkage maps have been produced by the European PiGMaP consortium (Archibald *et al.*, 1995), with which the University of Sydney laboratory is affiliated, and the United States Department of Agriculture (Rohrer *et al.*, 1996). As a result, the linkage map of the pig now has several thousand highly informative markers on it, and plans are afoot to add several thousand more (C. Beattie, personal communication). By comparison, the human and mouse linkage maps have over 6,000 markers each and are effectively saturated with markers, in the sense that any new marker will map to exactly the same position as an existing marker.

QTL mapping

Detailed linkage maps and highly polymorphic markers permit searching for the genes responsible for the variation in performance between animals in any pedigree where DNA and performance records have been collected. Although QTL studies in pigs are still in their infancy, there is promise in the early findings. Here some early results from a collaborative project with colleagues at the University of Hohenheim in Germany will be briefly reviewed to illustrate the benefits and the limitations of QTL mapping. Mr Seung Soo Lee performed the genotyping described here as part of his PhD project, and QTL analyses were carried out by Dr Gerhard Moser during his tenure of a PRDC Distinguished Visitor Award at the University of Sydney in 1998. The German group has bred a three-generation pedigree, consisting of all pairwise combinations of Pietrain, Wild Boar and Meishan, with over 300 F2 progeny being produced from each of the three crosses. The F2 progeny have been measured for 43 performance traits. This huge and valuable resource has been made available to international collaborators and the University of Sydney laboratory agreed to genotype the pedigree for markers on chromosomes two and five. For the Pietrain by Meishan pedigree, four microsatellites, genotyped by Mr Lee and the *Myogenin D1* (*MyoD1*) locus, genotyped by a Czech group, have provided sufficient data for a preliminary scan of about half of chromosome two (Lee S.S. *et al.*, 1998a,b).

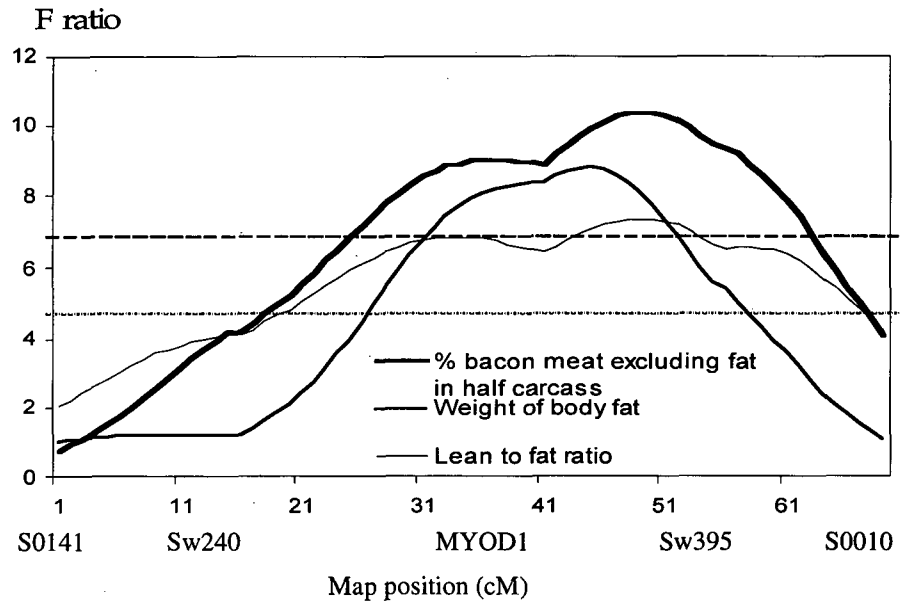

Figure 5. *Scan of part of porcine chromosome two for fatness and body composition QTL using the F2 of a cross between German Pietrain and Chinese Meishan pigs. Scale on the X axis is in centiMorgans (cM), a mathematically transformed measure of recombination frequency, with the positions of the markers indicated below the scale. The F ratio test statistic is shown on the Y axis, with the curves showing variation in this statistic with position across the chromosome for three traits. Statistical significance thresholds for QTL detection are shown as horizontal lines (upper dashed line is the stringent genome-wide threshold, lower dotted line is the less stringent chromosome-wide threshold). For all three traits, the test statistic exceeds the more stringent threshold and thus there is good evidence for existence of QTL.*

Scanning for QTL involves calculating test statistics at points across a chromosome in the vicinity of markers and determining whether this statistic exceeds a significance threshold, whose choice involves difficult statistical and philosophical issues not discussed here. This information is most simply presented as a plot along the chromosome map. The highest point in the scan exceeding the significance threshold is taken to be the most likely position of the QTL. The choice of the very stringent genome-wide threshold or a less stringent chromosome-wide threshold provides respectively greater reliability of the existence of detected QTL or less chance of missing real QTL, but at the expense of more artefacts. This preliminary scan detected 12 potential QTL from 44 traits measured, half of which exceeded the stringent genome-wide significance test. Eight QTL relate to measures of fatness or leanness and the nature of the traits and the position of the QTL suggest they may reflect pleiotropic effects of the same underlying gene(s). Three other traits, teat number, liver weight and early growth are unrelated to fat deposition or to each other and their QTL appear to map to different positions from the fatness QTL.

Figure 5 illustrates a chronic problem of QTL mapping, which has troubled gene mappers in all species. Unfortunately QTL are not accurately mapped in standard breeding designs involving two or three generation pedigrees. Although there are obvious peaks for these three traits, there is a quite broad region of the chromosome in which each QTL could lie. For example, for percentage of bacon meat excluding fat in a half carcase, the test statistic exceeds the genome wide significance threshold from about 25 to 63 cM, covering 54% of the map. Breeding larger F2 families or adding more markers to the map does not provide substantial improvements in map accuracy, as there

is a rapidly diminishing return on genotyping effort. The Swedish group, which discovered the QTL for backfat on chromosome four in the pig (Andersson *et al.*, 1994), is breeding deeper pedigrees, extending for five to six generations. This approach permits a gradual paring away at the edges of the QTL map and a refinement of QTL position (L. Andersson, personal communication). Such improved accuracy will make the QTL more amenable to exploitation by MAS or to positional cloning.

Most QTL studies in the pig, such as that illustrated in Figure 5 or the pioneering study of Andersson *et al.* (1994) have used wide crosses, involving an economically relevant breed, like Large White or Pietrain, and a distantly related economically irrelevant breed like Wild Boar or Meishan. There has always been a question about the relevance of any QTL found in those studies and a concern that they might give an inflated impression of the frequency and effects of QTL in commercial populations of pigs. In Australia, a QTL scan has been performed on a commercial population of pigs bred and performance tested at Bunge Meat Industries in Corowa. Eighteen performance traits were measured on four large sire families. With approximately 70% of the genome covered by the scan, 37 QTL were detected, 19 at a relatively stringent significance criteria and 18 at less stringent criteria (Table 1). The number of QTL, the magnitude of their effects and the reliability of their detection provides substantial optimism that these QTL can make a useful contribution in practical breeding programs in Australia.

Table 1. Estimated effects of putative QTL (±SE) affecting growth, meat quality and carcase traits detected in four sires from an Australian commercial population (PRDC project US36). In this study, QTL were detected by single trait mapping using a). more stringent Lander and Kruglyak (1995) thresholds, with additional putative QTL found with b). less stringent empirical thresholds (Churchill and Doerge, 1994).

a). Trait*	Boar	Effect of allele substitution, δ	b). Trait*	Boar	Effect of allele substitution, δ
ADG1	1	60 (15) gm	ADG2	2	217 (52) gm
ADG3	4	104 (30) gm	ADG2	2	140 (54) gm
FD3/4	1	1.43 (0.36) mm	ADG2	1	138 (44) gm
FDP2	3	1.85 (0.55) mm	ADG3	3	63 (19) gm
HCP2	1	1.73 (0.48) mm	ADG3	3	77.8 (24) gm
HCP2	1	1.86 (0.46) mm	ADG3	4	75 (27) gm
HAM	1	-0.44 (0.14) kg	DFDINT	2	0.26 (0.13)
HAM	2	-0.43 (0.13) kg	FDP2	2	1.26 (0.41) mm
HAM	4	1.39 (0.57) kg	HCP2	4	-3.67 (1.0) mm
HAM	4	1.42 (0.75) kg	HCP2	2	1.45 (0.77) mm
HAM	3	1.02 (0.17) kg	MD3/4	2	3.75 (1.02) mm
HAM	4	1.43 (0.25) kg	LW	4	1.08 (0.52) kg
LW	4	1.84 (0.53) kg	IMF	4	0.93 (0.63)
LW	3	1.56 (0.30) kg	CLD	2	6.2 (2.03)
LW	4	1.62 (0.46) kg	CSP	1	2.33 (1.29)
IMF	4	0.94 (0.27)	PH24	1	-0.10 (0.03)
CLD	1	3.46 (0.87)	PH45	3	0.32 (0.16)
PH24	2	0.13 (0.04)	PH45	3	0.30 (0.12)
PH45	3	0.34 (0.12)			

*ADG 1, 2 and 3, average daily gain from 3 to 18 weeks, from 18 to 22 weeks and lifetime respectively; DFDINT, daily feed intake 18-22 weeks; FD3/4, MD3/4, FDP2 and HCP2, fat or muscle depth at 3 and 4th last ribs or P2 position measured with real time ultrasound or Hennesy Chong probe; HAM, LW, weight of back left leg slash boned or bone in; IMF, intramuscular fat percentage; CLD, CSP, colour of *longissimus dorsi* or *superior spiralis* muscle; PH45, PH24, muscle pH 45 minutes or 24 hours after slaughter.

From Quantitative Trait Locus to Marker Assisted Selection

Any QTL identified in a genome scan is potentially exploitable in pig breeding by marker assisted selection (MAS). The linked markers identifying the QTL in the first place can be used to track inheritance of the relevant chromosomal region in breeding stock. The difficulty with linked markers is that the same marker alleles are not going to be associated with the same QTL alleles in different families. Further, even within a family, the marker/QTL associations can be changed by recombination, if the marker and QTL are not very closely linked. Both problems create difficulties but they are not insurmountable. Given that discovery of the gene(s) responsible for a QTL effect may be a long time coming and involve substantial research costs, it will almost certainly be economically worthwhile to use linked markers to make incremental improvements in efficiency of breeding programs now. Essentially marker genotypes will have to be gathered along with performance data to continually re-evaluate the information provided by the marker in particular families.

This is obviously a very complex procedure and requires thorough evaluation before it is used routinely. Professor Mike Goddard is now using computer simulation to evaluate the feasibility and economic benefit of MAS for pigs in a new PRDC project, US43. Using the empirical data on QTL positions relative to markers and QTL effects obtained from the US36 project, as well as relevant estimates of genetic and phenotypic parameters from Australian pigs, the benefits of MAS will be weighed against the costs of genotyping.

Gene Identification

Identification of the genes directly responsible for QTL effects is desirable. Obviously MAS will be more accurate if the gene causally responsible for the performance variation is used. Furthermore, in the absence of genotype-environment interaction, it will be possible to employ MAS in any family without the need for continuous evaluation of the association between trait and marker. So how are such genes identified?

As mentioned previously, the position of a QTL is generally mapped quite imprecisely. This means that any attempt to clone the underlying gene, based on the mapped position of the QTL, using a so-called positional cloning strategy, is fraught with difficulty. The starting position for the search is so imprecisely defined and the number of clones requiring evaluation is so enormous that positional cloning is not feasible. Even if the QTL could be very accurately mapped to within 1 cM, this would still leave approximately one megabase pairs (one million base pairs) of DNA to sift through in search of the gene. While this may be within the budget of some medical research groups, it is not economically feasible for animal researchers. Are there any shortcuts to the gene involved?

Candidate genes have been suggested to provide such a shortcut. These genes function in physiological, metabolic or regulatory processes, which suggest that their allelic variants could cause variation in a phenotype related to that process. For example, sequence variants of a gene involved in fat metabolism, like that encoding for lipase, might be tested for effects on fat deposition. The problem with this approach is the enormous number of candidates, even among currently known genes. This means that the candidate approach is more likely to result in a wild goose chase than to lead to the locus responsible for the performance variation of interest. To make matters worse, the identity and function of only about 15% or less of all genes in mammals is currently known. When the human genome is fully sequenced, conservatively in less than 5 years time, optimistically by the year 2000, the 70,000 to 100,000 genes found in the human genome will amplify this surfeit of candidates for explaining inherited quantitative variation in all mammals and indeed all vertebrates.

Fortunately, the recognition of this enormous number of candidates from whole-genome sequencing will also permit an enormous simultaneous refinement of the candidate approach. The precise order and position of all genes, including the currently unrecognised ones, will automatically follow from the genome sequencing. Even if a

human QTL is only inaccurately mapped to a particular chromosomal interval, this information will enable focussing on a subset of candidates located in that interval, excluding the vast majority of genes lying outside. Such genes are known as positional candidates. Each positional candidate can be evaluated and its variants tested for effects on the trait of interest. Total genome sequencing is neither feasible nor necessary in animals. Fortunately, very good comparative maps have been made between the human and most domestic animal genomes. The comparative map for pig and human (Goureau *et al.*, 1996) is the best for any domestic species and is still being refined. If a porcine QTL is detected within a particular chromosomal region, it will be possible to focus on candidates in the corresponding chromosomal region in humans. Such candidates are known as comparative positional candidates. Again there may be a large amount of effort to sift through even this refined list of loci, particularly if the original QTL effect was not particularly well mapped. However, there has been a recent spectacular success of the positional candidate approach in pigs, with two laboratories independently identifying the locus responsible for a large QTL effect on muscle mass and fat deposition located on pig chromosome two (Jeon *et al.*, 1999; Nezer *et al.*, 1999).

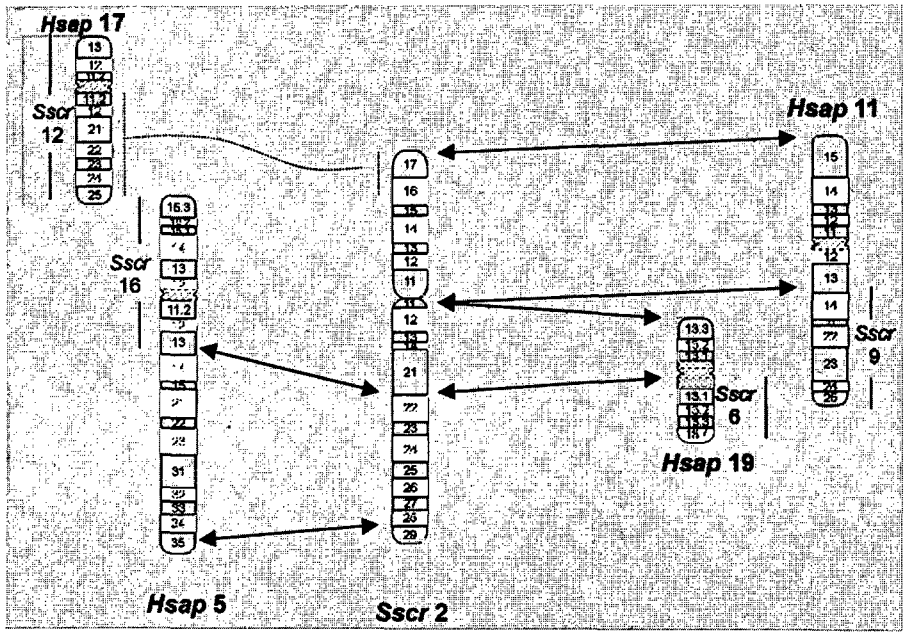

Figure 6. *Comparative map of pig chromosome 2 (Sscr2) showing correspondence to regions of human chromosomes (Hsap) 11, 19 and five. These corresponding regions are delineated with double-headed arrows. Although it could not be seen from the comparative fluorescent in situ hybridisation studies, part of Sscr2 may also correspond to part of Hsap17, since an homologous gene has been mapped to the corresponding region in pig and human. Similar comparative maps have been derived for all pig and human chromosomes. (Modified from http://www.toulouse.inra.fr/lgc/pig/compare/SSCHTML/SSC2S.HTM)*

Figure 6 demonstrates part of the human/pig comparative map, with full details for all other chromosomes available from the Institut National de la Recherche Agronomique (INRA) Web site in Toulouse, France (http://www.toulouse.inra.fr/lgc/pig/compare.htm). This comprehensive but relatively low resolution comparative map has been generated mainly using fluorescent in situ hybridisation (FISH). Chromosome-specific libraries have been made for all human and all porcine chromosomes. The individual libraries have been labelled with a fluorescent tag, hybridised to metaphase chromosome preparations from pig or human and the resulting hybridisation mapped to individual chromosomes. Since this process uses probes from one species on

chromosomes from another, it is called ZOO-FISH. The resolution of the comparative map can be increased by mapping individual genes, recognisably homologous in both species. The use of comparative anchor tagged sequence (CATS) primers represents one systematic attempt to do this, using PCR primers based on a consensus of published sequences from as many species as possible. At the University of Sydney, this approach has been applied to genes from human chromosomes nine, 10, 20 and 22, with 10 genes mapping to positions in the pig genome consistent with ZOO-FISH and other results (Lee J.H. *et al.*, 1998a,b).

Genetic disorders

The first example of the systematic use of DNA genotyping to control genetic disease in animals is provided by the so-called *halothane (hal)* mutation in pigs. In this disease, unlike many recessive disorders, homozygotes can survive and reproduce. Originally homozygotes could only be recognised by an abnormal reaction to halothane anaesthesia, showing muscle stiffening and an abnormal and frequently fatal increase in body temperature, also inducible by stress. Carriers could only be recognised by producing an affected offspring. The commercial significance of the disorder lay in the fact that homozygotes had severely compromised meat quality. The pale, soft and exudative (PSE) pork is unacceptable to consumers of fresh meat and has unfavourable properties for processing. The mutant allele reached quite high frequencies in many breeds, because of its favourable pleiotropic effect on backfat thickness. Because of intense interest in this mutation and its effects, the first small genetic region mapped in the pig surrounded this locus, long before any useful map existed for any other part of the genome. Even this rudimentary map, consisting of antiquated isozyme and blood group markers, played an important role in directing the cloning effort ultimately leading to recognition of the mutation, if nothing else demonstrating the value of map data in modern genetics.

Fujii *et al.* (1991) in Canada won the race to clone the gene and identified the single nucleotide mutation responsible for the entire syndrome of effects. With payment of substantial royalties to these Canadian discoverers and also to the company holding the rights to the PCR process, detection of affected (*nn*) and carrier (*Nn*) pigs now requires only a simple PCR test. The PCR primers enable amplification of a specific region of what has been renamed the calcium release channel 1 (CRC 1) gene. When incubated with the restriction enzyme, *Hha*I, PCR product from the normal allele is cut, but that from the mutant allele is not, as the mutation abolishes the *Hha*I recognition site.

In Australia, the Queensland Department of Primary Industry (QDPI) gained a licence to provide this test. Within four years of its introduction, there was so little residual demand for the test that the QDPI queried the need for their licence. The experience overseas has been similar and the *hal* mutation went from being the single most important genetic defect in pigs to being of negligible economic concern. Breeders could either get rid of it altogether or sequester it within lines where it was deliberately maintained. Clearly, knowledge of a particular DNA sequence directly responsible for economically important effects was of substantial value in this case and has set the scene and the expectation for further benefits from other DNA markers within genes in pigs.

Disease Resistance

Selection for resistance to diseases in animals represents a particular challenge. Almost all breeders are unwilling to subject their breeding animals to deliberate challenge by potentially lethal or debilitating pathogens, because of likely large economic losses. Yet without data on animal survival and performance in the face of such challenge, little or no improvement can be made. Reliance on sporadic outbreaks on disease in populations of animals vaccinated or treated with antibiotics or similar agents provides only very limited opportunity to recognise resistant animals. Virtually all pig breeders tightly quarantine their breeding facilities and would never dream of deliberately introducing pathogens or even animals challenged with pathogens into their breeding facilities.

For these reasons, DNA-based tests for recognition of pigs resistant to disease have an enormous appeal, especially if the actual gene and mutation responsible for resistance has been identified. Thus QTLs for disease resistance are of lesser value since continued exposure to the pathogen will be necessary to determine the association between marker allele and resistance QTL allele. Fortunately, there is one case in pig genetics where there is a reasonably high degree of confidence that the gene conferring resistance to a disease has been identified. Resistance to the pathogenic *Escherischia coli* (*E. coli*) F18 strain is due to a single locus, called ECF18R, located on pig chromosome six (Vögeli *et al.*, 1996). Originally, resistance could only be diagnosed using a complicated assay to test whether the bacteria bound to receptors in the gut lining. The map position yielded several local positional candidates of which fucosyl transferase 1 (FUT1) is almost certainly ECF18R (Meijerink *et al.*, 1997). It is believed that FUT1 causes a sugar to be attached to the surface of cells in the gut, which forms part of the receptor to which the bacteria bind. A non-functional form of the gene, not allowing attachment of this sugar, provides resistance. One allelic variant of FUT1 is almost perfectly associated with the resistance phenotype. If it is finally confirmed as the causal variant for resistance, selection for resistance to *E. coli F18* will require only a simple genotyping test of potential breeding stock without any need for disease challenges. The Swiss group, which discovered the association between the variants of FUT1 and resistance, has taken out a patent.

Coincidentally, the CRC1 gene and the ECF18R gene are located very close to each other on pig chromosome six. In the original Swiss population analysed by Meijerink *et al.* (1997), the favourable resistance allele at ECF18R was found associated on the same chromosome with the unfavourable CRC1 allele in about 93% of cases. If this association (linkage disequilibrium) is present in all populations of pigs, it would mean that programs to eliminate the unfavourable CRC1 allele would also have caused a strong reduction in the frequency of the favourable resistance allele at FUT1 as well. Unfortunately any selection program, whether performance based or DNA based, has the capacity to eliminate unrecognised favourable genes in the process of increasing the frequency of other favourable genes. In the euphemistic language of warfare, there can be some "collateral damage" at other loci in the process of increasing the frequency of a favourable allele at a particular locus. However, if favourable variants can be recognised at both loci by DNA tests, then it will be possible to identify those relatively rare animals having the favourable combination of alleles at each locus.

Geneticists in Belgium are hot on the trail of a gene for resistance to another bacterial disease in pigs caused by the K88 strain of *E. coli*. Peelman (1998) reported that his laboratory had very tightly linked markers for resistance to two of the three subtypes of K88 *E. coli* and had isolated a large insert clone containing the gene. The remaining task is to identify the gene within this clone responsible for resistance. This gene may well be new to science.

Productivity

Recently two laboratories in Sweden (Jeon *et al.*, 1999) and Belgium (Nezer *et al.*, 1999) have identified the gene underlying a substantial QTL effect on muscle mass and fat deposition which had been mapped to the tip of porcine chromosome two. This QTL has a very particular feature of expression, namely paternal imprinting, enabling it to be matched with insulin-like growth factor 2 (IGF2), which maps to the same location and is known from mouse and human studies to be imprinted. Paternal imprinting means that among progeny, only the paternally inherited allele is expressed. In the Large White by Wild Boar F2 resource examined by the Swedish group, the imprinted effects of the IGF2 locus were very large, with 31% of the F2 variance for % lean meat in ham, 24% for lean meat mass in ham, 10% for average backfat depth, 14% for weight of heart and even 8% for an indicator of meat quality, muscle reflectance, explained by the quantitative effect of this locus. The Belgian group analysed a large Pietrain by Large White F2 resource and estimated that the IGF2 and CRC1 loci have similar effects on fat deposition and jointly explain 50% of the breed difference between Pietrain and Large White for muscularity and leanness. Although the mutations at IGF2 responsible for the QTL effects in these two resources have not yet been identified, the imprinted pattern of expression makes it

almost certain that IGF2 is responsible since imprinted loci are relatively rare. What is clear though is that different mutations are responsible for these very large effects, with at least three QTL alleles detected in the two studies.

Reproduction

Reproductive performance traits typically have low heritabilities and as a result are intractable to improvement by conventional selection based on animal performance. For this reason, marker assisted selection, particularly using DNA markers for the causally relevant alleles, has a particular appeal. It would avoid the need to repeatedly measure a difficult trait and also would reduce generation interval. At this stage, there is only one success in the use of DNA markers for reproductive traits in pigs and this was arrived at via the candidate gene approach rather than from gene mapping. In the early to mid 1990s, workers in Rothschild's group at Iowa State University decided to evaluate restriction fragment length variants of the estrogen receptor (ESR) locus for effects on litter size. Initially the analyses were concentrated on resource families bred by crossing highly fecund Chinese pigs with European breeds and very large effects on litter size were observed. Later analyses were performed within breeds and smaller effects in general were observed, especially within European breeds. Rothschild *et al.* (1997) reported differences between female Chinese pig ESR BB and AA homozygotes of 2.3 pigs born alive for the first parity. In commercial Large White crosses, the difference is about 0.9 pigs per litter. Based on 9,015 litter records from 4,262 sows in four Large White-based commercial pig lines, Short *et al.* (1997) reported that total number born (TNB) and number born alive (NBA) were increased per favourable allele of ESR with additive effects of 0.42 (0.31) and 0.39 (0.31) pigs/litter in the first parity (later parities in parentheses), respectively. Dominance effects were near zero in parity one, but were 0.16 and 0.14 pigs for TNB and NBA, respectively, in later parities. Unfortunately, no other laboratories have evaluated the ESR effect on litter size in pigs and no general benefit to the industry has flowed from this discovery, as the rights to use it are held exclusively by the Pig Improvement Company (PIC). An indirect test of the effect of this locus on fecundity has been performed using a unique inbred line of mice produced at the University of Sydney with an average litter size of 15-16 pups per litter. Using backcross and F2 families bred between these mice and a standard inbred strain with an average of about 5-6 pups per litter, the University of Sydney group has not only scanned much of the genome for litter size QTL, but evaluated specific regions of interest, like the vicinity of the estrogen receptor locus. Due to the unavailability of markers within ESR in the mouse, very closely linked microsatellites flanking ESR were used. Preliminary analyses have indicated a highly significant effect on litter size in or near ESR in these mouse resource families (Arsenault *et al.*, 1998). A very large F2 resource consisting of over 600 F2 females with four litter records, including ovulation and implantation data for the last litter, are now ready for a very thorough evaluation of the ESR effect. This resource will also provide a very cost-effective means for finding other fertility QTL. The comparative map between mouse and pig is sufficient to enable quick transfer of this information to guide choice of hyperpolymorphic markers or better still comparative positional candidates in the pig.

The Future

Single Nucleotide Polymorphisms (SNPs)

As the above examples illustrate, the ability to detect DNA variants responsible for quantitative effects opens the possibility for precise identification of superior breeding animals without any need for performance testing. Undoubtedly there will be a huge increase in the number of such useful DNA variants available as the structure and content of the genomes of many mammals are more closely scrutinised. Numerous QTL studies are being performed in the pig in many laboratories throughout the world and hopefully gene identification will not lag too far behind. However, it is important to not underestimate the magnitude of the task involved in identifying these useful genes and polymorphisms. Ultimately, technology will be required to genotype many animals for

hundreds if not thousands of such polymorphisms to identify the best animals for use in breeding.

Fortunately human genetics, and more specifically the emerging discipline of pharmacogenomics, with its objective of gathering SNP data for every human gene, is providing the incentive for development of new technologies to automate the capture of such data. Many novel principles for so-called gene chips are being investigated although all involve allele-specific hybridisation in some form (Stoneking *et al.*, 1991; Howell *et al.*, 1999). Detection of the outcomes involves technologies ranging from mass spectrometry (Hall *et al.*, 1998) to semiconductor microchips (Gilles *et al.*, 1999). Acronyms and buzz words are proliferating – ASOH for allele specific oligonucleotide hybridisation, DASH for dynamic allele specific hybridisation and DNA chips to describe miniaturised oligonucleotide probe arrays – as this automated technology goes through a very rapid phase of development. Ultimately, there will be one or more technologies capable of automatically genotyping thousands of polymorphisms simultaneously and at low cost.

Radiation hybrid maps and sets of contiguous clones

Somatic cell hybrid mapping utilises interspecific cell hybrids, made by fusing cells in tissue culture. Chromosomes from a rodent species, usually mouse or hamster, are retained, while chromosomes from the species of interest, such as the pig, are randomly lost in culture. Analysis of the pattern of co-retention of markers in a small panel of hybrid cell clones enables mapping of genes or markers to chromosomes or so-called syntenic groups. Somatic cell mapping has already made a valuable contribution to gene mapping in the pig. The somatic hybrid panel, consisting of only 27 clones, produced and characterised by the INRA laboratories in Toulouse (Robic *et al.*, 1996; Yerle *et al.*, 1996) has enabled regional assignments to about 110 different locations within the pig genome. This panel has greatly assisted many laboratories, including the University of Sydney laboratory, to quickly work out reasonably accurate positions of genes. However, an even more accurate form of somatic cell mapping is possible. If the cells from the pig are irradiated in order to fragment the chromosomes prior to fusion with mouse or hamster cells, the resolution of the map will be greatly increased. The same French group, in association with collaborators from the University of Minnesota, has recently produced a porcine radiation hybrid panel (Yerle *et al.*, 1998) with a resolution approximately 18 times better than the best linkage map. Simple PCR analysis of only 126 clones from this panel enables resolution of gene and marker order virtually impossible with linkage maps. While radiation hybrid maps cannot identify QTLs, they will play a very important role in progressing from QTL to gene identification. Their high resolution will permit the alignment of large clones, such as yeast artificial chromosomes (YACs) or bacterial artificial chromosomes (BACs), into contigs. Contigs are ordered arrays of clones spanning chromosome regions. Already several groups have made BAC libraries for the pig, which jointly with very high resolution maps produced from radiation hybrids, will provide a resource for rapidly moving from an identified QTL effect to a small series of clones containing the gene or genes of interest. Together with comparative positional candidates obtained from human and mouse genomic studies, these resources will greatly assist pig geneticists on the trail of genes and their DNA variants influencing economically important traits. However, as quickly as this information is extracted from behind the veil of ignorance, it may be sequestered behind patents or other legal devices, such as trade secrecy. It will remain a challenge to the Australian pig industry to ensure economically feasible access to this information and the breeding tests based on it.

At the end of the trail, one would if possible like to be able to identify breeding animals using functionally characterised DNA variants. This is the ultimate goal of marker assisted selection. It will be very interesting to see how closely this goal is approached within the next five years.

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USE OF DNA TECHNOLOGY IN PIG BREEDING

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Abstract

The distribution of gene effects into three general classes of "polygenes", "oligogenes" and "major genes" is discussed. Analytical models that assume quantitative traits are influenced by many genes each of small effect (polygenes) are shown to be effective even in cases where few genes of large effect (major genes) are segregating. Segregation analysis is a statistical tool used to identify the existence of major genes. Traditionally, its application in pig breeding has been limited. However, with recent developments in algorithm design there is more optimism for its increased role in pig breeding. These developments are discussed. Segregation analysis can identify potentially recognizable genes of moderate to large effect (oligogenes). Assays at the DNA level must be used to confirm their existence. The first type of assay usually performed is the genome scan, which attempts to link certain regions of the genome with potential oligogenes. Such regions, tagged using genetic markers, are more generally referred to as quantitative trait loci (QTL). The statistical nature of a genome scan is quite different to that of estimating effects of discovered QTL in breeding. The reasons for this difference are outlined. The presence of DNA assays in breeding creates a myriad of extra technical information. The article forecasts an imminent surge in the existence of acronyms and accepted phrases to describe such information. The logistics of using DNA assays in breeding are also covered.

Introduction

The question of how many genes affect a typical animal performance trait has puzzled geneticists for generations. Quantitative geneticists commonly use a model that assumes an infinite number of genes each of small effect. This model, called the infinitesimal model, is justified on the basis of its mathematical simplicity, reliability and effectiveness. However, from early on geneticists recognized it is not perhaps the most realistic model.

Wright (1968) postulated an inverse relationship between the magnitude of the effect of genes and their frequency in the population. This relationship is shown in Figure 7. The distribution of gene effects has been divided into 3 major classes. 'Polygenes' are the many genes with small effect. The first section of this article explains why models that assume only polygenes have been and remain effective in predicting the genetic merit of farm animals, even in the knowledge that genes of moderate to large effect are segregating.

'Major genes' are genes with effects so large that their parameters can be estimated with sufficient accuracy using only phenotypic and family data. The next section of this article gives a brief introduction to the analytical method used to detect major genes, broadly referred to as segregation analysis. The potential for routine use of segregation analysis in genetic evaluation is also covered.

In 1934 in a landmark paper describing his work with guinea pigs, Sewall Wright (1934) used the term 'leading factors' to describe genes with large and potentially recognizable effects. However, more recently, 'oligogene' has become the more accepted terminology. Parameter estimation with sufficient accuracy by segregation analysis is unreliable for oligogenes, but not necessarily futile. Only modern DNA technologies provide the means to reliably detect and exploit oligogenes. The major aim of this article is to review how oligogenes are discovered, and more importantly, once discovered how the oligogenes can be exploited in practical pig breeding.

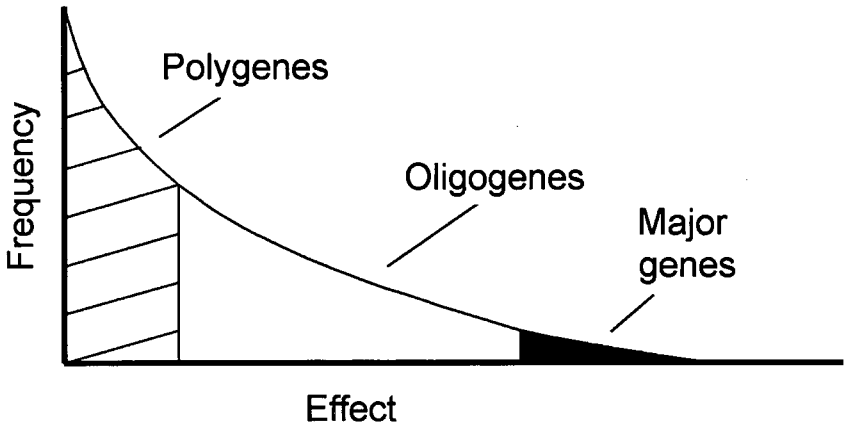

Figure 7. *Wright's postulated inverse relation between the frequency of a contributory locus and its effect (Wright, 1968).*

As in any scientific discipline nomenclature can often be confusing and misleading. The acronym QTL, which stands for quantitative trait locus, is an example of such nomenclature. It was introduced to distinguish loci with quantitative effects from those with primary qualitative effects, and could apply to each of the three classes of genes described. There are perhaps two reasons why the use of the acronym is so widespread in animal breeding. Generally there is a two step process to identifying oligogenes. Initially only a region on a genetic map is associated with a substantial and statistically significant effect on a trait. Because most traits in animal breeding are quantitative, this region becomes known as a QTL. A typical QTL is at least 8-10 million base pairs of DNA (Davis and Taylor, 1998), which is an immense area to fully characterize. Thus the second step is to use additional DNA technologies to reduce the QTL to the finest possible interval and attempt to identify the causal oligogenes. Because this second step is time consuming and requires great resources, rather than wait for the identification of oligogenes, many consider the information gained on the QTL should be immediately applied to breeding. There is some dispute over this proposition and the reasons for and against are discussed.

A further point in clarifying confusing nomenclature is the distinction between an oligogene and a major gene. A general rule of thumb is when the difference between the mean value of animals homozygous for the gene and animals not carrying the gene is equal to or more than one phenotypic standard deviation, then the gene is considered a major gene.

Polygenes and the infinitesimal model

One of the more important theorems in statistics is the central limit theorem. It states that data that are influenced by many small and unrelated random effects are approximately normally distributed. By assuming the genetic value of an animal for a quantitative trait is the result of many genes of small and unrelated effects, geneticists can safely assume breeding values follow a normal distribution, even after selection. It then becomes much easier to describe the genetics of a population in terms of means, variances and co-variances. A statistical procedure such as best linear unbiased prediction (BLUP) can predict individual points from a conditional normal distribution, which equate to animals' breeding values. Though it may seem less intuitive, it is certainly easier than identifying the effects of all contributing loci, which could number in the thousands.

However, if the distribution of gene effects is like that shown in Figure 7, does a polygenic model of inheritance ignore or account for the oligogenes and major genes segregating for a trait? To answer this question the authors of this article undertook a simple simulation study. A population was generated that resembled a typical pig breeding population. The population structure consisted of a base population of 10 sires

and 100 dams and five overlapping generations of 1000 animals (5110 animals in the pedigree). In each generation 10 randomly chosen males were each mated to 10 randomly selected females to produce 10 full sibs. The effect of a segregating oligogene was superimposed on an animal's polygenic breeding value, which was generated using a normal distribution. For all non-base animals, adding year, contemporary group and individual residual effects to the genetic effect simulated an observation. The variance due to the oligogene is a function of its frequency, its additive effect and the dominance deviation. A standard mixed model procedure was then used to estimate the genetic and environmental parameters fitting fixed effects of year and management group and a random effect due to polygenes. The results of several analyses in which the genetic variances differed are shown in Table 2. In each case the variance due to the oligogene was accounted for by the polygenic variance.

Table 2. Estimates of polygenic (Vpg) and residual variances (Ve) and their standard errors (SE) using simulated data. Simulated genetic variance was attributable to both polygenes and oligogenes (Vog).

Simulated values			Estimated values			
Ve	Vpg	Vog	Ve	SE	Vpg	SE
7.0	0.0	3.0	6.99	0.26	3.18	0.36
7.0	1.0	2.0	7.04	0.27	3.12	0.36
7.0	2.0	1.0	7.13	0.25	2.96	0.30

A breeder may well ask why invest effort in identifying oligogenes and major genes, considering that traditional polygenic models can account for their effects. The simplest explanation is that selection accuracy can be improved by knowing, with reasonable certainty, genotypes for genes of moderate to large effect. That is, more accurate than what is achieved through using just phenotypes and pedigree. Goddard (1999) will be discussing this topic in greater detail in this symposium.

Segregation analysis

Segregation analysis refers to any form of analysis undertaken on familial data with the objective to elucidate single gene effects. The implication is that no DNA assay is used. It has a prominent role in human genetics where traits are defined by phenotypes that are more likely to be affection status for a disease, rather than a continuous variable. Thus a single gene effect in the absence of any polygenic variation is often hypothesized. Furthermore, pedigrees are likely to be smaller and less complex than in animal populations. This allows the statistical formulation of the problem to become a lot more tractable. Nicholas (1997) reviewed some instances where segregation analysis has been successfully used in pig breeding, but generally it is not a routine procedure. The lack of its use in animal breeding stems just as much from statistical obstacles as the belief that animal performance traits are truly polygenic. Many of the early attempts at segregation analysis used 'likelihood' based approaches already in use in human genetics. A likelihood is a statistic that measures the probability of the data occurring, given a model and the parameters describing that model. The likelihood statistic is generated using a likelihood function. In mathematical language, the likelihood function is generally written as $l(\theta|y)$, where θ represents the parameter(s) and y represents the observed data. In Figure 8, there is an example of a curve generated by a likelihood function for a single, hypothetical parameter. The horizontal axis represents the range of possible parameter estimates and the vertical axis represents the value for the likelihood. The estimate of the parameter that maximizes the likelihood (maximum likelihood estimate or M.L.E.) has many desirable statistical properties, chiefly it has minimum mean square error. Usually numerical methods are used to locate a 'point' estimate of the parameter that maximizes the likelihood. Certain points on the curve are calculated in a systematic search for the maximum. There is no inherent interest in the actual shape of the curve. In Figure 8, this point estimate is represented by the symbol $\hat{\theta}$, which is the mode of the likelihood curve.

The likelihood in segregation analysis has to be computed by considering all possible genotype combinations. If a single locus with two alleles is considered, the number of possible combinations in an extended human family of 20 individuals is 3^{20}, which is a feasible number of combinations to traverse. Even with larger pedigrees, the computation remains feasible because of a procedure called "peeling" (Elston and Stewart, 1971). Peeling can be described as systematic use of relatives' information to minimize the number of operations needed to compute a likelihood. However in animal breeding two major complications arise. First, the animal geneticist has to consider the effects of a major gene against a background of polygenic variation. Second, animal pedigrees are prone to containing 'loops'. Loops are ambiguous paths by which a gene can be transmitted to an individual from its ancestors and one of their causes is inbreeding. Because of these complications the likelihood is "unpeelable". Several authors have developed an approximate form of peeling and maximum likelihood analysis (Hofer and Kennedy, 1993; Meuwissen and Goddard, 1997) but the resulting estimates can never be considered exact. Others have used Markov Chain Monte Carlo (MCMC) (Guo and Thompson, 1994; Janss *et al.*, 1997) which is a category of statistical tools used to explore the distribution of the likelihood without ever having to compute it. Stochastic simulation is used to generate the area or mass underneath the curve (shaded area in Figure 8). Provided there has been adequate sampling, the mode or mean of the sampled distribution is the estimate with the highest likelihood, given the data and any prior information.

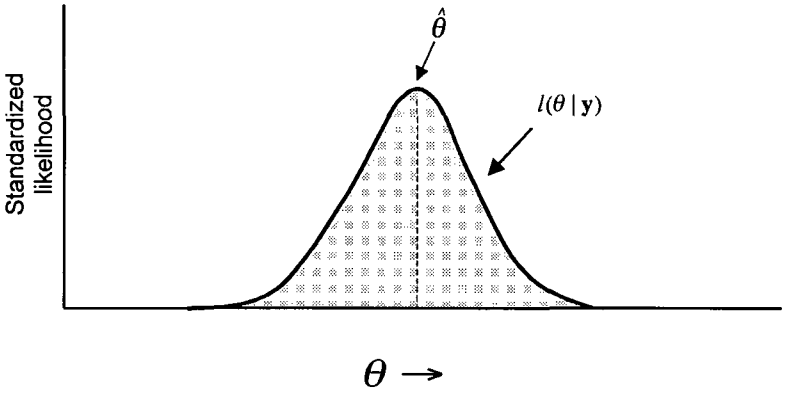

Figure 8. *Example of a standardized likelihood for observations y using a model with parameter denoted θ.*

However, use of MCMC to estimate the effects of major genes in animal pedigrees creates its own set of problems. The sampling of genotypes often experiences "lock in". "Lock in'" means a particular genotype combination for a section of the pedigree is continually re-sampled, and thus the complete range of genotype combinations is never explored. Genotypes are sampled sequentially, and a genotype for each individual is sampled based on their relatives' information. This information creates a feedback mechanism often causing the sampling to remain "motionless", irrespective of the order in which individuals are sampled. Tier and Henshall (personal communication) have recently developed a MCMC algorithm in which the genotypes of a complete pedigree were sampled jointly. As a consequence, their algorithm was able to sample the parameter space a lot more freely. The method was tested on data from a commercial pig line. Table 3 shows the results of the analysis on backfat depth measured with real time ultrasound. A general-purpose segregation analysis program using this novel method has been developed at the Animal Genetics and Breeding Unit (AGBU). It has been given the title 'The Gene Detective'.

Table 3. Results of segregation analysis for backfat data from a commercial pig line. Estimates of genetic parameters and their sampling variances (S_var) are given with and without fitting an effect due to a major gene. Parameters include half the difference between the mean value of animals homozygous for the gene and animals not carrying the major gene (g), variance due to the major gene (Vmg), polygenic variance (Vpg) and residual variance (Ve).

	With major gene effect				Without major gene effect	
	g	Vmg	Vpg	Ve	Vpg	Ve
Estimate	1.57	1.21	1.78	3.34	2.55	3.52
S_var	0.14	0.22	0.09	0.09	0.09	0.06

Nicholas (1997) has already indicated a program such as 'The Gene Detective' is useful for indicating whether a population has potentially recognizable oligogenes segregating for a particular trait or traits. Animals with high probability of carrying a putative oligogene can be identified and subsequently used in experiments to confirm the oligogene's existence. A major gene, if identified, could possibly be exploited without the need for confirmation using DNA assays. Instead a series of test matings may be sufficient. An example is a test that crosses putative homozygous sires to dams that are putative non-carriers. The F1 males are then crossed to non-carriers. The expectation that the F1 progeny are heterozygous would be confirmed on observing bi-modality in the distribution of the F2 progeny phenotypes. Once the major gene has been validated from these or similar test matings, the segregation analysis program can then routinely predict the genotype status of all breeding animals and this information can be used to aid selection decisions.

Scanning genomes for QTL

Discovery of major genes will at most be infrequent. Segregation analysis will most likely serve as a useful aid in detecting potentially recognizable oligogenes. Experiments involving DNA assays are needed to confirm their existence. Scanning the genome and establishing particular regions as being consistent with oligogenes is most often the first type of experiment performed. As stated in the introduction, it is more correct to label these chromosomal regions as QTL. It is possible that QTL could comprise two or more tightly linked oligogenes. It is only when further DNA assays can identify the actual gene or genes involved that the acronym QTL is no longer needed. It is also important to note that QTL scans will often proceed without any prior evidence that oligogenes are segregating. Scans for QTL have been undertaken only on the knowledge that the traits under study have a genetic basis and with the hope that the distribution of gene effects is as depicted in Figure 7.

A prerequisite for QTL scans is the genetic map. It is analogous to a road or a geographical map in that it provides references for the location of genes. Moran (1999) elsewhere in this symposium has covered the essentials of the genetic map and anonymous DNA markers.

Another prerequisite for QTL scans is the experimental design. Given the available resources, the experimenter has to choose a design that will maximize the probability of detecting QTL. What breed or breeds to use, how many families to be generated and family size are typical issues to be examined. The general reader could well ask why couldn't arbitrary, existing pedigrees be used for the purposes of scanning genomes for QTL? To answer this question it is necessary to examine the statistical issues involved.

The statistical challenges in detecting QTL with and without genetic markers are very similar. It was noted in the previous section that segregation analysis involves finding a mode of a likelihood distribution, either by computing actual points from the likelihood function or using stochastic simulation to provide an actual image of the distribution. There has to be a computation over, or adequate sampling from, a very large number of genotype combinations. The challenge in detecting QTL with markers is the

same except that the markers provide information on the actual QTL genotype combination in the studied population. There is a certain amount of computation over QTL genotype combinations because of meiotic recombination between marker loci and QTL and because of ambiguity in marker allele transmission, but it is certainly only minor in comparison to the situation with no markers. However, the animal geneticist still has to deal with the problem of polygenic effects. If not correctly accounted for, they can certainly bias estimates of QTL effects. The most efficient way of dealing with polygenic effects is to use a structured design. For example, a common design in animal genetics is the half-sib design. A select group of sires is mated over numerous dams to produce 100 or more progeny each. It is assumed the sets of dams mated to each sire are a random selection and that the mean of the polygenic contributions from each set of dams is the same as the population mean. Thus polygenic effects are not fitted in the model but are regarded as contributing to residual error, without causing undue bias. Other designs use full-sib family information. If a number of half-sib families are analyzed together, a sire effect can be fitted to account for the polygenic contributions from the sires. If more complex pedigrees were used, for example, multiple families over multiple generations, then the correlated structure of the polygenic effects would then have to be considered. This increases dramatically both the statistical and computational aspects of the problem. The current thought is that there is no great benefit to using complex pedigrees for the purposes of QTL scans.

Once QTL scans have been completed and QTL have been identified, there are two avenues to take. The first is to undertake further DNA assays to identify the actual oligogenes involved. The second is to apply the current information on QTL to pig breeding. It is most likely that both avenues are sought concurrently, though there are good reasons to only consider using information on identified oligogenes in pig breeding. These reasons will become clearer after the discussion in the next section.

Identifying oligogenes

The identification of oligogenes becomes less a statistical exercise and becomes more the domain of the molecular geneticist. Moran (1999) has referred to techniques such as positional candidate gene identification and positional cloning elsewhere in this symposium. Briefly, positional candidate gene identification requires the alignment of the pig genetic map with the more complete human or mouse genetic maps in order to identify candidate genes that lie within the region targeted by the QTL scan. Positional cloning is used when there is an absence of any candidate genes. First the QTL must be mapped to the finest possible region and every gene in that region is identified and evaluated as a possible candidate.

Using oligogenes in pig breeding

Before an identified oligogene can be used in pig breeding, a test must be developed that can identify the variants of the oligogene. There are three testing situations worth distinguishing and these are shown in Figure 9. In the figure, horizontal lines represent anonymous DNA sequence with no apparent function. The start and stop signals signify a region of DNA sequence that is a gene. Within the gene there is both DNA sequence with no apparent function (introns) and DNA sequence which specifies information necessary to build a protein (exons). Exons are represented by the black boxes. Shaded boxes represent small pieces of anonymous DNA sequence that can be used as genetic markers. The first situation shown is where the test involves typing the mutation that actually causes the effect as in the halothane *(Hal)* gene test. The second and third situation shown is where the test involves typing a genetic marker. In situation (ii) the marker is within, or very close to, the gene and in situation (iii) the marker is near the gene. Consider the last situation. Suppose there is a marker that has two variants *M* and *m* and an oligogene that has two variants *G* and *g*. Assume that the oligogene variant *G* has a favourable effect on a trait and the variant *g* has an unfavourable effect. Figure 10 shows homologous chromosomes pairing during meiosis. In case one, there has been no meiotic recombination and, following gametogenesis, gametes show either the *M/G* or the

m/g associations. In case two, meiotic recombination has separated the linked marker and the gene, resulting in cases where associations between marker types and variants of the oligogene have been inverted. That is, gametes now show *M/g* and *m/G* associations in addition to *M/G* or the *m/g* associations. A tendency for one pattern of associations (*M* with *G* and *m* with *g*) to be more frequent than alternative patterns of associations means the marker is in linkage disequilibrium with the gene. The correct terminology for describing the pattern of association is **linkage phase**. Linkage disequilibrium and equilibrium are discussed further in Goddard (1999) elsewhere in this symposium. When the linkage disequilibrium is complete such as shown in situation (ii) in Figure 9 this is as good as typing the actual functional mutation.

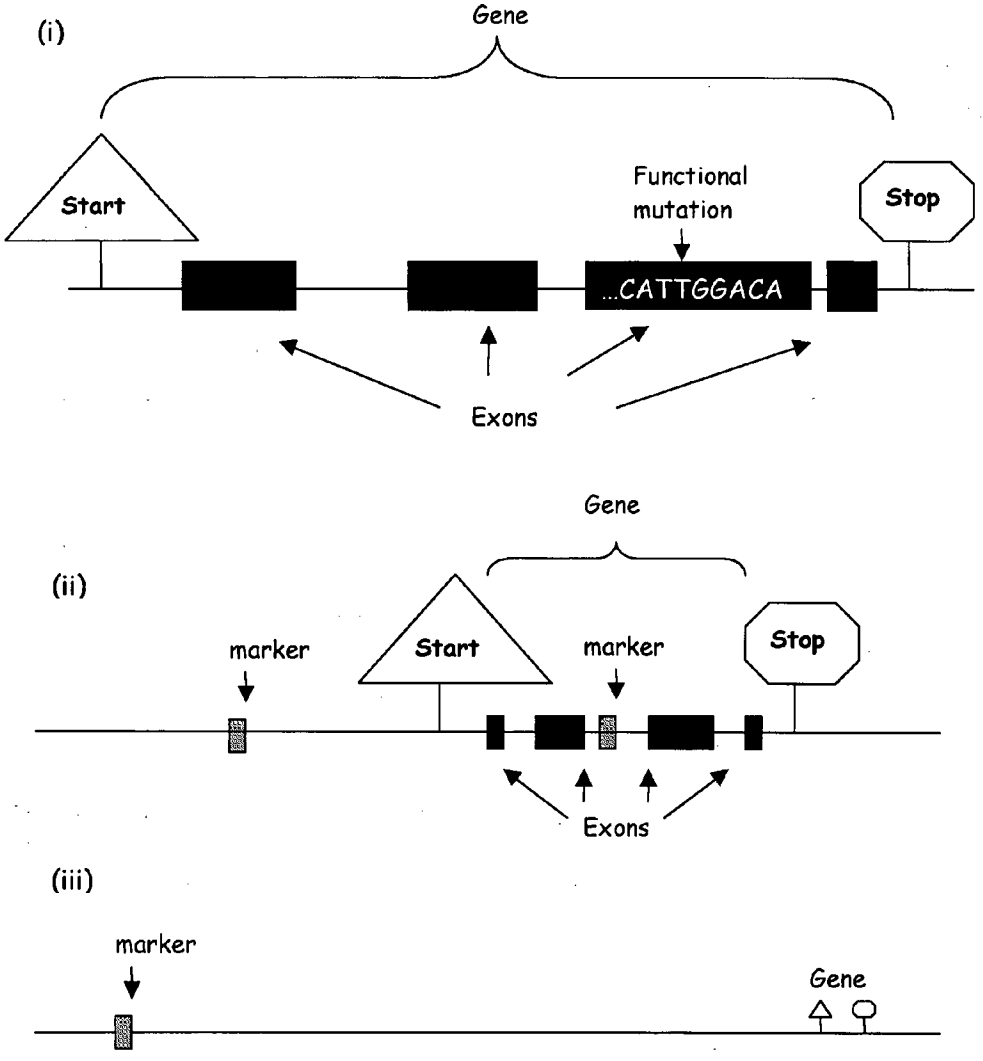

Figure 9. *Types of genetic markers: (i) mutation that actually causes the effect can be typed (as in the Hal gene); (ii) marker very close to, or within the gene (the marker is in complete linkage disequilibrium with the functional mutation); (iii) marker near a gene (linkage disequilibrium between marker and functional mutation is incomplete).*

Tests that involve typing markers where the linkage disequilibrium with the functional mutation is not complete are problematic. This is because linkage phase must be assessed for each family or equivalently the effect of marker variants must be estimated on a within family basis. If the recombination is high, linkage phase may also have to be assessed each generation. To illustrate, consider two breeders each with a boar for whom they wish to test the status of a hypothetical gene. The linked marker and the gene are close enough that recombination occurs once every 100 meioses on average. Thus a particular linkage phase is liable, but not certain, to continue within a family line for several generations. Genetic tests are performed on the boars and samples of their progeny. A progeny test is required to establish the linkage phase. Both boars are shown to be heterozygous for the marker, but are shown to have opposite linkage phases. It would be very confusing for both breeders to realize that the marker *M* indicates the opposite effect in the other's herd. It would also be embarrassing for a breeder to have sold a son of the tested boar on the guarantee that the son carries the marker allele indicating the favourable gene variant. Then only to be shown by the purchaser that the opposite seems to be case.

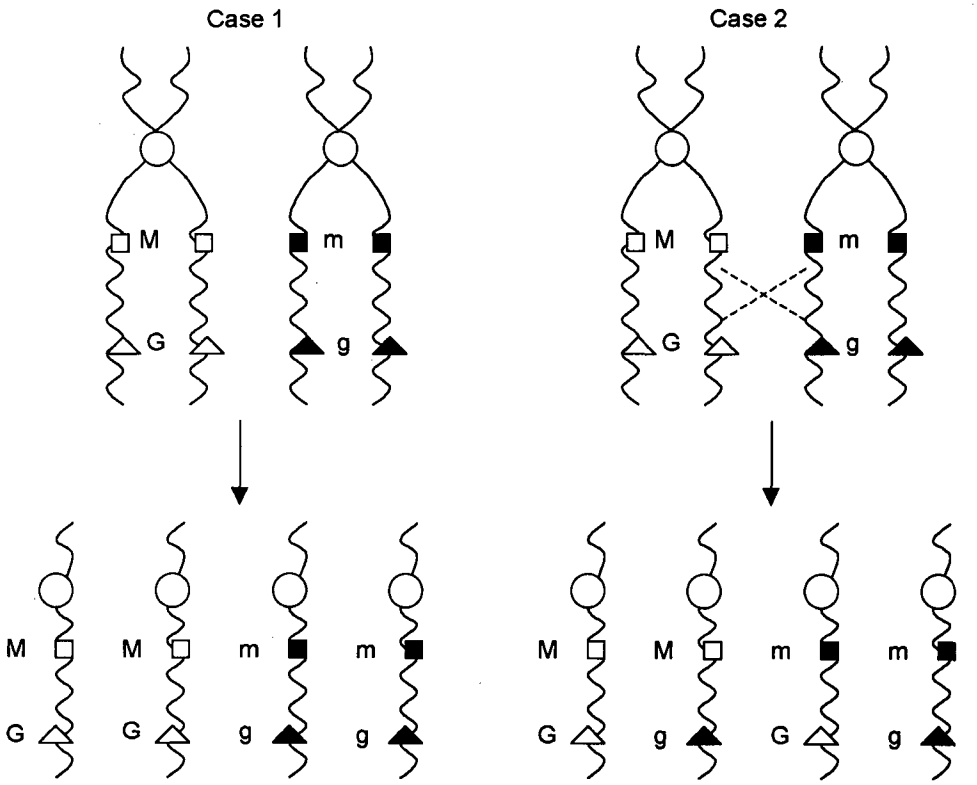

Figure 10. *Pairing of homologous chromosomes during meiosis. Each homologue forms two sister chromatids. Case 1. No exchange of genes between non-sister chromatids in the formation of gametes. Case 2. Breakage of non-sister chromatids, crossing-over and reunion resulting in reciprocal exchange of genes.*

These problems would never occur in the case of a marker that is in complete linkage disequilibrium with the oligogene, because the variants of the marker consistently indicate the variants of the oligogene. This desirable property warrants the considerable research effort in identifying the oligogene responsible for the QTL effect.

Using QTL in pig breeding

The amount of research effort and time consumed in tracking down oligogenes cannot be overstated. In swine genetics, there have been few reported discoveries of oligogenes responsible for previously reported QTL effects. Compare this with the plethora of reported QTL for growth and performance, meat quality, reproduction and disease resistance traits. A summary of reported QTL in the pig can be found in Rothschild (1997). Also a summary of current QTL mapping projects can be found on the World Wide Web site http://www.genetics-network.ch/Genomanalseprojekte/Survey_ADG_ANGENMAP.htm. Given that there is not going to be a sudden growth in the availability of markers in complete linkage disequilibrium with discovered oligogenes, the immediate focus is going to be on the incorporation of markers that have incomplete linkage disequilibrium with unidentified oligogenes (QTL) into mainstream pig breeding programs. Marker assisted breeding refers to the broad application of markers in breeding, e.g., aiding in: parentage verification; the diagnosis of genetic disease; and the selection of breeding stock. Marker assisted selection (MAS) refers only to the last category.

This section examines the following two areas: models of analysis for MAS; and logistics of undertaking MAS. The question of whether MAS is worthwhile compared to more conventional methods of selection has been covered by Goddard (1999) elsewhere in this symposium.

Models for analysis

Scanning genomes for QTL and the prediction of QTL effects in breeding are two quite separate issues. The misconception that they are the same is quite common. As stated in a previous section a researcher needs a genetic map and an experimental design to undertake genome scans. The result of the research is intellectual property, i.e., knowledge on the location of QTL and the markers linked to them. If this information is released into the public domain, bought, or obtained through some other contractual arrangement, the breeders can then contemplate MAS. Models of analysis for estimating the genetic and environmental parameters are liable to be quite different. Genome scans use structured experiments and as a rule do not use complex pedigrees. For predicting QTL effects in commercial breeding applications, complex pedigrees are likely to be the norm. Polygenic effects become important and cannot be simply fitted as "family effects" as they sometimes are in models used in genome scans. Computational complexity is an issue in genome scans because multiple sites across the genome are tested. In MAS, computational complexity is less an issue because only a few selected sites are investigated. With these points in mind, several methods of analysis for MAS are emerging. A method popular with animal breeders is the random effects model (Fernando and Grossman, 1989; van Arendonk *et al.*, 1994). The conventional BLUP animal model is extended to account for QTL allelic effects. The inverse of a matrix describing the correlated structure of the allelic effects is needed just as a numerator relationship matrix (NRM) is needed to describe the correlated structure of the polygenic effects. This matrix is often referred to as a gametic relationship matrix (GRM). To derive the inverse of the GRM the probabilities of QTL allele origin, conditional on marker information, are required, i.e., the probability the allele received from the sire is the same as that of the paternal grandfather or paternal grandmother. Deriving these probabilities in complex pedigrees is not an easy task. Generally, these methods have been developed for a single marker locus linked to the QTL. Meuwissen and Goddard (1996) have derived approximate methods for use in random effect models that take account of multiple marker loci.

The authors of this article are currently developing an MCMC approach to predicting marked QTL effects in complex pedigrees. Algorithms essential to 'The Gene Detective' will be enhanced to take account of multiple marker information. Similar in philosophy to 'The Gene Detective' in segregation analysis mode, the QTL genotype effects are considered as fixed effects and sampled as such.

It is important in any approach that multiple traits are considered. For QTL to be useful to animal breeders, not only must it have a positive effect for one trait in the breeding objective, but any negative effects on other traits must be quantified.

Logistics of MAS

The imminent use of DNA technology in pig breeding will demand additional genetic idioms to become common place. The advent of new accepted phrases and acronyms will be coupled with an increased complexity in the logistics of breeding. This section is an attempt at forecasting the "new logistics and language" of breeding with DNA technology.

Much depends on how the breeder wishes to access the technology. On the one hand, a breeder may consider within-herd evaluation of gene markers and ongoing selection using the self obtained gene marker information. On the other hand, another breeder may leave the evaluation of the gene markers to others and consider buying breeding stock with certified gene marker information. These two scenarios are now considered.

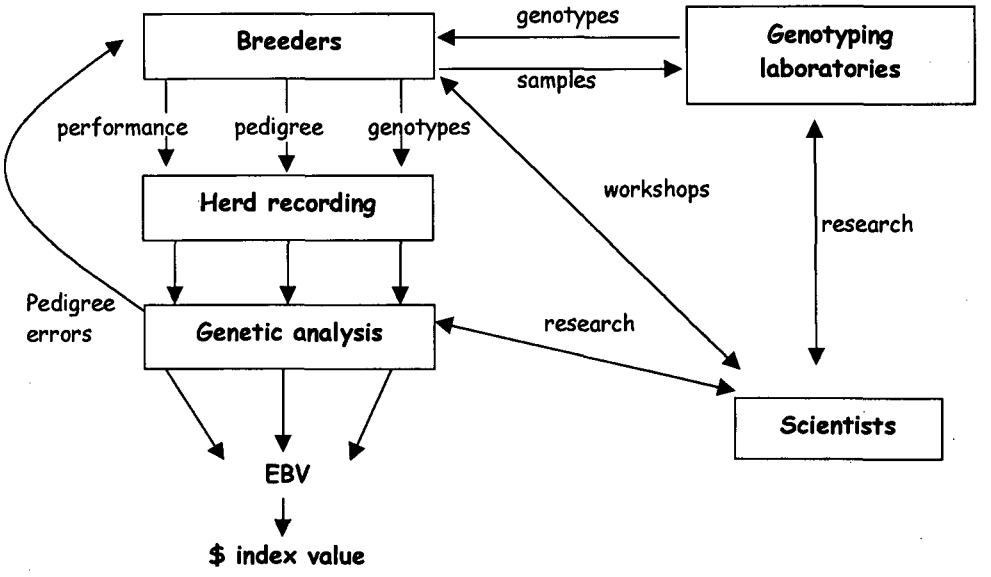

Figure 11. *Likely pathways required for an Australian pig breeder to implement MAS.*

First the breeder who undertakes within-herd evaluation of gene markers. Figure 11 is a flow diagram describing the likely pathways required for an Australian pig breeder to implement MAS. Many of the pathways currently exist, i.e., the on-farm herd recording of performance traits and pedigree; and breeding value prediction using the PIGBLUP software. To implement MAS, the breeder is required to send a blood or tissue sample to a genotyping laboratory, which in turn sends back genotype information. Herd recording systems will need to be enhanced to store such information. Future releases of PIGBLUP will be able to combine genotype information with performance data, within a multi-trait analysis, to predict a range of effects and compute a host of sundry statistics discussed below. At this point, it is emphasized that an estimated breeding value (EBV) will still be predicted for each trait in the breeding objective. The definition of an EBV will not change from what it currently is – an estimate of the value of an individual's genes to future progeny. That is, an estimate of the value of genes both of large and small effect. Gene markers enable the partitioning of the EBV into QTL and polygenic effects. For a breeder considering within-herd evaluation of gene markers and ongoing selection, there is less need to even know the value of each partitioned effect. It is anticipated that

the next PIGBLUP will provide the tools for the breeder to combine information on markers with other available trait information into a single index ranking.

For the breeder considering this scenario, logistics are vitally important, especially the links to and from the genotyping laboratory. In pig breeding, the period between DNA sample collection and selection of parents is less than in most other livestock species. In this time, marker genotypes have to be assayed and combined with other trait information to yield an EBV. Figure 12 shows a hypothetical case scenario. A QTL has been shown in previous experiments to affect meat quality. A breeder, having acquired access to the intellectual property, seeks linkage relationships between the markers and the QTL for some select sires and the size of the QTL effect in these sire families. To obtain this information with sufficient accuracy, approximately 50 progeny with performance data per sire are needed. This translates to 6 litters assuming 8 piglets per litter. Assuming 1.5 litters born per week using natural service, a select boar's first month of service is needed to produce the test progeny. The boar stays in service for another 2 months. Performance data for the meat quality trait is measured when the test progeny are slaughtered at approximately six months of age. At most, seven months is all the time available to genotype the 50 progeny. Even more limiting is the 2-month time span between data collection and when the boar's last progeny are available for selection. Data analysis must be completed within this time, otherwise all DNA testing has been futile. It is likely that a boar targeted for DNA testing should be left in service longer, enabling greater exploitation of the results. For ongoing selection using the results of the DNA testing the available sons must be genotyped to determine which variants of the QTL they are carrying. Sons that are available for selection prior to the outcome of the DNA tests should either be held over or have a semen sample collected.

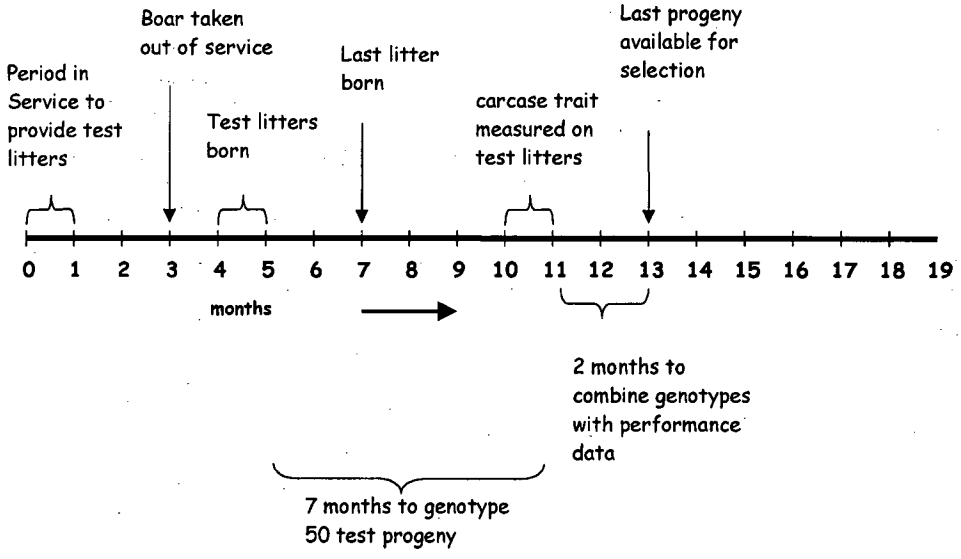

Figure 12. *Timing for evaluating gene markers in a boar for a hypothetical carcase trait. Time zero coincides with boar's introduction to service.*

It is time now to examine in greater detail one section of the pathway diagram in Figure 11. Specifically the section between genetic analysis and the procurement of a $ index value. Figure 13 shows a more detailed overview of the technicalities involved. Probably much to the relief of the breeder, much of this information can remain hidden within the black box of genetic evaluation. However, for the breeder who buys breeding stock with certified gene marker information, a single $ index value is of little value, since it does not relate to their herd. It is more essential that this type of breeder understands the breakdown of information leading up to the calculation of a $ index value.

This information is shown in the boxes within the parentheses in Figure 13 and is specific to trait two. Similar breakdowns may exist for other traits, depending on whether there are marked QTL for those traits. As a result of the analysis there are estimated effects for each QTL genotype, denoted as $\hat{QQ},\hat{Qq},\hat{qQ},\hat{qq}$. There are also probabilities for each animal being each of four possible QTL genotypes, denoted as P_{QQ}, P_{Qq}, P_{qQ} and P_{qq}. Finally there are residual polygenic values (RPV) conditional on a QTL genotype. Because of intellectual property agreements it is probable the identity of marker alleles will not be made available. Thus linkage phases will not be explicitly revealed to the breeder. However, QTL genotype probabilities inform the breeder how well linkage phase has been established. Linked markers will result in situations ranging from the worst case scenario of an animal having an equal 25% probability of being all four genotypes to having a QTL genotype identified with 100% certainty. In the former case, the markers could not determine linkage phase and the situation is the same as having no marker information. In the latter case, linkage phase has been established beyond doubt. It is to be anticipated that linkage phases for the majority of animals will be known with 80 – 100% accuracy. Obviously a breeder would be ill advised to purchase or use a sire that has less than 80% chance of being the desired genotype.

In situations where the mode of inheritance of the QTL is non-additive or non-Mendelian, knowledge of genotype probabilities will aid in the optimal expression of the QTL in breeding programs. Consider for example an imprinted QTL such as the recently discovered QTL with major affect on muscle mass and fat deposition (Nezer *et al.*, 1999). Only heterozygous individuals who inherit the favourable variant from their sire express increased muscularity and leanness. To exploit this QTL in commercial breeding programs two separate lines are required: a sire line that is homozygous for the favourable variant; and a dam line in which the favourable variant is not carried.

To illustrate the concept of a conditional RPV consider the extreme example of an animal that is 50% likely to be either the QQ or qq genotype. The effect of the QQ genotype is +1 and the effect of qq is –1. In theory the EBV for total genetic merit remains the same whether the particular animal is regarded as one or the other. If the animal is regarded as QQ the RPV must then be smaller than the RPV if it was regarded as qq.

The next layer of information brings together the QTL genotype probabilities and the estimated QTL genotype effects into a single marked locus value (MLV). It is not relevant here to state precisely what the accuracy of the MLV is in statistical terms but it is a function of the variance of the genotype effect estimates and the level of uncertainty of the individual's genotype. Conditional RPV are also expressed as a single weighted RPV. Its accuracy also reflects the level of confidence in its estimation. It needs to be emphasized to the breeder that although QTL effects or major genes may have been discovered, a strong focus on residual polygenic values is still important. A case in point is the history of *Hal* testing in Australian pig breeding. A group within the Queensland Department of Primary Industries gained a license for the test from the Canadian owners. Within four years of commencing testing demand almost ceased to exist, presumably because most breeders had successfully eradicated the unwanted *n* allele. There are two issues of some concern here. Firstly, there has been loss of polygenic response because selection pressure is used up on the *Hal* gene. Secondly, there is loss of polygenic variance at genes closely linked to *Hal* and in linkage disequilibrium with it. It is possible that if such variation proved to be beneficial in the future, some important gene variants could have been lost.

The final layer of information is the combination of the MLV and the RPV, resulting in an EBV for total genetic merit. Economic weights are used to then combine EBV for the various traits tested, into a single $ index ranking.

In summary, use of DNA technology in breeding generates a mass of information, additional to that currently produced. Applying this information in breeding is perhaps just as great a challenge as producing it. It is vital that quantitative and molecular geneticists assist the breeder, by providing education through workshops and up to date breeding service products. The lines of communication between breeder and scientist, as shown in Figure 11, become more important than ever.

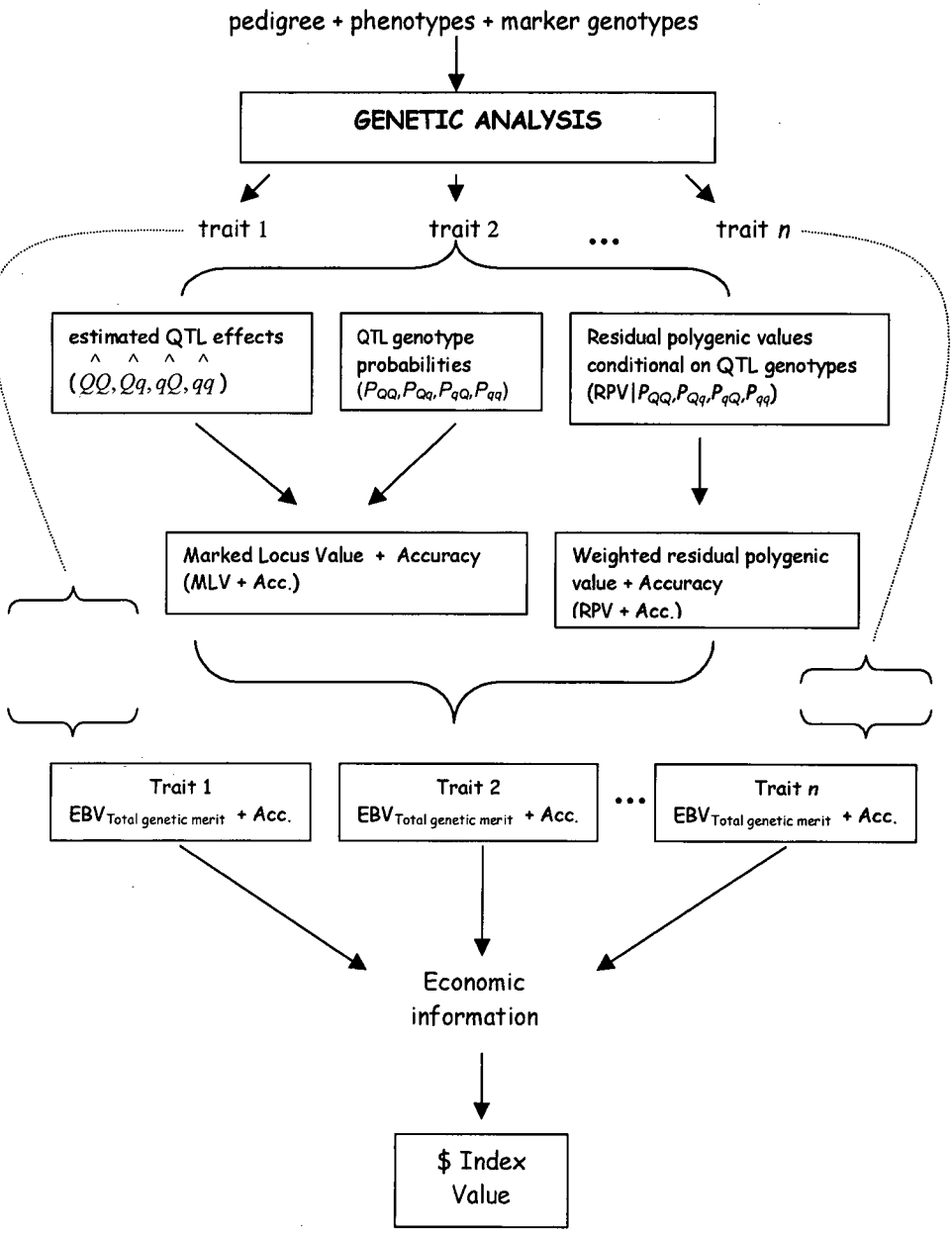

Figure 13. *Breakdown of information resulting from genetic analysis of commercial, performance recording data which includes genetic marker information. All information is eventually combined into a single $ index ranking which combines all available trait and economic information.*

There are some finer details not apparent in Figure 13 that are still yet to be discussed. Imagine if the halothane gene had never been discovered, but had presented itself in a routine genome scan as a QTL for reduced backfat. The QTL, if marketed cleverly, may have attracted the attention of many breeders wanting a rapid decrease in mean herd backfat levels. Then, only after MAS had been implemented, the undesirable effects of the QTL on meat quality came to light. This scenario demonstrates the necessity of breeders being aware of the possible pleiotropic effects of QTL, i.e., a QTL affecting

more than one trait. Characterizing a QTL is the responsibility of the researchers but indiscriminate use of available information is a common occurrence in marketing.

A problem currently facing geneticists, and having implications for the breeder wanting to purchase stock with gene marker information, is the choice between using a random or fixed effects model for analysis. Consider a sire that is advertised as carrying a QTL allele that has an effect of +1 in phenotypic standard deviation units. This effect had been estimated by treating the QTL as a fixed effect. Thus the effect could only theoretically be applied to the particular sample of individuals in which it was estimated and not to the wider population, for example a breed, from which the sample was taken. The breeder purchasing this sire has no guarantee that the advertised QTL effect will be transferable across different herds. The breeder has to consider whether the genetic backgrounds of the herd of origin and target herd are similar, and additionally if environments are similar. Then upon discounting possible QTL by genetic background or possible QTL by environment interaction effects, the breeder has only to consider the QTL allele frequency in the target herd. Introgressing a QTL into a target herd makes no sense if the favourable alleles are at high frequency. In some cases this might be obvious. For example, consider a hypothetical Meishan allele that increases litter size. Through a successful backcrossing program Large White genotypes were derived carrying the Meishan allele. A breeder wanting to purchase these genotypes can be assured the Meishan allele does not exist in the target herd. However, if the favourable allele was derived from similar genotypes to the target herd, the breeder has no other recourse but to undertake a series of test matings to determine the status of the gene. Use of a random effects model can potentially alleviate these problems of transferability of QTL effects between herds. A random effects model by definition means estimated parameters are applicable to a population as a whole, which potentially could encompass a breed. However, for a random effects model to be functional, variances due to the QTL need to be estimated, much the same way that polygenic variances are currently routinely estimated by research units. This would involve establishing a large representative data set for a breed, one that contains marker genotype information. The logistics of obtaining such a data set are considerable.

The future

The improvement of statistical methods to analyze DNA information will be motivated just as much by the breeder as the molecular geneticist. On the one hand, the ever-expanding array of DNA technologies will prompt new methods of analysis. However, just as importantly, enterprising breeders who initiate use of DNA information in breeding will be providing quantitative geneticists with the means to review and improve existing models of analysis. Their pioneering efforts will help establish how this information can provide the most benefit to pig breeds.

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Symposium continued on next page

BREEDING FOR INCREASED DISEASE RESISTANCE

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Abstract

Research into the improvement of disease resistance in pigs is reviewed. Susceptibility to infection, response to infections and *in vitro* measures of the immune response are under genetic control and therefore available for consideration in genetic improvement programmes. Although no selection has been applied to disease resistance in pigs in the past other than natural selection, the situation now exists whereby new technologies, both genetic and immunological, allow consideration of these traits in breeding objectives. Molecular and quantitative genetic research into the improvement of disease resistance is providing the information necessary for the use of traditional breeding methods as well as molecular information both for specific diseases and general immune response parameters. Models are being developed to assess the impact of genetic changes in disease resistance taking account of the epidemiology of specific diseases.

Introduction

Selection programmes in pigs have concentrated on improving production traits, mainly lean tissue growth, and to a lesser extent reproduction traits, such as number born alive. The populations undergoing selection are often maintained as high health status units and so the animals are not routinely challenged by disease causing agents. As a result, undesirable changes in disease resistance traits may have occurred as a result of unfavourable genetic correlations with traits in the selection objective or through genetic drift. Where selection has not had a deleterious effect upon disease resistance, it may still be possible to find a genetic solution to disease problems in order to improve herd health and welfare.

Rothschild (1998) quotes the costs associated with pig diseases in the United States at 1.5 billion US dollars annually. These losses result from mortality, reduced production efficiency due to sub-clinical disease, veterinary costs, and loss of the end product.

Performance in the commercial tier is routinely below that predicted from the nucleus tier. The Pig Research and Development Corporation is currently funding a major project investigating the causes of this 20-30% "Growth Gap". Williams *et al.* (1997) have shown that pigs eat more and gain weight faster and more efficiently in a high health status environment. Similarly, Holck *et al.* (1998) reported higher growth rate and lower amounts of α-1-acid glycoprotein (AGP) and lymphocytes of type CD4 (CD4) in pigs finished in an unrestricted research environment compared to those in a commercial environment. Amounts of AGP and numbers of CD4 increase in response to challenge of the immune system.

The genetic improvement of disease resistance may help to address the costs associated with disease, the reported "Growth Gap" and reduce potential risks. The purpose of this paper is to review the prospects for the genetic improvement of disease resistance in pigs. This paper concentrates on resistance to diseases caused by some biological pathogen, rather than conditions that result from an interaction between an environmental factor (other than pathogen presence) and a genotype. Examples of such conditions would include malignant hyperthermia, or halothane sensitivity. Needless to say, the understanding of the genetic mechanism of malignant hyperthermia and success of the molecular approaches to reducing the incidence are an impressive advertisement for molecular genetic approaches to pig improvement. For more information on the halothane gene story see Nicholas (1997) and Hermesch (1997), who cover the genetics and production effects of the halothane gene.

There are two mechanisms that can be considered for the improvement of genetic resistance to disease. The susceptibility of animals to infection can be reduced (i.e.,

prevent the disease getting a foothold in the animal's system) or the animal's ability to counter the effects of an infection (the symptoms of the disease and its effects upon production) via the immune response can be improved.

Selection for immune system responsiveness has been suggested as a method of improving disease resistance (Buschmann *et al.*, 1985; Warner *et al.*, 1987). In a multi-tiered breeding pyramid immune responsiveness traits may be a useful source of information with regard to predicting production in the commercial tier, where animals are likely to be subjected to more frequent and varied disease challenges than their counterparts in the breeding tier.

Genotype × environment interactions

Genotype × environment interactions (G×E) are observed when animals rank differently according to their genetic merit in different environments. Genotype × environment interactions have been reported between different levels of the pig production pyramid. This may be due in part to differences in health status across the units involved. For example, if high production potential and poor disease resistance are genetically related, then the offspring of high performing breeding animals will tend to suffer from more disease and hence under-perform in the low-health commercial tier compared to offspring of 'worse' breeding animals which are less susceptible to diseases.

Frank *et al.* (1998) reported significant genotype × health environment interactions when comparing three terminal sire lines of low, medium and high genetic potential for percent lean, the medium line being the result of crossing the high and low lines. Pigs from these lines were raised in either a low (conventionally weaned into an all-in, all-out nursery building) or high (early weaned into a cleaned, disinfected building) health status environment. The genotype with the low genetic potential for percent lean performed best in a low health status environment, while in a high health status environment the three genotypes were similar.

The inclusion of one or more indicators of disease resistance in the selection objective in the breeding herd could have an impact on reducing G×E. It would be necessary that the trait was heritable and genetically correlated with disease resistance and production in the commercial herd. If disease resistance were to be included in a genetic evaluation system it would be necessary to know the genetic relationships between the disease resistance trait(s) and performance traits in both the nucleus and commercial tiers. This is necessary in order that the genetic merit of performance in the commercial tier can be calculated and used to rank breeding animals. In this situation the genetic merit of the commercial animals for disease resistance traits is not necessarily of interest. The changes in disease resistance will be reflected in the increased production.

The genetic control of susceptibility to infection

Kennedy and Moxley (1980) reported a heritability of 0.12 for susceptibility to atrophic rhinitis, which is caused by a viral infection. They reported a very low heritability for the severity of the infection (0.03) and no significant between breed differences for either incidence or severity of atrophic rhinitis infections. Breed differences have been reported for resistance to respiratory diseases (Lundeheim, 1979) and porcine reproductive and respiratory syndrome (PRRS) virus (Halbur *et al.*, 1998). There is anecdotal evidence of lines of pigs that show resistance to the African swine fever virus, and bush pigs that are tolerant to the infection.

The genetics of resistance to *Escherichia coli* infections have been widely studied. Strains of *E. coli* exist which carry different adhesion factors. These adhesion factors bind to receptors in the gut of the pig, from where the bacteria are able to reproduce and cause various diseases. It follows that a pig that does not have receptors for a specific *E. coli* adhesion factor will not develop diseases from infections by that *E. coli* strain.

Neonatal diarrhoea is caused by the *E. coli* K88 strain of bacteria. The genetic basis for resistance to neonatal diarrhoea was proposed by Sellwood *et al.* (1974). Gibbons *et al.* (1977) demonstrated recessive inheritance of resistance to neonatal diarrhoea. Gibbons *et al.* (1977) did not find a relationship between production traits and the susceptible

genotype, and hypothesised that it remains in the population as a result of passive immunity conferred on the piglet through the dam's colostrum. Edfors-Lilja *et al.* (1986) found that susceptible animals grew faster and more efficiently than the resistant genotype. The maintenance of the gene for susceptibility is probably due to a combination of passive immunity, selective advantage for production traits and the maintenance of high health breeding herds.

Resistance to post-weaning diarrhoea and oedema disease is due to a recessively inherited allele of a gene encoding receptors for *E. coli* F18 (Bertschinger *et al.*, 1993). This gene has been mapped to a region on chromosome six (Vögeli *et al.*, 1996) and a candidate gene has been proposed. This gene is *FUT1*. A mutation in this gene has been reported which was always associated with lack of F18 receptors (Meijerink *et al.*, 1997). A polymerase chain reaction test for this mutation and its use in breeding schemes has been patented (Meijerink *et al.*, 1997).

The major histocompatability complex (MHC) is a very gene-dense region of the genome that has been shown to be associated with disease resistance and immune response in many species. In pigs the MHC is known as the swine leucocyte antigen complex (SLA). Lunney and Butler (1998) have recently reviewed the immunogenetics of the pig, including the SLA. They list a range of immune, cellular and disease response parameters which have been shown to be associated with the genes of the SLA. The disease resistance parameters listed included response to infection by the *Trichinella spiralis* parasite and occurrence of melanomas.

The genetic control of immune response

Immune responses can be measured either *in vivo*, e.g., by challenging the pig by vaccination and then taking blood samples for analysis later, or *in vitro*, by running laboratory tests on blood samples taken from healthy pigs. The vast range of challenges available makes the comparison of results across studies difficult, since the traits measured frequently vary in some aspect of the test applied.

Breed differences have been reported for both *in vivo* and *in vitro* measures of immune response. Rothschild and co-workers found significant breed differences in antibody response to pseudorabies (Rothschild *et al.*, 1984a) and *Bordetella bronchiseptia* (Rothschild *et al.*, 1984b) vaccinations. *Bordetella bronchiseptia* is the primary causative agent of atrophic rhinitis. Meeker *et al.* (1987a) found little or no heterosis for antibody response to pseudorabies and *B. bronchiseptia* vaccinations.

Buschmann *et al.* (1985) reported significant breed differences in antibody response to joint immunisation with tetanus toxoid and ovalbumin. They also reported breed differences for a range of *in vitro* measurements, including lymphocyte proliferation, percentages of different cell-types and phagocytic (cell killing) activity.

Within-breed genetic variation has also been demonstrated. Low to medium heritability estimates have been reported for response to vaccination (Rothschild *et al.*, 1984a and 1984b; Meeker *et al.* 1987b). The importance of the maternal environment upon these traits has also been demonstrated, particularly for young weaners (Meeker *et al.*, 1987b).

In vitro immunological assay traits have also been shown to be heritable (Edfors-Lilja *et al.*, 1991, 1994b; Mallard *et al.*, 1998). Heritability estimates for these traits are generally in the low to medium range. The relationship to actual disease resistance of immune response traits remains unclear. Edfors-Lilja *et al.* (1994a, 1998) have identified a number of significant and putative quantitative trait loci (QTL) for immune response traits.

Immune response parameters have also been shown to be associated in part with the SLA (Rothschild *et al.*, 1984b, Buschmann *et al.*, 1985).

Genetic improvement without molecular genetic information

Morris (1990) outlined two philosophies of selection for disease resistance that are prevalent in agriculture in New Zealand. In the first of these (Type 1), as used in the New Zealand dairy industry, selection is on production traits and resistant animals are

assumed to be among this group. The alternative approach (Type 2) used in some New Zealand sheep group breeding schemes includes the response to specific diseases in the selection objective.

Type 1 selection assumes that the animals that are the best producers will include the animals that are resistant to disease, irrespective of any genetic correlations between disease resistance and production traits. That is to say, healthy animals will tend to produce better than diseased animals and will therefore tend to be selected. The problem with this approach in the pig context is that selection for production traits in pigs is frequently carried out in high health herds with considerable veterinary input. As a result, there is likely to be little disease challenge to the animals under selection and hence no selection pressure placed on disease resistance traits (Rothschild, 1998).

In the Type 2 approaches of Morris (1990) some form of disease challenge is required in order that disease resistance can be assessed. This challenge may be made to either the animal itself, its siblings or clones of the animal (Rothschild, 1998). Challenging the animal itself is likely to result in some production loss. Challenging siblings or clones would permit repetition and hence increased accuracy of the selection for disease resistance. Use of clones would be best, since the performance of any clone directly reflects the performance of the genotype of the animal itself. However it is likely to remain prohibitively expensive for some time.

Survival analysis

One method by which Type 2 selection could be carried out in pig populations would be via survival analysis of either sow longevity, culling reasons or disease occurrence. Specific challenges would not need to be made, data could be collected on multiplier or commercial animals to provide information on the breeding parents or more likely, because of the time lag, their offspring in the breeding herd. Data on survival traits has a special form and requires special statistical tools. Ducrocq and Sölkner (1998) have provided a set of computer programs designed to analyse survival data that incorporate genetic effects. Recently, this methodology has been used in the analysis of pig data.

Yazdi (1999, UK Pig Breeders Round Table, unpublished) reported on an analysis of sow longevity (in days) in Swedish Landrace and Large White populations. Heritability estimates for these traits ranged from 0.1 to 0.25 and from 0.2 to 0.3 in the Landrace and Large White breeds, respectively, depending upon the model fitted. This implies that genetic progress could be achieved in these breeds for sow longevity.

Data on occurrence of specific diseases in a central boar test station (Henryon, 1999, UK Pig Breeders Round Table, unpublished), revealed heritabilities on the underlying scale of between 0.1 (for first diagnosis of any disease) to 0.26 (for first diagnosis of diarrhoea). This study also detected differences between breeds in the chance of getting any of the diseases studied.

Selection for components of immune response

The use of selection on immune response parameters to improve disease resistance has been suggested (Buschmann, 1985; Warner *et al.* 1987). Wilkie *et al.* (1998) reviewed the findings of a selection experiment for an index of immune response traits. Four traits were selected based on their genetic variability and the genetic relationships among them. Two of these traits are involved in antibody responses and two are involved in cellular immunological responses. It was intended that both aspects of the immune response would be improved simultaneously. High and low immune response lines and a control group were maintained for eight generations of selection on BLUP estimated breeding values. The lines diverged until generation three, by which time the high and low lines differed by approximately one phenotypic standard deviation for the index. Little response was observed after generation three and this is thought to be due to inbreeding.

Pigs selected for increased immune response showed greater antibody responses to a range of antigens, both in terms of the quantity and avidity of antibodies produced. When infected with *Mycoplasma hyorhinis*, the high line produced more antibodies which is associated with increased protection against infection. Post-mortem investigations

revealed more evidence of peritonitis and pleuritis in low line pigs, but more arthritis in the high line. The arthritis may be the result of an over sensitive immune response.

The high line pigs reached market weight at least 10 days sooner than low and control line pigs. This may reflect a lower level of subclinical disease in this line or correlated genetic changes in growth traits. This experiment has demonstrated the use of an immune response index as a selection tool. The nature of the relationships between the immune response and disease resistance and production still needs to be elucidated. Work to investigate the increase in arthritis is under way.

Genetic improvement using molecular genetic approaches

There are a number of ways that molecular information can be used in genetic improvement programmes. Other papers in this symposium cover these in more detail (Goddard, 1999; Kerr *et al.*, 1999). Molecular genetic information can either be used to give more accurate selection decisions for a trait that can be, or already is, recorded or to provide a method of selecting for a trait that would not otherwise be practical to select for. Traits associated with disease resistance and immune response would be appropriate for both of these approaches.

Using molecular information in selection decisions

Visscher and Haley (1998) cite disease resistance as an ideal area for the use of marker assisted selection. This is a result of disease resistance traits being expensive and difficult to record in commercial populations. Using genetic marker information would be an effective and hopefully cheap way to improve these traits. Quantitative trait loci have been reported for immune response traits (Edfors-Lilja *et al.*, 1998) and a genetic test for the FUT1 candidate gene for *E. coli* K88 resistance has been developed and patented (Meijerink *et al.*, 1997). Both selection for immune response QTL marker genotypes and genotyping for resistance to a specific disease have the problem of identifying the associated changes in production traits and other areas of health. This may be more easily addressed for the known genetic mutation, where the effect of a change to a single gene product needs to be studied.

Moving disease resistance genes into a population of interest

It seems likely that different genes or QTL associated with disease resistance will be found in different breeds. There is evidence of breed differences for disease resistance traits (Lundeheim, 1979; Kennedy and Moxley, 1980; Halbur *et al.*, 1998). Edfors-Lilja *et al.* (1998) have identified QTL for immune response traits in a wild pig-Swedish Yorkshire cross.

If QTL for disease resistance can be discovered in one line or breed, then it is possible to introduce the desirable sections of the chromosome into any line of interest via a process known as *introgression*. A series of backcrosses with the breed of interest can achieve a very low proportion of the genome originating from the breed with the QTL. How can transfer of the piece of DNA of interest be ensured? If markers exist close to the QTL, then by selecting the parents for the next backcross based on marker genotype the QTL of interest should also be carried with it. This process is termed *marker-assisted introgression*. The success of this depends upon the proximity of the markers to the QTL; the closer together they are on the chromosome the less likely they are to become separated by recombination during meiotic cell division.

Assessing strategies for the genetic improvement of disease resistance

Many of the techniques discussed in this paper are relatively expensive compared to ordinary selection procedures for production traits. In most cases some form of laboratory work is required in order to obtain either immune response phenotypes or marker (or actual gene) genotypes. Before instigating an improvement program for disease resistance it therefore makes sense to consider the outcome of any particular

strategy. Mackenzie and Bishop (1998) proposed a group of simulation models for assessing the outcome of a genetic improvement strategy in terms of the epidemiology of the disease in question. They call these models genetic epidemiological models (GEMs).

Genetic epidemiological models depend on epidemiological parameters of the diseases in question, namely the basic reproductive rate of the infective agent (R_0), the proportion of animals ever infected and the maximum proportion of animals infected. By modeling genetic change in the population by some method (e.g., traditional or marker assisted selection, introgression) the changes in the epidemiological parameters can be used to answer specific questions.

At the 1999 Pig Breeders Round Table in the UK (no published proceedings), Bishop and Mackenzie presented an example of the use of stochastic GEMs for the case of transmissible gastroenteritis. This disease was chosen, as estimates of the required epidemiological parameters were available. The results of their studies using GEMs suggest that, in general, selection is feasible where either fast genetic progress can be made (e.g., with a high heritability for disease resistance or with accurate marker information) or R_0 is low. Also, when introgressing resistance genes into a population it is not necessary to take these genes to fixation in the population. This is a result of the dynamics of animals infecting one another, such that once a certain proportion of the animals are resistant then the population is effectively protected (*cf.* vaccination in man).

The use of GEMs does not relate to selection for immune response traits, since any change is likely to affect resistance to a multitude of diseases all with different epidemiologies. However, selection for immune response could be partially assessed through the relationship with production in the commercial population. In their selection lines, Wilkie *et al.* (1998) observed higher rates of growth in the line selected for increased immune response.

Conclusions

Although no selection has been applied to disease resistance in pigs in the past other than natural selection, the situation now exists whereby new technologies, both genetic and immunological, allow consideration of these traits in breeding objectives. Interest in disease resistance traits of pigs is increasing as the voices demanding a reduction in the use of antibiotics in livestock production are getting louder. A lot of research is currently under way which may help to address questions about the effects of selecting for disease resistance as well as identifying the underlying genetic mechanisms of resistance.

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Symposium continued on next page

SYMPOSIUM CONCLUSIONS

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The aim of every pig breeder is to select individual animals that maximise profitability of the overall production system. At the beginning of this symposium, Goddard (1999) described how the formal definition of a breeding objective allows selection of animals with the highest breeding value for profit. It is important to note that the breeding objective includes all traits that affect profitability of pig production. However, breeding objectives often do not include traits that are difficult to measure. Traits describing disease resistance of a pig are often not included in the breeding objective despite the fact that pig diseases cost the United States 1.5 billion US dollars annually (Rothschild, 1999) as pointed out by Crump (1999). Crump (1999) described two pathways for the improvement of genetic resistance to disease; reduction of the susceptibility of animals to infection, and improvement of the immune response to an infection. New developments in molecular genetics and statistical computing packages allow breeding for disease resistance through both pathways. Finally, Crump (1999) also explained how different breeding strategies for disease resistance could be evaluated through simulation models (ie. genetic epidemiology models).

In contrast to disease resistance traits, many performance traits are easy to measure. The development of Best Linear Unbiased Prediction (BLUP) technology allows use of performance records of the breeding animal itself, and its relatives, to estimate a breeding value for an animal. However, Goddard (1999) explained how new information obtained from DNA technology provides new sources of information about a pig's genetic value. This new information can be combined with existing phenotypic information to estimate the breeding objective for an animal. Goddard (1999) quantified the benefits of utilising additional DNA information in selection indices. Information obtained from DNA technology is most useful when it explains a large proportion of the genetic variation in profit through traits whose conventional EBVs have a low accuracy.

Microsatellites are highly variable and therefore informative DNA markers. Moran (1999) showed how microsatellites have been used for parentage testing and analysis of breed relationships. In addition, microsatellites have been critical to the development of detailed genetic maps through linkage mapping. Linkage maps and highly polymorphic markers are essential for the mapping of quantitative trait loci (QTL). Results from an Australian QTL scan based on a commercial population indicate that QTLs can be useful in practical pig breeding in Australia. Moran (1999) described current use of major genes influencing disease resistance, performance traits and litter size.

In order to include information about genes with a large effect a number of statistical challenges have to be met. Kerr *et al.* (1999) described statistical difficulties of segregation analysis; a statistical tool which identifies the existence of a major gene without using any molecular information. Recently, a software package called "The Gene Detective" has been developed which will aid detection of major genes through DNA assays. In addition, Kerr *et al.* (1999) discuss the logistics of using marker information in practical pig breeding and how this additional marker information is combined with phenotypic information to estimate the breeding value for profit of an animal.

Practical pig breeding will become more complex which requires close collaboration of pig breeders and scientists from a number of disciplines. However, Australian pig breeders have shown during the past ten years that they are not afraid of adopting new technologies, which aid them in improving the profitability of pig production.

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PRE-FARROWING POSTURE AND BEHAVIOUR OF GILTS SELECTED FOR COMPONENTS OF EFFICIENT LEAN GROWTH

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Selection for components of efficient lean growth rate has generated responses in reproductive performance of gilts (Kerr and Cameron, 1995). Pre-farrowing behaviour of gilts was examined in the current study to determine if particular selection strategies were associated with specific behaviours immediately prior to farrowing which may have contributed to the responses in reproductive performance.

The study included 123 Large White gilts from lines divergently selected over seven generations for daily food intake, lean food conversion efficiency, lean growth rate on *ad libitum* feeding (LGA) and lean growth rate on restricted feeding (LGS) (Cameron and Curran, 1994). Gilts were housed in slatted farrowing crates with no bedding for three days prior to farrowing and video images of gilts' behaviour were recorded for 2 h prior to farrowing. The binomial data were analysed with a generalised linear model and a logit link, with the relationship between the proportion of time (p) exhibiting a behaviour and the logit score (x) equal to $p = e^x/(1+e^x)$.

The proportions of observations for postures were: lying 0.8, standing 0.1 and sitting 0.1; and for behaviours were: alertness 0.6, restlessness 0.2, nesting 0.1 and sleeping 0.1. High LGS gilts adopted the lying posture earlier than low LGS gilts with a higher ($P \leq 0.05$) proportion of high LGS gilts observed lying up to 0.5 h before farrowing (0.96 vs 0.72) (logit score: 3.2 vs 0.9, sed 0.60). High LGS gilts spent a higher proportion of time lying with the udder facing the creep in the period 2-1.5 h before farrowing than gilts in other selection lines (0.77 vs 0.29) (logit score: 1.2 vs -0.9, sed 0.49).

The most common behaviour of gilts was alertness, which proportionally increased from 0.45 to 0.62 (logit score: -0.18 vs 0.49, sed 0.12) during the observation period. The proportion of time in alert behaviour was lower (0.38) in the low LGA line 0.5-0 h before farrowing than in the other selection lines. Nesting behaviour decreased from 0.11 to 0.0 (logit score: -2.1 vs -3.6, sed 0.24) during the observation period, with high LGS gilts exhibiting a significantly lower level of nesting behaviour than the other lines (0.02 vs 0.08) (logit score: -4.0 vs -2.4, sed 0.57).

Posture and behaviour of the high LGS gilts suggested that they had completed nesting behaviour earlier and adopted the lying posture earlier than low LGS gilts. Divergent selection on DFI, LFC or LGA was not associated with consistent responses in gilt posture and behaviour traits prior to farrowing, although responses in reproductive performance were detected with selection on DFI and LFC. Either genetic correlations between posture and behaviour traits prior to farrowing and performance test traits in the selection criteria or heritabilities of gilt pre-farrowing posture and behaviour traits were not significantly different from zero. Selection strategies for components of efficient lean growth rate, based on *ad libitum* feeding, can focus on production traits without anticipating correlated responses in pre-farrowing posture and behaviour traits of gilts.

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POSTURE AND BEHAVIOUR AND LOCATION OF PIGLETS DURING AND AFTER FARROWING OF GILTS SELECTED FOR COMPONENTS OF EFFICIENT LEAN GROWTH

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Differences in maternal behaviours between sows of breeds with high or low lean growth rate have been reported (Meunier-Salaun *et al.*, 1991). In the current study, gilt posture and behaviour and piglet locations during the farrowing period and the 2 h post-farrowing period were measured to determine if particular selection strategies were associated with maternal behaviours which may have contributed to the observed responses in reproductive performance.

The study included 139 Large White gilts from lines divergently selected over seven generations for daily food intake, lean food conversion and lean growth rate on *ad libitum* or restricted (LGS) feeding (Kerr and Cameron, 1995). Gilt posture and behaviour traits and the locations of their piglets were determined from video recordings assessed at 5 min intervals. Gilts were housed in slatted farrowing crates with no bedding. The binomial data were analysed with a generalised linear model and a logit link and the relationship between the proportion of time (p) exhibiting a behaviour and the logit score (x) is:

$$p = e^x / (1 + e^x)$$

During farrowing, the proportion of time gilts spent lying and changing posture were 0.74 and 0.19, respectively, with an alert (0.69) or restless (0.19) behaviour exhibited. High LGS line gilts spent significantly more time lying on their side (0.96 vs 0.80; logit score: 3.2 vs 1.4, sed 0.5) and less time changing posture (0.05 vs 0.30; logit score -3.0 vs -0.8, sed 0.4) than low LGS gilts. Gilts proportionately spent 0.94 of the time lying. Gilts were alert (0.81), or restless (0.05) or sleeping (0.08). There were no significant line differences in gilt post-farrowing behaviour or posture.

The proportion of observed piglets relative to the number born in the litter averaged 0.41 during farrowing and 0.51 in the 2-hour post-farrowing period. Piglets spent most time at the udder (0.55 during farrowing and 0.87 during 2 hours post-farrowing) and at the gilt's head, feet and vulva (0.24) during farrowing. High LGS piglets spent significantly less time (0.06 vs 0.15; logit score -2.8 vs -1.7 sed 0.5) at the gilt's head, back and vulva or at the creep during farrowing. In the post-farrowing period, there were no significant differences between selection lines in the time spent by piglets at the udder.

There was greater variation in gilt posture and behaviour during farrowing than in the post-farrowing period. Divergent selection for components of efficient lean growth rate, based on *ad libitum* feeding was not associated with correlated responses in gilt behaviour and posture or in piglet location. However, relative to low LGS line gilts, selection for high LGS had positive effects on gilt behaviour and posture during farrowing; characteristics which are thought to be beneficial for the welfare of the gilt and her piglets.

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PREDICTION OF FARM GROWTH RATE FROM PARENTAL ESTIMATED BREEDING VALUES

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The ranking of pigs on estimated breeding values (EBVs) from their parental average has been shown to be well correlated with the rankings of on-farm performance (Hermesch *et al.*, 1997). This paper examines if on-farm growth rates, predicted from parental EBVs of both sires and dams from the National Pig Improvement Program (NPIP) perform as expected.

On-farm growth rates (GR) of pigs performance tested in 1998 were collated from 10 herds participating in NPIP (Macbeth, 1999). Across herd sire EBVs ($EBV_{sire,dam}$) for growth rate were calculated using PEST (Groeneveld, 1990) from data submitted prior to the 1998 test period. Linear regressions of progeny growth rates on parental EBVs were determined while accounting batch and sex as fixed effects from 9,131 Large White, 4,135 Landrace and 2,102 Duroc records (Table 1).

Table 1. Regressions for parental EBVs on progeny growth rate (GR).

	Parental EBVs		
Breed	EBV_{sire}	EBV_{dam}	$EBV_{(dam+sire)/2}$
Large White	0.44 ± 0.03[a]**	0.54 ± 0.04**	1.01 ± 0.05**
Landrace	0.35 ± 0.04**	0.30 ± 0.05**	0.73 ± 0.07**
Duroc	0.38 ± 0.14*	0.41 ± 0.08**	0.86 ± 0.14**

Significance of regression *(P≤0.05), **(P≤0.01). [a]Standard error.

The expectation of offspring performance predicted from an EBV_{sire}, is half the EBV_{sire}, as the offspring receives half of its genes from its sire. The regressions in Table 1, for both Large White and Duroc, met this expectation but Landrace fell below 0.5 (P<0.01). The regressions were similar for dams, (EBV_{dam}).

The expectation of offspring performance is the average of the parental EBVs, ($EBV_{(dam+sire)/2}$) and the regressions for Large White and Duroc are not significantly different from 1.0 so meet this expectation, but in 1998 Landrace had a regression coefficient less than 1.0 (P<0.01). Landrace grown in the previous year (1997) did meet this expectation 1.10 + 0.07 $EBV_{(dam+sire)/2}$. It is unlikely that deviations from expectation are the result of differences in heritability as they are similar for all three breeds (Del-Bosque-Gonzalez *et al.*, 1999). In general progeny performance predicted from their sire EBVs behaved close to expectation.

It is particularly important for producers to understand the commercial importance of EBVs, particularly sire EBVs of commercially available AI boars. Using AI from sires with high EBV_{sire} will improve herd profitability. A boar with a +70g/d EBV advantage will produce progeny that will have an average improved growth rate of +35g/d. A 35g/d improvement represents an increased return of $90/year for every sow. A 200 sow herd would be $90 x 200 = $18,000 better off each year. The returns may not be realised from the use of any one individual boar, but by using several boars over a period of time the average on-farm improvement will be close to that predicted

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SELECTION FOR EFFICIENT LEAN GROWTH UNDER RESTRICTED FEEDING: 2. GROWER PERFORMANCE

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Large White pigs, selected by McPhee *et al.* (1988) for lean growth rate on a restricted feeding scale displayed a reduction in fat and an increase in lean growth rate, when compared with an unselected control on *ad libitum* feeding. There were also increases in appetite and apparent rate of glycogen depletion with fasting in the selected pigs (McPhee *et al.*, 1995). These responses were consistent with a change in the partitioning of metabolizable energy towards lean and away from fat deposition and an increase in maintenance heat production.

To examine this hypothesis further, two lines of 36 sows and six boars were newly formed by sampling within Large White litters. These are being divergently selected for high and low growth rates over a 6-week period starting at 50 kg live weight (LW). Over this test period, pigs of both lines are fed a grower diet at a fixed intake amounting to about 80% of the *ad libitum* intake of the unselected base animals. Measurements are made of live weight gain and ultrasonic P2 backfat thickness. Selection intensities are two out of 24 males and six out of 24 females for high or low live weight gain only. At 50 kg LW, litters are divided into two samples, one for performance testing on restricted feeding, and the other for comparing the lines on *ad libitum* feeding also over 6 weeks. Data from the progeny of first to third generation selected parents were subject to REML analysis (Genstat 5, 1997) with line, feed and sex and their interactions as fixed, and batch and sire within line as random effects. In Table 1 the means of the high and low lines fitted for each of the measured traits are presented.

Table 1. Means of traits of growing pigs of the high and low selection line measured when fed at restricted and *ad libitum* food intakes.

Food intake	Restricted		*Ad libitum*		
Selection Line	High	Low	High	Low	sed[a]
Weight gain (kg/d)	0.77	0.72	0.95	0.93	0.01
Food intake (kg/d)	2.11	2.10	2.66	2.77	0.002
Food conversion ratio	2.76	2.95	2.83	3.01	0.03
Backfat at P2 (mm)	11.5	12.1	12.1	12.7	0.02
Number of pigs	747	616	278	200	

[a]Standard error of difference

Relative to the low line, the high line grew faster with a lower food conversion ratio and backfat. On *ad libitum* feeding, intake was also reduced. This contrasts with an increase in intake found by McPhee *et al.* (1988) when selection was for lean growth on restricted feeding. The contributions of changed energy partitioning favouring lean over fat and reduced maintenance to the responses here have yet to be determined.

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SELECTION FOR EFFICIENT LEAN GROWTH UNDER RESTRICTED FEEDING: 3. SOW PERFORMANCE

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Two lines of 36 sows and six boars described by McPhee *et al.* (1999) were formed from sampling within litters of an outbred Large White population free of the halothane gene. These animals are being divergently selected for high and low growth rate on restricted feeding over a 6 week period starting at 50 kg live weight (LW).

This paper reports a preliminary comparison of sow littering performance between these two lines. The herd was farrowed in batches and these included 12 sows from each line. Lactating sows were fed *ad libitum* a 14 MJ DE, 0.55 g/MJ lysine diet, and feed intakes were measured from 7 days to 35 days after farrowing. Piglets were cross fostered among sows of the same line prior to 7 days of age. Data from the first to third selected generation were subject to REML analysis (Genstat 5, 1997) with line, batch and parity as fixed effects. Sow within line was the random effect for all traits except for piglet birth weight where piglet was an additional random effect. Number of piglets suckled was a covariate for sow food intake. The means fitted for each of the traits measured for the high and low line sows are given in Table 1.

Table 1. Means of traits of the high and low selection line sows measured on *ad libitum* feeding.

Trait	High line	Low line	sed[a]
Pigs born alive	10.9	10.1	0.44
Pigs weaned	8.9	8.9	0.24
Birth weight (kg)	1.45	1.39	0.03
Food intake (kg/d)	5.92	6.00	0.21
Mating weight (kg)	191	193	3.9
Farrowing age (d)	654	644	13
Number of litters	188	150	

[a]Standard error of difference

High line piglets were significantly heavier at birth than low line piglets. Otherwise, there were no differences between lines in any of the traits. The apparently higher number of piglets born alive in the high than the low line did not reach significance. These results show that food intake differences between the lines recorded by McPhee *et al.* (1999) for growers on *ad libitum* feeding were not carried through to sows during lactation.

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HERITABILITY ESTIMATES FOR CARCASE TRAITS OF PIGS RECORDED UNDER *AD LIBITUM* AND RESTRICTED FEEDING

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In Australia, genetic improvement of efficient lean meat growth is based on *ad libitum* feeding in most breeding programs. Selection experiments have shown that restricted feeding might be the preferable feeding regime in order to select for efficient lean meat growth (Cameron and Curran, 1995; McPhee *et al.*, 1988). These selection experiments were conducted in research herds and may not reflect commercial conditions. Therefore, a project was initiated to estimate genetic parameters for economically important traits recorded under both feeding systems in a commercial herd. The aim of this study was to compare heritabilities and variance components for carcase traits recorded under both feeding regimes in a commercial herd.

Abattoir data were available for 4,464 Large White and Landrace animals with offspring of 185 sires and 2,107 dams evenly distributed over both feeding regimes (the mean *ad libitum* feed intake was 33 MJ ME/d, restricted feeding was a 11% reduction of *ad libitum* intake, the test live weight range was 80-105 kg). The traits were back leg weight (BLW), and weight of the trimmed back leg (HAMB) and the slash boned ham (HAM). Fixed effects included management group of the animal and breed. Heritabilities were estimated using the program VCE4 (Groeneveld, 1998).

Heritability estimates obtained from univariate analyses were moderate to high for BLW, HAMB and HAM recorded under *ad libitum* feeding (Table 1). In comparison, heritabilities were significantly lower for BLW and HAMB under restricted feeding. These lower heritability estimates were caused by a reduction in the additive genetic variance. Only HAM had a similar heritability estimate under both feeding regimes.

Table 1. Heritabilities (h^2) and litter effect estimates (c^2) both with standard errors (se) along with variance components for back leg weight (BLW), and weight of the trimmed back leg (HAMB) and slash boned ham (HAM).

Feeding regime	Trait	N	h^2 (se)	c^2 (se)	σ^2_a*	σ^2_c	σ^2_e
Ad libitum	BLW	1970	0.28 (0.05)	0.07 (0.03)	0.31	0.08	0.74
	HAMB	1269	0.46 (0.06)	0.02 (0.03)	0.35	0.01	0.40
	HAM	680	0.27 (0.08)	0.18 (0.06)	0.17	0.11	0.34
Restricted	BLW	1714	0.14 (0.04)	0.13 (0.04)	0.14	0.13	0.71
	HAMB	995	0.21 (0.05)	0.05 (0.05)	0.16	0.04	0.55
	HAM	694	0.30 (0.08)	0.11 (0.07)	0.15	0.06	0.30

*σ^2_a: additive genetic variance; σ^2_c: variance due to litter effect; σ^2_e: residual variance.

The reduction in feed intake of 11% in the restricted group in comparison to the *ad libitum* group did not reduce genetic variation for HAM. Therefore, pigs under restricted feeding were able to express their potential for lean meat growth. However, restricted feeding reduced variation and heritabilities for BLW and HAMB indicating a reduction in fat deposition and possibly bone growth under restricted feeding.

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PIG RESEARCH AND DEVELOPMENT CORPORATION

GENETIC CORRELATIONS BETWEEN CARCASE TRAITS OF PIGS PERFORMANCE RECORDED UNDER *AD LIBITUM* AND RESTRICTED FEEDING

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A genotype by environment interaction exists when the genetic correlation between the same trait recorded in two environments differs from one. Different feeding systems also constitute different environments; e.g., *ad libitum* versus restricted feeding. The aim of this paper is to present genetic correlations between carcase traits recorded under *ad libitum* and restricted feeding in a commercial herd in Australia.

The carcase traits included in this paper were back leg weight (BLW) and weight of the trimmed back leg (HAMB) and slash boned ham (HAM) defined as different traits under each feeding regime. Information about feeding regimes, data structure, models and heritabilities were presented in Hermesch *et al.* (1999a). Genetic correlations were estimated in a single six-trait analysis using the program VCE4 (Groeneveld, 1998).

Litter mates were tested under both feeding regimes in order to obtain genetic correlations. Genetic correlations between the same trait recorded under *ad libitum* and restricted feeding (underlined in Table 1) were not significantly different from one for all three traits. Consequently, genetic correlations between trait combinations were similar when compared within and across feeding systems ranging from 0.72 to 0.96. Environmental correlations ranged from 0.65 to 0.91.

Table 1. Genetic correlations for back leg weight (BLW), ham weight including bone (HAMB) and excluding bone (HAM) recorded under *ad libitum* and restricted feeding.

	A_HAMB	A_HAM	R_BLW[1]	R_HAMB	R_HAM
A_BLW	0.90[2]	0.83	<u>0.99</u>	0.88	0.72
A_HAMB		0.91	0.91	<u>0.99</u>	0.81
A_HAM			0.89	0.96	<u>0.98</u>
R_BLW				0.91	0.81
R_HAMB					0.89

[1]A_trait: trait recorded under *ad libitum* feeding; R_trait: trait recorded under restricted feeding. [2]Standard errors for genetic correlations ranged from 0.02 to 0.09.

Genotype by feeding regime interactions have been found for some performance traits indicating the use of restricted feeding as the preferred method for selection of efficient lean meat growth (Hermesch *et al.*, 1999b). However, no genotype by feeding regime interaction exists for these carcase traits. Therefore, in breeding programs BLW, HAMB and HAM can genetically be regarded as the same trait under both feeding regimes.

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ACROSS-HERD GENETIC PARAMETER ESTIMATES FOR BACKFAT THICKNESS (P2) AND AVERAGE DAILY GAIN

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Accurate prediction of the genetic merit of animals requires appropriate estimates of genetic parameters. The National Pig Improvement Program (NPIP) currently uses literature estimates of genetic parameters that are not specific to the NPIP populations and are predominantly from within-herd studies. The current study estimated genetic parameters from data on all NPIP herds.

Backfat thickness at P2 (BF) and average daily gain (ADG) were recorded in three breeds of the NPIP. There were 20,356 (9 herds), 43,151 (9 herds) and 74,294 (11 herds) animals with records for the Duroc (Du), Landrace (Lr), and Large White (Lw) breeds. Data was analysed with the ASREML program (Gilmour *et al.*, 1998). Within-breed analyses were performed using the same model as del-Bosque-Gonzalez *et al.* (1999) with no genetic grouping, but common environmental effects were included in the model as random effects. There were 7,225, 14,688, and 26,063 litter effects for Du, Lr and Lw breeds, respectively.

Table 1. Genetic parameter estimates (± SE) for backfat and average daily gain in Duroc (Du), Landrace (Lr) and Large White (Lw) breeds.

	Backfat thickness (P2)			Average Daily Gain			
Breed	$h^2 \pm SE$	$c^2 \pm SE$	σ_P^2	$h^2 \pm SE$	$c^2 \pm SE$	σ_P^2	$r_g \pm SE$
Du	0.44±0.02	0.06±0.006	3.27	0.31±0.02	0.11±0.007	3538	0.14±0.051
Lr	0.53±0.02	0.05±0.004	4.08	0.28±0.01	0.11±0.005	3294	0.09±0.034
Lw	0.49±0.01	0.05±0.003	4.24	0.25±0.01	0.11±0.004	3450	0.04±0.028

h^2, heritability; c^2, litter effect variance as a proportion of the phenotypic variance; σ_P^2, phenotypic variance; r_g, additive genetic correlation between BF and ADG.

Estimates of the heritability for BF and ADG and the genetic correlation between BF and ADG were of the same order of magnitude among the breeds, although there were statistically significant differences between parameter estimates for particular breed-trait combinations. Estimates of the litter effect parameter (c^2) were significantly different from zero for all breeds and traits, and did not differ among breeds.

The results obtained are in close agreement with those from within-herd studies (Hermesch *et al.*, 1999) and with those already in use by the NPIP. Changing the genetic parameters used by the NPIP for genetic evaluations would not be expected to have a large effect upon the predicted breeding values.

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PIG RESEARCH AND DEVELOPMENT CORPORATION

SEQUENCING OF DNA SHOWS MANY PORCINE ENDOGENOUS RETROVIRUSES (PERVs) ARE DEFECTIVE IN WESTRAN INBRED PIGS

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For safety, financial, ethical and practical reasons, pigs are regarded as the best source of organs and tissues for transplantation into humans. The main obstacle to xenotransplantation of organs is hyperacute rejection (Cozzi and White, 1995). Porcine retroviruses have emerged recently as a potential new problem, as they infect human cells *in vitro* (Patience *et al.*, 1998), although there is no evidence that this occurs *in vivo* (Paradis *et al.*, 1999). There is now an embargo in the UK and Australia on xenotransplantation of porcine tissues while this risk is evaluated. The viral envelope is the major determinant of host range and essential for infection. Two main types of pig retrovirus, PERV-A and PERV-B, differing by 320-390 nucleotides within the envelope (*env*) gene, are widely distributed in different pig breeds (Le Tissier *et al.*, 1997), with PERV-A predominant in European pigs (~70%) and PERV-B predominant in Asian (~60%).

The Westran (Westmead transplantation) line is descended from a pair of pigs released on Kangaroo Island in 1803. Captured feral pigs were transferred to Adelaide University for use in biomedical research in 1976 (McIntosh and Pointon, 1981). After being maintained as a very small colony for about 15 years, a pair of full sibs was transferred to Westmead Hospital in Sydney for transplantation research. The core breeding line has been maintained by deliberate full-sib mating up to the current eighth generation. Their highly inbred status has been confirmed by microsatellite homozygosity (data not shown). Endogenous retroviruses have been analysed from a generation six boar. Cloned *env* PCR products (64 clones) were screened by restriction digestion (*Kpn*I and *Mbo*I) and approximately 1.8 kb of single pass insert sequence was read from 18 clones.

Nine types of clones were recognizable by restriction digestion, with 31% classified PERV-A, more consistent with an Asian origin for the Westran pigs. Five restriction types occurred only once, but the most abundant restriction type, a PERV-B, accounted for 41/64 clones. The 18 clone sequences, representing all nine types recognized by restriction digestion, clustered in neighbour joining dendrograms, 9 with PERV-A and 9 with PERV-B published sequences. Two unique clones were recombinants between A and B. The first was 1.4 kb of PERV-A, followed by 0.4 kb of PERV-B. The second was 1.1kb of PERV-B, then 0.5 kb of PERV-A and finally 0.2 kb of PERV-B sequence. These corroborate the recombinogenic potential of retroviruses and highlight the potential danger of interspecies recombination of PERVs and human endogenous retroviruses in xenotransplantation. All PERV-A and two PERV-B sequences (11/18) have stop codons within the envelope protein-encoding region, which would prevent the retrovirus from making full length envelope protein recognizable by cell surface receptor for the virus. If these stop sequences are confirmed, it will show that many PERVs constitute little potential hazard in xenotransplantation.

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EFFECTS OF INTRAUTERINE SEMINAL PLASMA ON RESPONSES OF PIG FOLLICLES TO GONADOTROPHIC HORMONES AND GROWTH FACTORS

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Recent research on the biological effects of seminal plasma (SP) in the female reproductive system has demonstrated the existence of active constituent(s) that elicit inflammatory responses accompanied by elevated cytokine secretion in the endometrium. These effects have been postulated to facilitate embryo growth and implantation (Robertson *et al.*, 1997). Seminal plasma has also been reported recently to advance ovulation in gilts by shortening the interval from the preovulatory luteinizing hormone (LH) surge to ovulation (Waberski *et al.*, 1997).

The hypothesis to be tested is that the ovarian effects of SP treatment are mediated via proinflammatory molecules of endometrial origin that enhance follicle cell responses to gonadotrophic hormones and growth factors. Gilts were inseminated with either 100 ml SP (n=4) or phosphate-buffered saline (PBS) (n=4) at onset of oestrus, ovaries collected at autopsy 36 h later, and follicles aspirated to recover follicular cells and fluid. Granulosa cells (GC) and thecal tissues (TC) from pooled follicles within each gilt were isolated and cultured for 24 h in triplicate or duplicate, respectively. Cultures were performed in serum-free medium (TCM199) ± insulin-like growth factor-I (IGF-I, 10 ng/ml) with or without epidermal growth factor (EGF, 2 ng/ml), porcine follicle-stimulating hormone (pFSH, 10 ng/ml) or human chorionic gonadotrophin (hCG, 5 IU/ml) (Evans *et al.*, 1980). Progesterone secretion by GC and TC was assessed by radio-immunoassay of culture media, and DNA synthesis in GC by measurement of incorporation of ^{3}H-thymidine. Data were analysed by ANOVA with gilts, *in vivo* and *in vitro* treatments, and culture replicates as factors, and significance inferred at $P<0.05$).

In vitro addition of IGF-I and EGF increased ^{3}H-thymidine incorporation in GC above that of control cultures without supplementation (40% and 28% increase respectively, $P<0.01$), whereas pFSH and hCG were ineffective. *In vivo* treatment with SP increased basal *in vitro* ^{3}H-thymidine incorporation in GC 40% above that observed in cells of PBS-treated controls ($P<0.01$) and enhanced their *in vitro* responses to IGF-I and EGF by 60% and 80%, respectively ($P<0.01$).

Mean secretion of progesterone by GC *in vitro* was increased approximately 2-fold by both IGF-I and hCG treatment *in vitro* ($P<0.01$). *In vivo* treatment of gilts with SP did not significantly influence either basal GC progesterone secretion or their secretory responses to these agents. Mean secretion of progesterone by isolated TC of control gilts was increased 2-fold by combined *in vitro* treatment with IGF-I+hCG ($P<0.01$) but not by treatment with either agent alone. *In vivo* SP treatment increased mean progesterone secretion by TC by 2.4-fold over all *in vitro* treatments ($P<0.01$) and enhanced their *in vitro* response to hCG ($P<0.01$).

The observed seminal plasma enhancement of steroidogenic response to hCG was restricted to thecal cells, consistent with a role of inflammatory cells resident in the thecal layer in mediating follicle cell responses. Enhanced follicle growth, indicated by increased follicle size (data not shown) and granulosa cell DNA synthesis, may be a secondary effect of steroids or other paracrine agents of thecal origin on granulosa cells. These preliminary results support the hypothesis that SP treatment of gilts enhances ovulation by altering responses of follicular cells to gonadotrophins and growth factors, although the specific mediators and mechanisms of the SP effects remain unknown.

PIG RESEARCH AND DEVELOPMENT CORPORATION

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SEMINAL PLASMA INDUCES A LOCAL INFLAMMATORY RESPONSE IN THE REPRODUCTIVE TRACT OF GILTS

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In the pig, early embryonic mortality is the principle cause of reproductive loss. The factors determining success of implantation are still largely unknown but are likely to be influenced by the maternal immune response. In rodents, seminal plasma has been shown to evoke an inflammatory response in the uterus, which leads to improved embryo viability through the induction of (1) embryotrophic cytokine expression and (2) immunological tolerance to paternal antigens expressed on the semi-allogeneic conceptus (Robertson *et al.*, 1997). The active moiety in murine and human seminal plasma has now been identified as transforming growth factor beta (TGFβ), produced in the latent form in the seminal vesicle and activated in the female reproductive tract after mating. Artificial insemination in the pig currently involves diluting and/or discarding certain fractions of seminal plasma, a practice that is likely to diminish any priming effect of the active seminal plasma components. The aim of the current studies is to examine the acute effect of seminal plasma proteins on indicators of an inflammatory response in the uterus.

Eight crossbred gilts (Large White x Landrace) of similar age and weight were randomly selected for this study. At onset of gonadotrophin-induced oestrus (Ainsworth *et al.*, 1980), gilts were infused cervically with 100 ml of whole seminal plasma (SP) or saline (PBS). The reproductive tracts were collected post-slaughter thirty six hours after treatment. Uterine weights, uterine luminal fluid volumes, vascular index and CD45 % positivity are shown in Table 1. Vascular index is a visual measurement of the extent of uterine vascularisation. Endometrial tissues from proximal and distal sections of the uterine horns were recovered for immunohistochemical analysis of the resident leukocyte population. Antibody reactivity (% positivity) was quantified by video image analysis (Robertson *et al.*, 1996) using monoclonal antibodies specific for CD45 (MCA1222), MHC class II (MSA3), and macrophages (HB142).

Table 1. Effects of intrauterine infusion of seminal plasma on uterine weight, luminal fluid volume, vascularity and leukocyte infiltration 36 hours after treatment.

Treatment	Uterine weight (g) (mean ± SEM)	Luminal fluid volume (ml) (mean ± SEM)	Vascular index (median)	CD45 (% positivity) (mean ± SEM)
SP	116.89 ± 7.77*	23.25 ± 9.20	++++	73.75 ± 2.88**
PBS	79.18 ± 4.54	6.66 ± 1.22	++	46.62 ± 4.02

*P=0.034, **P≤0.001 (Student's t test).

The finding of increased weight, luminal fluid volume, vascularity and leukocyte content in the endometrium of gilts treated with seminal plasma supports the hypothesis that components of seminal plasma induce a local inflammatory response in the uterus after mating. The infiltrating leukocytes were comprised primarily of macrophages and MHC class II positive cells (data not shown). Future studies will analyse the cytokines induced by seminal plasma treatment to further elucidate the molecular basis of this response and determine whether TGFβ is the active seminal constituent.

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PIG RESEARCH AND DEVELOPMENT CORPORATION

THE EFFECT OF UTERINE PRIMING OF GILTS ON SUBSEQUENT REPRODUCTION

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Research both in Australia (Bischof, 1994) and elsewhere (Murray and Grifo, 1983, Han, 1988) has shown that insemination of oestrous gilts with dead boar semen improves subsequent reproductive performance. This procedure is known as uterine priming. Increases in litter size in the order of 1.3 to 1.8 pigs have been reported. Hughes (personal communication) has reported an improvement in both litter size and farrowing rate, and has suggested that the improvements may extend beyond the first parity.

Nine pig farmers (members of the Bell Pig Discussion Group) allocated 181 gilts to either a treatment or a control group. The gilts in the treatment group were inseminated at the first observed oestrus with dead boar semen supplied by Associated Pig Genetics. At the second oestrus, both the gilts in the treatment group and the control group were mated according to standard practice for the farm. Seventy-five of the gilts were monitored during their second parity. The results are shown in Table 1.

Table 1. Effects of uterine priming on total pigs born and pigs born alive per litter at the first and second parity (mean ± SD).

	Control	SD	Treatment	SD	P value
Parity 1	(106 gilts)		(75 gilts)		
Born total/gilt	10.00	2.72	10.84	2.30	0.030
Born alive/gilt	8.97	2.97	10.08	2.26	0.007
Parity 2	(31 sows)		(44 sows)		
Born total/sow	9.42	3.39	10.09	3.28	0.380
Born alive/sow	9.02	3.45	9.42	2.75	0.590

Treated gilts showed an improvement in first parity litter size, with a significant increase in the number of piglets born in total and born alive ($P<0.05$), equating to increases of 0.84 and 1.11 pigs/litter respectively. The improvement in second litter size was not statistically significant. This trial involved a number of pig farms that made control of confounding variables difficult. However, the improvement in reproductive performance following uterine priming treatment is consistent with the findings of other studies, and the technique has been utilised elsewhere in agriculture, including the cattle, sheep and goat industries (Kumar *et al., 1998;* Nottle *et al., 1997;* Zicarelli *et al., 1997).* Meo and Cleary (1998) quote an average sow replacement rate of 72% per annum, indicating that gilts contribute substantially to the reproductive performance of the herd. Adoption of uterine priming could increase gilt reproductive performance and appreciably increase herd output.

The project was carried out in conjunction with the Bell Pig Discussion Group, Queensland, and Associated Pig Genetics.

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PIG RESEARCH AND DEVELOPMENT CORPORATION

IN VITRO PRODUCTION OF PORCINE BLASTOCYSTS WITH SPERM PASSED THROUGH A FLOW CYTOMETER

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Fluorescence activated cell sorting is the only scientifically validated method of sorting X and Y sperm. Passage through a flow cytometer results in a decrease in motility of sperm which may affect fertility. This experiment was carried out to investigate whether boar sperm which have been stained for flow sorting and passed through a flow cytometer (FACS sperm) are capable of fertilization and producing blastocysts in an *in vitro* embryo production system.

Sperm were collected the day before use, transported to the laboratory and stored at 18°C overnight. The cells were stained with Hoechst 33342 and passed through a Coulter EPICS V flow cytometer modified for sperm sorting (Johnson and Pinkel, 1986). Oocytes were aspirated from 2-4 mm follicles on ovaries collected from pre-pubertal pigs and matured for 44 h in modified oocyte maturation medium 37 (Funahashi *et al.*, 1997). Oocytes were transferred to 100 µl droplets of modified Tyrode's albumin lactate pyruvate polyvinylalcohol solution (Bavister, 1989) + 4 mM caffeine, and fertilized with 80 x 10^3 /ml frozen-thawed epididymal sperm (control) or FACS sperm at four concentrations (10 x 10^3, 20 x 10^3, 40 x 10^3 or 80 x 10^3 /ml). Six hours after fertilization the embryos were washed twice to remove excess sperm and transferred to North Carolina State University 23 medium (NCSU23; Petters and Wells, 1993). On day 5 embryos were transferred to bovine serum albumin-free NCSU 23 + 10% (v:v) fetal calf serum. All stages of the experiment were carried out at 39°C in 5% CO_2 in air. Some oocytes (n=15) were removed 17 h after insemination and assessed for maturation and fertilization by staining with Hoechst 33342 and examination under UV light. The remaining embryos were cultured for 7 days and examined for development to blastocyst stage.

Table 1. *In vitro* fertilization rates and blastocyst production using FACS sperm.

Treatment	Control	FACS sperm			
Sperm concentration	80 x 10^3	10 x 10^3	20 x 10^3	40 x 10^3	80 x 10^3
No of oocytes inseminated	38	47	44	48	50
% Total sperm penetration of matured oocytes	54	20^a	54	50	78^a
% Monospermic penetration of matured oocytes	36	20^b	18^c	25^d	64^{bcd}
% Polyspermic penetration of matured oocytes	18	0^e	36^e	25	14
No. of presumptive embryos cultured to day 7	23	32	29	33	35
No. which developed to expanded blastocysts	2	3^f	8^f	7	6

^{a,b,c,d,e,f}Values in each row with the same superscripts are significantly different (P≤0.05).

These results show that boar sperm which have been stained for flow sorting and passed through a flow cytometer are capable of fertilization of *in vitro* matured oocytes at the same rate as control sperm and that the resulting embryos can develop to blastocysts at the same rate as embryos produced from control sperm.

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PIG RESEARCH AND DEVELOPMENT CORPORATION

THE PROTEIN REQUIREMENT DURING PREGNANCY FOR FIRST PARITY PERFORMANCE OF GENETICALLY LEAN SOWS

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As a consequence of inadequate protein intake during gestation, the sow becomes fat, which in turn reduces voluntary food intake during lactation (Revell *et al.*, 1995). Commercial diets fed to mated gilts and sows are often the same and the relatively low protein content of these diets may be inadequate for the young growing gilt. In this experiment the hypothesis tested was that fertility and lactation performance should be improved by feeding mated gilts a high-protein gestation diet.

Two hundred and ninety-four Large White x Landrace F1 cross gilts were mated in May and allocated to a 12.9 MJ DE/kg diet containing either low crude protein (CP) (139 g CP/kg; 6 g total lysine/kg) or high CP (179 g CP/kg; 10 g total lysine/kg). The mean live weight and ultrasound P2 of animals allocated to the low CP diet was respectively 136 kg and 17.4 mm and 137 kg and 18.5 mm for those allocated to the high CP diet. Animals were individually stalled during gestation and fed 2.2 kg/day. All animals were offered the same diet (14.0 MJ DE/kg; 0.8 g total lysine/kg; 210 g CP/kg) *ad libitum* during a 27 ± 0.1 day lactation. Weaned sows were offered a gestation diet (12.9 MJ DE; 0.6 g total lysine/kg; 140 g CP/kg) *ad libitum* and mated at the first post-weaning oestrus. All animals were fed 2.5 kg/day of this diet during the subsequent gestation period.

Table 1. The effect of protein intake of gilts on live weight and backfat (P2) change during first gestation, and *ad libitum* intake, and live weight and backfat change during a 27 day lactation.

Treatment	During gestation		During lactation			
	LW change (kg)	P2 change (mm)	Sow intake (kg/day)	LW change (kg)	P2 change (mm)	Litter gain (kg/d)
Low protein	42.6	-0.2	5.0	9.9	3.4	1.98
High protein	41.0	-2.0	5.1	10.9	2.7	2.05
SED[1]	0.99	0.21	0.05	0.63	0.18	0.03
Significance[2]	NS	*	NS	NS	*	NS

[1]SED, Standard error difference. [2]NS, mean values not significant, *P<0.01.

There was no significant effect of increasing protein intake during the first pregnancy on reproductive performance of the first parity. The mean farrowing rate (number sows farrowed as a percentage of mated) was 85.0 ± 2.0 % and the mean litter size born alive was 9.7 ± 0.16. The sows fed the high protein diet during first pregnancy deposited less maternal fat (Table 1) than sows fed the low protein diet at a similar body weight. Sows fed the low protein diet were significantly fatter (P<0.01) post-farrowing and mobilised more fat during lactation. However, the magnitude of the difference in body fatness due to low protein intake during pregnancy did not significantly reduce intake or increase the amount of weight lost during lactation. Litter weight gain during lactation was similar between the two treatment groups (Table 1), indicating that feeding more protein during pregnancy did not increase milk production. Subsequent farrowing rate and litter size born live were unaffected by dietary protein intake in the first parity (81.6 ± 2.9% and 10.2 ± 0.24 respectively). Based on this study, the protein intake for lean gilts during their first pregnancy need not exceed 300 g CP/day or 13 g lysine/day for maximum reproductive and lactation performance in the first two parities.

PIG RESEARCH AND DEVELOPMENT CORPORATION

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CIRCADIAN MELATONIN PROFILES IN EUROPEAN WILD BOAR (*SUS SCROFA SCROFA*) AND DOMESTIC PIGS

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The European Wild Boar subspecies (*Sus scrofa scrofa*), from which the domestic pig (*Sus scrofa domestica*) is derived, is a seasonal breeder (Mauget, 1982). A remnant of this seasonality is still evident in domestic pigs. Photoperiodic information is converted into an endocrine signal, in the form of melatonin, which drives seasonal breeding. It has been suggested that attenuation of the melatonin response in domestic pigs may explain the reduced influence of season on reproduction (Green *et al.*, 1996). The aims of this study were to establish that the European Wild Boar in its normal light environment exhibits a circadian pattern of melatonin secretion and to compare this pattern with that of domestic pigs maintained in a light environment typical of domesticity.

This study took place in midsummer in southern Finland and used five pure-bred post-pubertal European Wild Boar (3 gilts and 2 boars), weighing 53-89 kg, and five cross-bred post-pubertal domestic gilts weighing 90-110 kg. The European Wild Boar were from an eight-hectare forested enclosure where the animals live in a semi-natural environment. During the experiment the European Wild Boar were housed outdoors in small individual stalls under natural lighting conditions. Domestic pigs were housed indoors in individual pens under light conditions typical of commercial piggeries. Blood samples were collected at two-hour intervals for 48 hours via medial saphenous arterial catheters from the European Wild Boar and via ear vein catheters from domestic pigs. Serum samples were analyzed using a commercial melatonin radioimmunoassay (Bühlman Laboratories AG, Swizerland). The sensitivity of the assay was 0.3 pg/ml. Light intensity was measured using a digital light intensity meter.

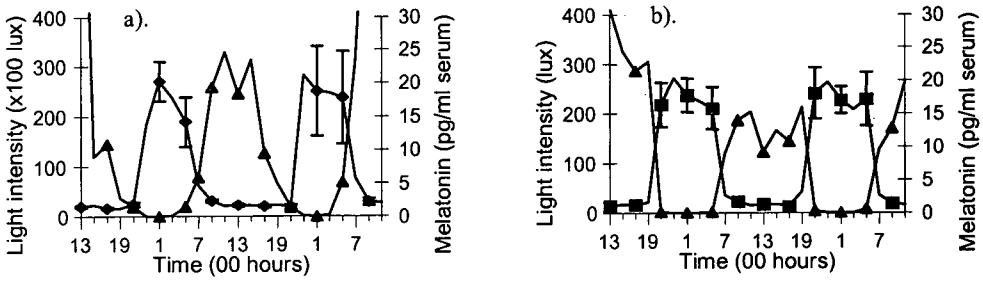

Figure 1. *Pattern of light intensity (lux, ▲) and serum melatonin concentrations (pg/ml, mean ± SEM) in a). European wild boar (◆) and in b). domestic pigs (■) over a 48 hour period.*

The European Wild Boar showed a distinct circadian pattern in melatonin concentrations similar to domestic pigs (Figure 1). The mean dark phase (<100 lux) melatonin concentration for the European Wild Boar (18.1 pg/ml ± 4.0 vs. 17.4 pg/ml ± 2.8) was not significantly different from that for the domestic pigs despite the dramatic difference in light intensities during the photophase.

It appears that there is no difference in the circadian melatonin response of the European Wild Boar in its natural environment and domestic pigs housed indoors. Therefore the reduced effect of season on reproduction associated with domestication, cannot be explained by a weakening of this response whether due to altered genotype or light environment.

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EFFECTS OF SOW PARITY ON PIGLET SURVIVAL AND GROWTH

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Piglet mortality is both an ethical and an economical problem for the pig industry. Usually, piglets born in large litters have low birth weights and high mortality (Rydhmer, 1992). Both the number of piglets born and piglet mortality rate increases with sow parity (sp) (Gatel *et al.*, 1987). Other relationships between sow traits and piglet survival and growth have not been studied.

From 1984 to 1994 data on piglet live weights, mortality and causes of death were collected from the 80 sow (mainly Swedish Landrace x Swedish Yorkshire) pig herd of the University. Records from 1,539 farrowings comprising 18,461 piglets have been used to study effects related to the sow on survival and early growth in piglets.

Pregnant sows were kept in stalls and were moved to farrowing pens without crates 3 weeks before farrowing. Pigs were weaned at 5 weeks and remained in the farrowing pen until 9 weeks of age. Piglets were creep fed on the floor from 1 week and in hoppers from 3 weeks of age. Standard sow feeding was 26 MJ ME daily during gestation and 74 MJ ME daily at a litter size of 10 during lactation. Number of farrowings in sp1 to sp4 was 366, 336, 277 and 214 and in sp5 or higher 346. The effect of parity on numbers of piglets and on piglet live weights and growth rates was examined using ANOVA. For piglet weights and growth rates litter size was included as a covariate in the model. Results are given as least-squares means. The effect of sow parity on mortality rate and the dependence between the sow's first and later litters was evaluated with Pearson correlation coefficients.

Number of piglets born per litter increased from sp1 to sp9 (10.9 to 13.5) and decreased thereafter. Piglet mortality rate increased with parity from 1.6 in p1 to 4.6 in sp8. Accordingly, litter size at weaning and at 9 weeks of age reached maximum values (10.0 and 9.6) as early as in sp3. Number of piglets overlain by the sow increased until sp6, whereas numbers stillborn and numbers dead from general weakness increased through to sp10 and higher. Piglet mortality rate (total, stillborn, overlain and weak piglets) was significantly correlated to parity ($P<0.001$), whereas mortality due to diarrhoea was not.

Sow parity had a significant influence on piglet birth weight ($P<0.001$). Birth weight was 1.40 kg in sp1, 1.47 kg in sp2 and decreased to 1.23 kg in sp8 or higher. Sow parity affected the piglet pre-weaning growth rate ($P<0.001$), with piglets in litters from sp2-sp3 sows growing faster (206 g/d) than piglets of older (195 g/d, $P<0.001$) or sp1 sows (200 g/day, $P<0.01$). Growth rates in sp1 piglets tended to be higher ($P<0.10$) than in piglets of sp4 or older sows. A similar influence of parity on piglet growth rate from birth to 3 weeks of age was recently reported by Högberg (1998). Between 5 and 9 weeks of age piglet growth rates were superior in sp1-sp3 litters ($P<0.01$).

Significant correlations ($P<0.001$) were found between the sow's first and later litters regarding number of piglets born per litter ($r=0.22$), piglet birth weights ($r=0.55$), piglet weaning weights ($r=0.22$) and live weights at 9 weeks of age ($r=0.30$), but not regarding piglet mortality ($r=0.08$, NS).

Piglet performance is influenced by parity. Except for the gilt litter, which generally has a poorer performance, piglet birth weights, survival and growth deteriorate with increasing parity. Low birth weights in litters of old sows probably contribute to lower survival and growth rates. The underlying causes of influence of parity on piglet performance deserve further investigation.

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A REVIEW - PRODUCTION AND PROCESSING IN AUSTRALIA: BREEDING FOR THE NEEDS OF BOTH

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Abstract

Apart from lean meat percentage, Australian pig breeders to date have placed little emphasis on processor requirements in their selection indices. Processor objectives will vary depending on their specific markets, i.e., fresh or processed products. Quality traits that can potentially affect processor profitability include meat and fat colour, water holding capacity, eating quality and processing yield. The decision to include any of these traits into the breeders' objectives is based on the relative economic importance of the trait, cost of measurement and its genetic architecture.

In terms of genetic options for the improvement of processor objectives, the most rapid improvement would be through the elimination of the Halothane (Hal) gene in both maternal and terminal sire lines. With respect to continued genetic gain, measurement of both colour and pH of the *longissimus dorsi* muscle, 24 hours after slaughter on a proportion of carcasses, and the inclusion of these traits in selection indices is probably the most cost effective method of improvement of meat quality. In the longer term, marker assisted selection and the use of candidate genes may offer a cheaper and more effective alternative. However, in order to alter their programs, breeders require financial incentives, which do not exist under current payment systems.

Introduction

Pig breeding programs to date have concentrated primarily on improving the lean to fat ratio in the carcass and reducing cost of production by improvements in growth rate, feed efficiency and reproductive rate.

Payment systems in Australia are usually based on price grids encompassing carcass weights and subcutaneous fat thickness measured at the P2 site. Depending on whether the pork is sold fresh or as processed pork products, other quality factors may have an impact on processor profitability to varying degrees.

The aim of this paper is to examine the effect of current selection programs on the broader processor objectives and discuss prospects and strategies for the inclusion of these objectives within our breeding programs.

Breeding objectives

The first step in determining whether this objective can be achieved is to identify the traits that affect the profitability of the two sectors. Examples of breeding objectives for a producer and processor are listed in Table 1.

Precise definition of the relevant importance of the traits in the processor's objectives varies depending on the market they are servicing. The market for fresh pork consists of two segments; bone-in product, e.g., traditional roasts and pork chops and boneless products, which form the basis of the new fashioned pork range. A major difference between these two market segments is the weight of the carcass required. Bone-in products are traditionally derived from carcasses less than 65 kg hot standard carcass weight (HSCW) and boneless products come from carcasses up to 95 kg HSCW. In terms of fatness, bone-in products require less than 12 mm of fat at the P2 site, while boneless products can be derived from significantly fatter carcasses without penalty.

Pork for both segments should be a light even colour, with low drip loss and good eating quality. Eating quality can be expressed in terms of flavour, tenderness and aroma.

The manufacturing market is also segmented, depending on the final product mix, especially with respect to how the middle is processed. If rind-on bacon is being

produced, the carcass weight tends to be around 75 – 80 kg HSCW, with fat thickness less than 14 mm at the P2 site. If the bacon is to be rindless, carcass weight and fatness restrictions can be relaxed. Manufacturing properties of pork, including freeze-thaw loss, pump yield and cooking loss are important as well as fat distribution through the primals, especially in the middle. Meat colour, fat colour and fat quality can also influence the saleability of manufactured products.

Table 1. Breeding objectives.

Producer	Processor
Feed efficiency	Carcass weight
Growth rate	Lean meat percentage
Reproductive efficiency	Meat colour
Mortality rate	Fat colour
Dressing percentage	Fat quality
P2 fat depth	Fat distribution
	Drip loss
	Cooking loss
	Eating quality

Inclusion of additional processor objectives in current breeding programs

The decision to include an additional trait into a breeding program is based on:

- The relative economic value of the new trait
- Heritability of the trait and its genetic correlations with other traits in the objective
- Cost of measurement of the new trait

The reasons why particular processor traits have not been included historically in these breeding programs can stem from one, or a combination of all of the above.

Economic value

The relevant economic values are one of the factors used to weight the amount of selection pressure placed on each individual trait in the selection objective. One method used to determine relative economic values is to identifyi the change in overall profitability by improving a particular trait by one unit, while holding all other traits constant. The values estimated rely on a number of assumptions with respect to input costs, prices, production and marketing systems. Economic values for a number of traits derived using this method are given in Table 2. This is a relatively simple procedure for traits, which affect the cost of production of either the producer or the processor.

Improvements in traits, which effect the saleability of the product such as eating quality factors, i.e., tenderness, flavour or aroma or the colour of the meat and fat, are more difficult to value. A mechanism is required in the market place, which differentiates quality on price. The Meat Standards Australia program in the beef industry is one example of how this could be done. Based on a number of criteria beef cuts are categorised into four grades, which reflect the likely eating quality of the product (Cox, 1999). The product is then labelled at the retail level with respect to the grade, allowing differentiation in pricing. Without such a mechanism, the breeders face the dilemma of including traits in their selection objectives and indices that may not lead to any increase in overall profitability. This may occur at the expense of other traits that breeders are more certain will improve producer profitability.

Table 2. Economic values for a number of producer and processor traits.

Trait	Economic value per unit per progeny (75 kg HSCW) $
Daily gain (gm)	0.07
Feed efficiency (1 unit)	-28.00
P2 (mm)	-3.50
Number born alive (1 piglet)	3.50
Drip loss (1 percent)	-3.00
Cooking loss (1 percent)	-4.50
Sensory overall acceptability (1 unit)	?
Colour (1 unit)	?
Fat quality and colour	?
Fat distribution	?

Genetic Parameters

The genetic architecture of the trait is an important factor that can affect the relative weighting placed on different traits in a selection index. In other words, to what extent will the trait respond to selection? This is quantified by the heritability, and to what extent other traits change with selection of a particular trait. The relationship between any two traits is quantified by the genetic correlation between the traits. Average heritability values for a number of the producer and processor objectives are given in Table 3. The selection response will be directly related to the heritability values in Table 3 if the measurements are based on the candidate itself. For a number of processor traits this will not be useful, as measurements can only be taken on relatives of the candidate. Depending on the closeness of the relationship and the number of relations measured, selection response for these traits will be reduced, compared to selection utilising the candidate's own information (Cameron, 1998). The advent of cost effective cloning in pigs would circumvent this problem, with the relevant data being collected on identical clones after slaughter.

Table 3. Heritability of producer and processor traits.

Trait	Average heritability	References[a]
Growth rate over lifetime	0.27	1,2
Feed Conversion Ratio	0.26	1,2
P2 Fat depth	0.40	1,2
Number born alive	0.10	2
Lean meat percentage	0.50	2,3
Drip loss	0.16	2,3
Cooking loss	0.16	2,3
Overall acceptability (Sensory Panel)	0.25	3
Meat colour	0.24	2,3

[a]References: 1. Clutter and Brascom (1998); 2. Hermesch (1996); 3. Sellier (1998).

As breeders are usually selecting on more than one trait at any one time, the genetic correlations between traits will affect the relative selection response for each trait. In Table 4, genetic correlations between a number of production and processor traits are presented. Based on the above correlations, current indices based on growth rate, carcass leaness and feed efficiency will lead to unfavourable changes in a number of the processing traits.

Table 4. Genetic correlations between current selection criteria and processor objectives.

Current selection criteria	Processor trait	Genetic correlation	References[a]
Carcass leaness	Colour	0.2	1,2
	Drip loss	-0.2	1,2
	Ham manufacturing yield	-0.4	1
	Tenderness	-0.2	2
	Juiciness	-0.2	2
	Pork flavour	-0.3	2
	Fat firmness	-0.4	2
Average daily gain	Colour	0	1
	Drip loss	0.1	1
	Ham manufacturing yield	-0.3	1
Feed efficiency	Colour	-0.6	1
	Drip loss	-0.6	1
	Ham manufacturing yield	0.3	1

[a]References: 1. Hermesch (1996); 2. Sellier (1998).

In Table 5, annual genetic gains for a number of traits are presented for three lines with different selection indices. The genetic gains were simulated using Estimated Breeding Values generated in Bunge Meat Industries' breeding program and commercial selection intensities for replacement boars and gilts. A maternal index was used for Line 1, with Lines 2 and 3 based on terminal sire indices. The aim in Line 2 was to minimise the change in colour of the loin, while maximising selection response in efficiency, growth and carcass lean. The major emphasis in Line 3 was on improving carcass lean. The data in the table show that without some emphasis being placed on 'meat quality' in the index, it will decline.

Table 5. Annual genetic gains with different selection indices.

Trait	Line 1	Line 2	Line 3
Number born	0.20	0.09	-0.10
Feed efficiency (kg feed/kg gain)	-0.05	-0.12	-0.13
Growth rate over lifetime (g/day)	9	20	20
P2 (mm)	-0.70	-0.50	-1.5
L.D. Colour (L * Units)	0.20	0.10	0.40

Cost of performance recording

The final factor that can influence the inclusion of traits in a selection index is the cost of measurement of the respective traits.

The majority of breeding programs in Australia are based on selection indices which include growth rate, P2 fat depth and number born alive. All three traits are relatively easy and inexpensive to measure. In terms of producer objectives, although feed efficiency has a relatively high economic value, few breeders are directly recording the trait, mainly because of the cost of recording.

A similar situation holds for the majority of the processor traits. Direct measurement for the processing and meat quality traits requires individual tracking of carcasses through the processing and, possibly, retail chains. For example obtaining data on individual ham or bacon yields, or eating quality traits from individual animals is prohibitively expensive.

An alternative is to identify traits less expensive to measure but highly correlated genetically with the traits of interest. The traits that have been most extensively reviewed include pH at forty-five minutes and twenty-four hours after slaughter, intramuscular fat and reflectance or colour twenty-four hours after slaughter (Sellier 1998). In terms of fulfilling the three criteria outlined above, pH and colour twenty-four hours after slaughter and intramuscular fat appear to be the most promising. The heritability values for all three traits are approximately 0.20, with genetic correlations above 0.5 with a number of the processing and meat quality traits (Cameron, 1990; Berger *et al.*, 1994; de Vries *et al.*, 1994; NPPC, 1995; Hermesch, 1996). The measurement costs for both pH and colour will depend on the breeders access to slaughter and boning facilities, as pigs must be tracked through to at least where the carcass is broken into primals. Measurement of intramuscular fat currently requires samples to be taken also at this stage, with analysis using chemical extraction or near infra-red measurement. A cheaper alternative being explored is the analysis of ultrasound generated images of the *longissimus dorsi* muscle. This technology would have the added advantage of being able to generate data on the pig prior to selection.

Table 6. Cost of measurement of trait.

Trait	Cost per animal[a]
	$
Daily gain (gm)	1.00
Feed efficiency (1 unit)	18.00
P2 (mm)	2.00
Number born alive (1 piglet)	1.00
Drip loss (1 percent)	5.00
Cooking loss (1 percent)	8.00
Sensory overall acceptability (1 unit)	100.00
Meat colour (1 unit)	3.00

[a]The costs for the post slaughter measurements include only labour and material costs, i.e., it assumes access to both abattoir, boning and manufacturing facilities.

New technologies

So far the discussion has focused on traditional methods of genetic improvement. Over the past decade there have been major advances in the field of DNA marker technology. Accompanying these advances has been the opportunity to improve the rate of genetic progress. The two major applications of the technology are the identification of candidate genes and the use of marker assisted selection (MAS).

Candidate or major genes are ones whose variation has shown to directly affect performance traits. The economic value of theses genes can be substantial; achieving in a single generation what would take several generations of conventional selection. The two most well known examples both relate to genes which also affect processing and meat quality traits, the Hal and acid meat (RN) genes.

Sellier (1998) has reviewed the effects of both genes on a number of performance traits. In both cases the genes were found to have both positive and negative commercial effects. In the case of the Hal gene, heterozygous progeny have been found to be worth approximately $5.00 more, due to superior lean content, dressing percentage and feed efficiency (Luxford, 1995). However, the amount of pale soft exudative meat in the study was 16% higher in the same pigs compared to the homozygous negative animals (Luxford, 1995). Whilst there is no payment system for meat quality, it is understandable why the halothane gene has not been eliminated from all breeding lines.

The potential use of MAS has arisen with the development of linkage maps of the porcine genome, i.e., the identification of numerous polymorphic genes or markers across all the pigs' chromosomes. Variation in these polymorphic markers is then matched to variation in performance of any particular trait in animals of known pedigree. Unlike the case of the candidate gene, the marker gene is most likely not functionally responsible for the variation in performance, but is closely linked to the responsible gene on the same chromosome. These genes are referred to as quantitative trait loci (QTL).

There are a number of studies around the world searching for useful QTLs, including a Pig Research and Development Corporation (PRDC) funded program in Australia. Preliminary results from this program indicate potential QTLs for intra muscular fat percentage, carcass lean, colour of the *longissimus dorsi* and pH at both forty-five minutes and twenty-four hours after slaughter. The identification of the QTL in an experimental situation is the first step in the application of MAS within pig breeding programs. Further work is required in each commercial herd to verify that the same QTL alleles are associated with the same marker alleles. This may not be the case due to recombination. There is also the question of the cost of analysis of the markers and also the development of software to incorporate the marker information into our current genetic evaluation programs, e.g., by PigBlup.

Environmental manipulation of processing traits

The majority of the traits that would potentially improve processor profitability have heritability values equal to, or less than 0.25. Therefore at least 75% of the variation seen in these traits is due to non-genetic factors. Wood *et al.* (1998) put forward the proposition that it is possible to achieve a 50% improvement in the tenderness of fresh pork with the use *ad libitum* feeding, electrical stimulation of the carcass, pelvic suspension of the carcass, ageing of the meat and rapid chilling after slaughter. The decision making process, with respect to whether additional traits are included in our breeding programs, should take into account whether it would be simpler and less expensive to improve the traits with alternate technology.

Conclusion

A review of the current selection indices in relation to the breeding objectives of producers and processors highlights a bias in favour of the producers' requirements. This is not due to any technical impediments to the inclusion of additional processor traits, but rather, has a basis in economics. Under the existing payment schedules in Australia there is no incentive to improve any trait other than P2 fat depth.

Inclusion of additional processing traits will reduce the potential genetic gain in existing traits in the index, as well as adding additional costs to the breeding programs, which may be substantial. It could be argued that even without pricing changes, it may be worthwhile to individual breeders to alter their selection indices in the hope that their producer clients become preferred suppliers, this then resulting in greater market share.

This line of thinking could also be extended to an industry basis, with the aim of increasing pork consumption at the expense of other meats by improving its appeal.

In terms of genetic options for the improvement of processor objectives, the largest and quickest improvement would be through the elimination of the Hal gene in both maternal and terminal sire lines. With respect to continued genetic gain, measurement of both colour and pH of the *longissimus dorsi* muscle twenty-four hours after slaughter on a proportion of animals and its inclusion in selection indices is probably the most cost effective method of at least maintaining meat quality.

In the longer term MAS and the use of candidate genes may offer a cheaper and more effective alternative.

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MAPPING QUANTITATIVE TRAIT LOCI (QTL) FOR CARCASS AND OTHER TRAITS ON CHROMOSOME 2 IN PIGS

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As part of an international collaboration attempting to identify economically important genes in pigs, nine microsatellite markers have been genotyped at intervals of 10 to 20 centiMorgans (cM) to scan for QTLs on chromosome 2 in Meishan x Piétrain (n=316), Wild Boar x Piétrain (315) and Wild Boar x Meishan (335) F_2 resource pedigrees, bred and measured for 43 performance traits at the University of Hohenheim. Genotyping employed an ABI 373 automatic system. Quantitative trait loci were mapped using multiple marker least squares regression. Threshold values of the F statistic were calculated by permutation (Churchill and Doerge, 1994). Genome-wide thresholds applied a Bonferroni correction to account for chromosomes not included in the current analysis.

Preliminary results for the Meishan x Piétrain pedigree indicate that the multipoint linkage map [S0141(0.0)-Sw240(12.7)-*MYOD1*(32.8)-Sw395(44.3)-S0010(56.2)], shown with map position in cM in parentheses for five markers, is consistent with published maps. These markers were used to detect QTLs affecting 11 traits at the 5% chromosomal significance level. Table 1 indicates the estimated positions of these QTL, their significance and the proportion of the residual F_2 phenotypic variance explained by the QTL. Percentage figures for lean and fat measurements are by weight as a proportion of half carcass weight. Meishan alleles were associated with increased fatness of the carcass and lower weights of lean cuts. The evidence for the last three QTLs, with F ratios exceeding the stringent 5% genome-wide significance threshold, is conclusive.

Table 1. QTL significant at the 5% chromosomal level in Meishan x Piétrain family.

Trait	Position(cM)	F ratio	% F_2 variance
Weight of bacon meat (kg)	22.7	5.43	3.3
Weight of back fat (kg)	41.8	5.60	3.5
% dissectable fat in half carcase	40.8	5.84	3.7
Weight of shoulder meat (kg)	24.7	6.06	3.8
% bacon including fat in half carcass	38.8	6.21	4.1
pH 24h p.m. in *m longissimus dorsi*	38.8	6.28	3.9
Ratio of weights of lean to fat in half carcass	35.8	6.45	4.1
Liver weight (g)	28.7	6.67	4.3
Average number of teats	6.0	9.35	6.0
% lean (shoulder meat, bacon meat, chops and fillet) in half carcass	38.8	10.92	7.2
% bacon meat excluding fat in half carcass	37.8	11.09	7.4

The preliminary results have provided conclusive evidence for the existence of QTL for important traits on chromosome 2, but these QTL will only be exploitable if they are also found segregating in commercial populations. Nezer *et al.* (1999) and Jeon *et al.* (1999) recently mapped an imprinted QTL with a large effect on muscularity and fat deposition to a region of chromosome 2 not yet covered by the map referred to above, using a commercial cross and wide cross respectively. This QTL is now being evaluating in the Hohenheim resource pedigree.

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GENETIC PARAMETERS FOR GROWTH RATE AND BACKFAT FOR LARGE WHITE AND LANDRACE PIGS RAISED IN A TROPICAL ENVIRONMENT

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Accurate genetic parameter estimation of performance traits is important for prediction of the breeding value of stock. Genetic parameters for performance traits have been estimated in many studies for temperate environments but only a limited number of studies can be found for tropical environments. This study focuses on a tropical environment and estimates the genetic parameters for growth rate and backfat for Large White (LW) and Landrace (LR) pigs in Indonesia.

Performance test data was recorded between May 1998 and March 1999 in a piggery on the Indonesian island of Bulan, south of Singapore. Pigs were reared in normal climatic temperatures for the region, 22 to 32°C, and fed a diet of 16.9% crude protein and 13.5 MJ/kg of digestible energy *ad libitum*. Growth rate from birth to 28 weeks of age (GR) and live animal fat depth measured at the P2 site (P2) were recorded. The data included 2788 LW and 1821 LR pigs. Both breeds were tested at 196 days of age averaging 92.4 kg and 95.5 kg live weight for LW and LR, respectively. Data were analysed with the program DFREML (Meyer, 1997) and the model included the unit the pig was raised in, sex, source of genotype (either pure Indonesian or Australian derived) and week of performance testing. Weight was also fitted as a linear covariate for P2. The random effect of litter was fitted to the model for both traits.

Table 1. Heritabilities (h^2) and litter effects (c^2) with standard errors (in brackets), variance components and the genetic correlation (r_g) between growth rate (GR) and backfat (P2) with standard error (in brackets) for LW and LR pigs.

Breed	Trait	h^2	c^2	σ^2_a*	σ^2_c	σ^2_e	r_g
LW	GR	0.21 (0.06)	0.10 (0.03)	764	361	2527	0.12 (0.19)
	BF	0.23 (0.05)	0.07 (0.03)	0.68	0.20	2.08	
LR	GR	0.38 (0.08)	0.06 (0.03)	1321	203	1956	0.20 (0.15)
	BF	0.40 (0.07)	0.00 (0.03)	1.39	0.02	2.10	

*σ^2_a, σ^2_c, σ^2_e are the additive genetic, litter and residual variances, respectively

Heritabilities found were not significantly different from estimates of genetic parameters for LW and LR in tropical conditions by Dzama and Mungate (1998). Positive genetic correlations between GR and P2 were found for both breeds. These are economically unfavourable correlations and may result from *ad libitum* feeding. Heritabilities estimated in this study indicate that improvement for GR and P2 could be made under tropical conditions with the possibility of greater improvement in both traits for LR than LW animals due to higher heritabilities. Correlations between the two traits might become more favourable if the practice of restricted feeding was used, as was found by Dzama and Mungate (1998).

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PIG RESEARCH AND DEVELOPMENT CORPORATION

THE EFFECTS OF SEX AND FEEDING REGIME ON CARCASE TRAITS IN AUSTRALIAN PIGS

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Identification of factors that effect the level of production is useful for management, and it is an essential part of genetic evaluation. Previous experiments have identified a significant effect of feeding regime (a controllable aspect of management) on carcase traits and lean meat growth (Cameron *et al.*, 1999). In this study it was possible to include the sex of the animal in the analysis. The aim of this study was to investigate the effect of sex on carcase traits and the interaction between sex and feeding regime (S x F), using data from a commercial herd.

The study used abattoir data from a total of 4,464 Landrace and Large White animals, with gilts and boars recorded under *ad libitum* or restricted feeding. Feed restriction was 11% of *ad libitum* feed intake, and the diet contained 13.6 MJ ME/kg. Pigs were tested from 80 to 105 kg live weight. The traits included were hot carcase weight (HCW), backfat at the P2 site (P2), weight of the whole back leg (BLW), weight of the trimmed back leg (HAMB) and weight of the slash boned ham (HAM). The model included the fixed effects of sex, breed, feeding regime, slaughter month, housing pen (approximately 30 animals/pen), and a random sire effect. Least Square Means (LSM) were calculated using the MIXED procedure in SAS (SAS, 1993).

Sex was significant in all traits, with the gilts being lighter and fatter than the boars (Table 1). There were significant S x F interactions for HCW, BLW and HAMB. The BLW difference between sexes under *ad libitum* feeding was 0.08 kg compared to 0.28 kg under restricted feeding. For HAMB, the differences between the sexes were 0.69 kg and 0.53 kg for restricted and *ad libitum* feeding, respectively. Sex had a significant effect on HAM, and the differences between sexes were 0.36 kg and 0.29 kg for restricted and *ad libitum* feeding respectively. Feeding regime within sexes did not affect HAM.

Table 1. Differences in LSM (standard errors) of carcase traits - restricted gilts set to 0

	Gilts		Boars		
Trait	Restricted	Ad libitum	Restricted	Ad libitum	Significance[1]
HCW	0 (0.31)	3.80 (0.35)	3.23 (0.29)	5.31 (0.27)	S, F, S x F
P2	0 (0.13)	0.55 (0.13)	-2.00 (0.12)	-1.48 (0.11)	S, F
BLW	0 (0.05)	0.45 (0.05)	0.28 (0.04)	0.53 (0.04)	S, F, S x F
HAMB	0 (0.05)	0.31 (0.06)	0.69 (0.05)	0.84 (0.05)	S, F, S x F
HAM	0 (0.05)	0.03 (0.06)	0.36 (0.05)	0.32 (0.05)	S

[1]Significant at $P \leq 0.05$; S, sex; F, feeding regime; S x F, sex by feeding regime interaction.

The results for BLW, HAMB and HAM indicate that this level of restriction does not have an effect on HAM, but does have an effect on the fat and bone growth, which is different between sexes. Therefore, the feeding regime could possibly be manipulated for each sex. From a management perspective this would indicate that the level of feed restriction can be managed in the commercial herd so that each sex reaches the optimal level of fat and lean that is desired in a specific production and market system.

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PIG RESEARCH AND DEVELOPMENT CORPORATION

PATTERN OF FOOD INTAKE DURING THE GROWING PHASE DETERMINES DEPTH OF BACKFAT IN PIGS AT SLAUGHTER

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Pork processors prefer uniform carcass quality and pay less for over-fat pigs. A study in a large, commercial piggery showed that the average depth of backfat (P2) for pigs varied from 12 to 16 mm over 12 months and that this was not accounted for by differences in live weight (LW) at slaughter (Trezona *et al.*, 1999). It was hypothesised that the pattern of food intake varies during the year because of ambient temperature, and this in turn influences the deposition of fat relative to lean. Pigs with a restricted intake during the grower phase compensate during finishing when the gain of lean to fat is reduced and will be fatter at slaughter than pigs with the opposite pattern of intake.

To test this hypothesis the pattern of food intake was altered and the depth of backfat was measured. The AUSPIG simulation model (Black *et al.*, 1986) was used to determine the intake required to induce the differences in P2 that had been recorded in the earlier study (Trezona *et al.*, 1999). Twenty-four Large White x Landrace female pigs were allocated to three treatments (n=8) at weaning (5.5 kg LW) on the basis of litter and LW. Treatment AA pigs were fed *ad libitum* throughout, whereas treatment AR pigs were fed *ad libitum* until 40 kg LW. Following this their intake was reduced by 10% of that eaten by their littermates on the AA treatment from 40 to 60 kg LW, and then 20% from 60 kg LW until slaughter. In the third treatment (RA), the feeding intake was limited by 20% of that eaten by their AA littermates until 40 kg LW and they were then fed *ad libitum* until slaughter. Pigs were individually housed in thermoneutral conditions. Regardless of treatment all pigs were fed the same commercial pelleted diets appropriate to their weight range. Post-slaughter P2 was measured and, after mincing half of the carcass, fat and protein content were determined by chemical analysis. Data were analysed by ANOVA and treatment means were compared using orthogonal contrasts.

Table 1. How pattern of intake influences average daily gain (ADG), live weight at slaughter (LW), depth of backfat (P2) and the % of fat and lean in the carcass.

Treatment	ADG (g)			LW	P2	fat	protein
	5.5–40 kg	40-100+ kg	5.5-100+ kg	(kg)	(mm)	%	%
AA	497	949	667	101.6	17.5 [ab]	23.8	18.0
AR	505	839	643	102.1	15.9 [a]	23.9	17.3
RA	396	945	641	104.7	18.7 [b]	23.8	17.9

[a,b]Values in the same column with different superscripts are significantly different ($P \leq 0.05$).

The hypothesis was supported because, when food intake was restricted in the period up to 40 kg LW followed by *ad libitum* feeding thereafter (RA), pigs had higher P2 ($P \leq 0.05$) than those that ate comparatively less in the later stages of growth (AR) (Table 1). Despite this difference in P2, there was no difference in either fat or protein content of the carcass. It is concluded that pattern of food intake may explain much of the difference in P2recorded in commercial piggeries. Since total fat in the carcass was not affected it indicates that fat has been re-distributed and suggests that P2 is not always an accurate predictor of total body fat in pigs.

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PIG RESEARCH AND DEVELOPMENT CORPORATION

THE DEVELOPMENT OF A CIRCADIAN PATTERN OF CORTISOL SECRETION IN SALIVA OF NEONATAL PIGLETS

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A circadian rhythm in cortisol concentrations has been recorded from salivary samples collected from pigs older than 8 weeks of age (Ekkel *et al.*, 1996). This investigation was an observational study to measure cortisol in saliva collected from neonatal piglets to gain a better understanding of the secretory dynamics of the hypothalamic-pituitary-adrenal (HPA) axis in the newborn piglet using a minimally-invasive approach.

Saliva was collected from two litters of piglets (n=16) from multiparous sows (Large White x Landrace) at 0700, 1100, 1500, 1900 hours on days 0, 1, 2, 4, 6, 10, 14, 18, 22, 26 and 30 *post partum*. Saliva was collected using a plastic pipette attached to a vacuum pump, inserted gently under the piglet's tongue. Salivation was facilitated by administering 2 drops of 5% citric acid onto the tongue before collection. Salivary cortisol concentration was determined by radioimmunoassay.

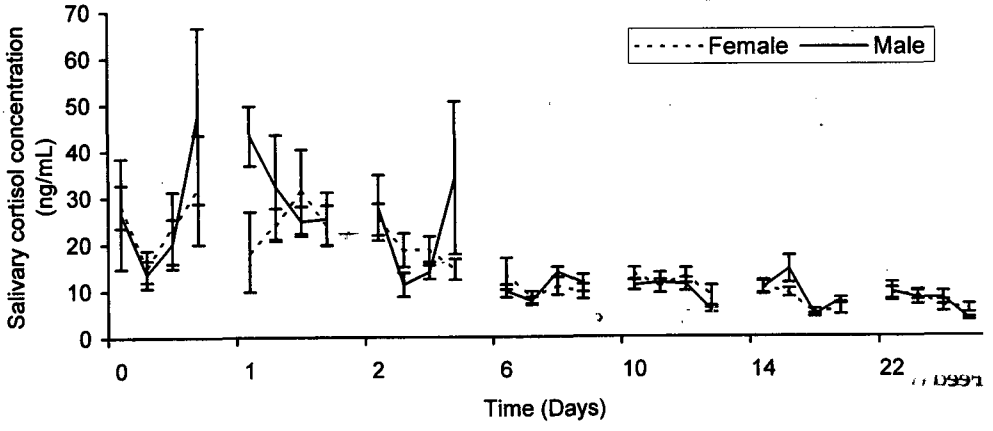

Figure 1. *Mean (± SEM) circadian cortisol secretion in saliva of male and female piglets from day 0 to day 30 post partum. (Points represent concentrations at 0700, 1100, 1500 and 1900 hours respectively).*

Salivary cortisol concentration was high perinatally and declined with age, consistent with the observations made by Kattesh *et al.* (1990) in piglet plasma. Two litters of control piglets (sampled on day 22 only) displayed similar salivary cortisol profiles in both rhythm and magnitude of cortisol secretion at 22 days of age (9.0 ± 2.00 ng/ml at 0700 h, 3.9 ± 0.78 ng/ml at 1900 h) suggesting no adaptation to repeated handling procedures.

Santiago et al. (1996) defined that a circadian rhythm occurs when the 1900 hour concentration of hormone is 75% or less than the 0700 hour concentration. A circadian pattern was evident, therefore, in female pigs from day 6, while males displayed a circadian pattern by day 10. This pattern, which in the present study was established prior to weaning, is similar to that reported by Ekkel *et al.* (1996) in pigs after weaning. These results demonstrate that the secretory pattern of the HPA axis in the newborn piglet is established during the first few days after birth.

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EFFECT OF PORCINE SOMATOTROPIN ADMINISTRATION BEFORE WEANING ON GROWTH PERFORMANCE IN PIGS

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The administration of porcine somatotropin (pST) to growing pigs decreases carcass fat and increases carcass lean content. Usually, the treatment is required over a long period of time up to slaughter. It has been shown in sheep and rats however, that a prolonged change in body composition can be induced by somatotropin administration before weaning (McCutcheon *et al.*, 1994; Kadim *et al.*, 1996). These favourable changes in body composition presumably reflect an inhibition of adipocyte number and/or size, particularly in the subcutaneous depots. The aim of this project was to investigate whether the same long-term effect on carcass composition can be obtained in pigs.

Twenty two Large White x Landrace x Duroc female pigs from 7 different litters were weighed and randomly allocated within litter to one of the two treatment groups at birth. The piglets were treated from one up to 24 days of age by twice-daily intramuscular injections of saline (control, n=11) or pST (0.3 mg/kg/d, n=11). The piglets were weaned at 28 days of age and were then kept in groups of five or six and fed to "appetite" twice daily up to slaughter (85 kg body weight, BW). Individual feed intake was recorded between 70 days of age and slaughter. Backfat thickness was measured at a set distance (cm) from the midline at the last rib using real-time ultrasound technology (Aloka 210 DX, 3MHz probe) at 25 days of age (2 cm from the midline) and at 10 weeks of age (6 cm) and by direct measurement at slaughter (6 cm from the midline). Treatment effects on individual data were determined by analysis of variance.

Table 1. Growth performance and backfat thickness of female pigs treated with saline or pST from birth to 24 days.

Treatment	Control	pST	SE	Significance[1]
Growth rate 0 to 25 days (g/d)	241	266	12	NS
Growth rate 25 days to 70 days (g/d)	437	436	20	NS
Growth rate 70 days to slaughter (g/d)	777	808	21	NS
Feed conversion ratio (kg/kg)	2.76	2.68	0.11	NS
Backfat thickness at 25 days (mm)	4.30	3.80	0.19	*
Backfat thickness at 70 days of age (mm)	5.30	5.20	0.27	NS
Backfat thickness at slaughter (mm)	11.20	11.10	0.38	NS

[1]NS, Not significant; *P≤0.05.

At the end of the treatment period, pST treated piglets had significantly less backfat (P<0.05) than their saline-treated counterparts (Table 1). However, the difference in backfat thickness had disappeared by 10 weeks of age. No differences in average daily gain or feed conversion ratio were observed between the treatment groups at any time. Dunshea *et al.* (1997) also reported a lack of response in growth rate when piglets were treated with pST between 4 and 31 days of age. It is concluded that although pST treatment up to weaning caused a reduction in backfat during the administration period, it failed to induce long-term improvements in feed utilisation or carcass composition.

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NEONATAL TREATMENT OF PIGLETS WITH GROWTH HORMONE RELEASING HORMONE ENHANCES GROWTH BY UP-REGULATING GROWTH HORMONE SECRETION IN MALES ONLY

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The endocrine environment which neonates experience in the first few days *post-partum* programmes the pattern of hormone secretion throughout their life (Csaba, 1986). In the case of growth hormone (GH), both growth hormone releasing hormone (GHRH) and somatostatin act on the somatotrope in the pituitary to control the template pattern of GH secretion as it is established in the neonate. This study investigated the effect of exogenous GHRH administration to newborn piglets on GH secretion and growth performance.

Litters of piglets from eight primiparous Large White x Landrace sows (Bunge Meat Industries Ltd., Corowa, NSW) were randomly allocated to one of four treatments: (1) saline injection, (2) 0.125, (3) 0.25 or (4) 0.50 nmol/kg live weight of D-Ala^2 hGHRH (1-29)NH_2. Injections were administered on day 0, 1 and 2 postnatally. Teeth and tail clipping and routine injections were performed on day 4 *post-partum*. On day 28, prior to weaning, three blood samples were collected from eight piglets per treatment at intervals of 15 minutes to record basal plasma GH status. Live weight was recorded at birth, and on days 28, 48, and 70. Circulating GH concentrations were determined by radioimmunoassay. Data were analysed by linear regression of means for each litter.

Table 1. Effects of neonatal treatment with hGHRH on mean basal circulating GH concentrations at weaning (day 28), and mean live weight at days 28, 49 and 70 in male (M) and female (F) pigs. All data are calculated on a mean litter basis.

		GHRH (nmol/kg)				Pooled	P value
		0	0.125	0.25	0.5	SEM	Linear
Basal GH (ng/ml)	M	6.6	9.6	11.3	14.5	2.10	0.050
	F	10.3	5.8	12.8	9.1	2.27	0.644
Birth weight (kg)	M	1.26	1.31	1.34	1.25	0.034	0.777
	F	1.26	1.26	1.34	1.25	0.105	0.994
Weaning weight (kg)	M	5.27	5.48	6.69	6.83	0.443	0.036
	F	5.05	5.47	6.32	5.98	0.414	0.264
Day 49 weight (kg)	M	10.1	10.4	12.7	12.5	1.16	0.201
	F	11.0	11.2	13.5	12.3	0.90	0.329
Day 70 weight (kg)	M	19.2	20.8	24.7	24.9	2.28	0.140
	F	22.7	23.5	26.1	24.0	2.09	0.602

Basal GH concentration in male weaner piglets increased linearly with dose of hGHRH analogue administered neonatally (P=0.05). This was associated with a dose-dependent increase in weaning weight (P=0.036), a trend which was still evident (although not significantly so) at day 70 of age (P=0.140). In contrast, there were no effects of GHRH treatment in female piglets.

These results suggest that the steroidal environment of the neonate may be important in directing the function of pituitary somatotropes subsequently through the growth phase. The results have thus identified a potential mechanism for boosting endogenous GH status and therefore growth efficiency without having to resort to repeated GH injections.

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PORCINE SOMATOTROPIN (REPORCIN®) IMPROVES GROWTH PERFORMANCE AND DECREASES BACKFAT IN PIGS UNDER COMMERCIAL CONDITIONS

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Porcine somatotropin (pST) treatment of pigs consistently improves average daily gain (ADG) and feed:gain (Campbell *et al.*, 1990; 1991). However, most studies have been conducted with individually penned pigs, of unimproved genotype and over a lighter finisher phase than current practice. The present study was designed to determine whether a commercial pST (Reporcin®) treatment regime would improve growth performance in heavy boars and gilts of an improved genotype and housed under commercial conditions.

The 2 x 2 factorial experiment involved 160 nineteen-week old Large White x Landrace pigs (80 males and 80 females) in 20 pens of eight pigs/pen. The respective factors were sex (boars and gilts) and dose of pST (0 and 5 mg/day). Pigs were fed a wheat-based diet formulated to contain 200 g crude protein, 10.2 g available lysine and 14.6 MJ DE/kg to ensure that responses to pST and sex were expressed. Injections of pST were given daily for 4 weeks prior to slaughter using a commercial applicator gun designed for this purpose. Pigs were bled by venipuncture on d 7 and 28 of treatment and the plasma samples analysed for plasma urea nitrogen (PUN) using a commercial kit (Sigma). A veterinary pathologist assessed the degree of stomach ulceration at slaughter.

Table 1. Effect of sex (S) and pST (ST) treatment on performance of finisher pigs.

Sex (S)	Boar		Gilt			Significance		
Treatment (ST)	Control	pST	Control	pST	sed	S	ST	S x ST
Initial weight (kg)	79.4	79.1	78.6	77.9	0.7	0.07	0.31	0.66
Final weight (kg)	114.7	115.3	107.7	113.5	1.7	0.004	0.023	0.055
Average daily gain (g/d)	1261	1293	1039	1273	50	0.005	0.003	0.015
Feed intake (g/d)	3061	2726	3005	2704	117	0.64	0.002	0.84
Feed:gain	2.43	2.11	2.90	2.12	0.07	<0.001	<0.001	<0.001
Carcass weight (kg)	89.4	89.3	85.5	88.2	1.3	0.021	0.18	0.15
P2 backfat (mm)	17.3	15.0	16.6	13.4	0.8	0.084	<0.001	0.45
Plasma urea (mmol/l)	5.30	3.90	5.85	3.76	0.20	0.17	<0.001	0.018

Daily pST treatment increased ADG, particularly in gilts as indicated by the interaction between sex and pST. Thus, gilts treated with pST grew 23% faster than control gilts whereas the pST treated boars grew only slightly faster (+2.5%) than control boars. Feed intake was similar for boars and gilts and was decreased in both sexes by 10% during pST treatment. Feed:gain was higher in control gilts than in boars and was improved by pST treatment. This was particularly evident in the gilts such that there was no difference in the feed:gain of pST-treated gilts and boars. Backfat at P2 was reduced by 2.3 and 3.2 mm in boars and gilts, respectively. The only stomach lesions observed were very minor in severity and there was no effect of pST on the proportion of pigs exhibiting stomach ulcers (7/80 and 9/79 for control and pST treated pigs, respectively; χ^2 =0.31, P=0.58) or possible lesions at the injection site (0/80 and 1/79; χ^2 =1.01, P=0.31). The PUN response mirrored the effects of sex and pST on feed:gain. Indeed, there was a high correlation (r=+0.84, P<0.001) between feed:gain and PUN. In conclusion, Reporcin® treatment of finisher pigs of an improved genotype and housed under commercial conditions improved growth performance, decreased backfat and negated sex differences.

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INSULIN-LIKE GROWTH FACTOR-I (IGF-I) ALTERS THE MORPHOLOGY OF EPITHELIAL TIGHT JUNCTIONS IN THE DUODENUM OF 36-HOUR OLD PIGLETS

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Early growth and development of the intestinal tract in the pig plays a major role in post-weaning growth performance. An important parameter of intestinal function is its permeability, which is influenced by the morphology of tight junctions (TJ). The role of TJ formed between intestinal epithelial cells (mucosa), is to provide a barrier against the absorption of foreign bodies. Thus, TJ play a major role in maintaining animal health and consequently affecting growth performance. Burrin *et al.* (1996) showed that IGF-I given orally stimulates mucosal growth in 4-day old piglets. The present study aimed to investigate whether IGF-I also improves epithelial TJ morphology in the small intestine.

Twenty newborn unsuckled Large White piglets were maintained in individual metabolism cages at 34°C room temperature. They were divided into an initial slaughter group (ISG) and four treatment groups that were fed by bottle (i) sows' colostrum (SC) or (ii) sows' milk (SM) or (iii) sows' milk supplemented with recombinant human IGF-I to provide IGF-I at the same concentration as in sows' colostrum (Tivey *et al.*, 1997) (SM+IGF-I) or (iv) sows' milk supplemented with double the concentration of IGF-I (SM+2IGF-I). All treatment groups received equal gross energy (1.61 MJ GE/kg body weight) in hourly pre-warmed meals (38°C) from 3 to 36 h after birth. Animals in the ISG were killed at three hours of age and piglets in the treatment groups were killed at 36 h of age (one hour after their last meal). The duodenal region of the small intestine was dissected from all groups, fixed overnight in 4.0% paraformaldehyde + 1.25% glutaraldehyde and processed for transmission electron microscopy (TEM). The tissues were cut to 70 nm and stained with 5% uranyl acetate and 2% lead citrate. The TJ between the epithelial cells were located and photographed using TEM (magnification x 34,000). The length of TJ at the occlusion zone (OZ), adhesion zone (AZ) and the total length (OZ + AZ) between six epithelial cells per animal were measured by image analysis. Data were analysed by ANOVA.

Table 1. The effect of IGF-I on duodenal tight junction morphology in 36-hour old piglets.

Length (nm)	ISG	SC	SM	SM+IGF-I	SM+2IGF-I	SED
Occlusion zone	557^{a}	616^{a}	602^{a}	619^{a}	769^{b}	44
Adhesion zone	127.9	158	166	161.4	155	21
Total	716^{a}	836^{ab}	824^{ab}	860^{bc}	1036^{c}	66

^{a,b,c}Means within a row with different superscripts are significantly different ($P \leq 0.05$), LSD.

The provision of extra IGF-I to twice that present in sows' colostrum increased the length of TJs by 23% (1,036 vs about 840 nm; $P<0.05$) due solely to an increase in the occlusion zone. These results indicate IGF-I can modulate TJ structures that are associated with intestinal permeability. Thus IGF-I may play a role in regulating passage of material between epithelial cells of the small intestine in early postnatal life.

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THE EFFECT OF PORCINE SOMATOTROPIN TREATMENT IN THE LATE-PREGNANT SOW AND WEANER PIG ON GROWTH AND CARCASS QUALITY DURING THE FINISHER PHASE

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Porcine somatotropin (pST) administration to the young pig (10-25 kg LW) results in a preferential partitioning of nutrients to muscle growth (Harrell *et al.*, 1997), while the treatment of pregnant gilts in early- to mid-gestation caused an increase in foetal and placental weight (Sterle *et al.*, 1998). The objectives of this study were to determine the impact of pST administration to: (1) the late-pregnant sow with the aim of boosting IGF-I secretion into colostrum and therefore gut maturation; or (2) the weaner piglet to provide the metabolic drive to overcome the check to growth imposed by weaning; or (3) both of these treatments in combination.

Thirty primiparous sows (Large White X Landrace) and their litters were allocated to a 2 x 3 factorial experiment involving two pST treatments to the sows and three pST treatments to the piglets. Each sow was injected intramuscularly with either diluent or pST (2.7 mg/day; Reporcin, Southern Cross Biotech) from day 105 of gestation to farrowing. Litter size was standardised to 10 piglets per sow and weaning occurred at 28 days of age. Piglets within sow treatments were injected intramuscularly for four days before weaning and three days after weaning with either diluent, 0.75 or 1.5 mg pST/day. Live weight of the piglets was recorded at day 108 and 163 (slaughter). Individual food intake of male and female pigs was recorded in group pens with computerised electronic feeders during the finisher growth phase (day 108 to 163). Backfat thickness (P2) was recorded at slaughter. A linear mixed model was used in the statistical analyses, which included fixed effects of sow and pig treatments and sex and their two-factor and three-factor interactions, and the random effects of finisher, grower and weaner pens.

Table 1. Mean growth performance during the finisher phase (day 108-163) and carcass measurements of pigs raised from sows injected with either diluent or pST in late gestation and/or at weaning (n=5 litters per treatment and 10 pigs per litter).

Sows (S) pST dose	0			2.7			SEM	Main[1,2]	
Piglets (L) pST dose	0	0.75	1.5	0	0.75	1.5		S	L
Daily gain (g)	734	729	730	682	655	733	39	NS	NS
Feed:gain	2.76	2.82	2.71	2.90	3.14	2.90	0.12	NS	NS
Carcass weight (kg)	71.7	72.5	70.2	74.3	72.1	73.2	1.8	NS	NS
P2 (mm)	9.7	9.7	9.3	8.3	10.5	10.5	0.7	NS	NS

[1]Main effect: NS, not significant P>0.05. [2]No S x L interactions were significant.

Treatment of the piglet and/or sow had no significant effect on growth or feed conversion efficiency during the finisher growth phase. Similarly, there were no significant (P>0.2) main treatment effects on P2 backfat status, although there was a trend for a decrease in P2 following the treatment of the sow only, which appeared to be nullified by the administration of pST postnatally. The failure to observe any response to either treatment may be related to both the dose and the sensitivity of the target tissue in these physiological states.

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THE EFFECTS OF INSULIN-LIKE GROWTH FACTOR I-SUPPLEMENTED DIETS ON THE BRUSH BORDER OF EPITHELIAL CELLS IN THE SMALL INTESTINAL TRACT OF 36 HOUR OLD PIGLETS

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Insulin like growth factor I (IGF-I) administered with milk in bottle-fed pigs induces increases in intestinal mass, villous height and crypt depth (Burrin *et al.*, 1996). In a similar study Tivey *et al.* (1997) demonstrated that the small intestinal mass of piglets significantly increased when they were fed colostrum compared with milk. This response may be in part due to the higher concentration of IGF-I in colostrum. The aim of the present study was to further investigate the effect of IGF-I on the small intestine brush border (microvilli) in piglets fed colostrum or milk, with or without supplementary IGF-I.

Twenty newborn unsuckled Large White piglets were allocated to an initial slaughter group (ISG) and four treatment groups, as described by Zarrinkalam *et al.* (1999), to include piglets fed sows' colostrum (SC), sows' milk (SM), and sow's milk with supplementary IGF-I (SM+IGF-I and SM-2IGF-I). Animals in the ISG were killed at three hours of age, and piglets in the treatment groups were killed at 36 hours of age (one hour after their last meal). The small intestine was rapidly dissected, flushed with ice-cold phosphate buffered saline (PBS), blotted dry and samples taken from the mid-region of the duodenum and ileum. The specimens were fixed in 4.0% paraformaldehyde + 1.25% glutaraldehyde overnight at room temperature to prepare for transmission electron microscopy (TEM). They were post-fixed in 2% osmium tetroxide in PBS for 1 hour, followed by dehydration and embedding in epoxy resin. The samples were cut at 70 nm thickness, stained with uranyl acetate (5% in ethanol) and lead citrate (2%). Brush border regions were captured at a magnification of x 7,900 for later image analysis. For each animal, five epithelial cells were examined, and 15 measures of brush border height were made per cell. Data were analysed by ANOVA.

Table 1. The effect of diet on brush border height in the duodenum and ileum at 36 hours of age.

Height (nm)	ISG	SM	SC	SM+IGF-I	SM+2IGF-I	SED
Duodenum	1246^{b}	1392^{bc}	1017^{a}	1299^{b}	1470^{c}	104.1
Ileal	831	761	702	812	790	87.5

^{a,b,c}Values within rows with different superscripts are significantly different ($P \leq 0.05$).

The highest concentration of IGF-I supplementation (SM+2IGF-I) increased the height of the brush border in the duodenum by up to 45%. Brush border height was lower in piglets fed colostrum (SC) than ISG piglets and those fed sows' milk with or without supplemental IGF-I. This suggests that the positive effect of colostrum on gut physiology and function that is frequently observed (e.g., Burrin *et al.*, 1996; Tivey *et al.*, 1997) is not mediated by stimulating brush border height, but more likely via responses in villus height, crypt depth, intestinal length or enzyme activity. However, the provision of high concentrations of supplemental IGF-I (as in group SM+2IGF-I) may improve the ultrastructure of enterocytes and increase nutrient absorption by stimulating brush border height. This may have particular implications to pigs consuming milk that has a lower concentration of beneficial factors present in colostrum.

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K-DIFORMATE (Formi™LHS) IN DIETS FOR PIGS

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Antibiotic growth enhancers in pig production have become restricted due to concerns about resistant bacteria, animal welfare, environmental, and consumer issues, so alternatives are needed. Organic acids have received much attention as a potential alternative. Formic acid is one of the most effective organic acids against pathogenic bacteria, but its use has been limited by problems with handling, strong odour, and corrosion during feed processing and on the farm. The product, Formi™LHS, is a dry, odourless, easy to handle compound of potassium di-formate, and is an effective growth promoter in diets for weaner pigs (Paulicks *et al.*, 1996) and grower-finisher pigs (Øverland and Lysø, 1997). The objective of this study was to determine the effects of Formi™LHS on growth performance and carcass traits of grower-finisher pigs.

Ninety-six (Landrace x Yorkshire) x (Landrace X Duroc) pigs from 16 litters (27.1 kg and 105.0 kg initial and final body weight) were used. Pigs were allotted to treatments by weight, litter and sex, and were individually fed, so each pig was one experimental unit. There were 16 barrows and 16 gilts per treatment. The dietary treatments were: 1) a basal diet, 2) basal diet + 6 g/kg Formi™LHS, and 3) basal diet + 12 g/kg Formi™LHS. Pigs were fed twice daily according to a restrictive feeding scale providing a moderate feeding intensity during the grower period, followed by an increasing feeding intensity during the finisher period. The diets were comprised of barley, oats, wheat bran, fishmeal, meat and bone meal and canola meal. The diets provided 12.1 MJ ME and 154 g CP per kg diet, and 0.7 g lysine per MJ ME. The percentage of lean (lean-%) and fat (fat-%) in the carcass were determined by the primal cuts method as described by Øverland *et al.* (1999). The GP2Q carcass lean-% involved measuring the depth of the loin muscle and the back fat thickness at two sites: between the 10th and 11th rib, 6 cm from the midline and behind the last rib, 8 cm from the midline. Orthogonal polynomials were used to test linear responses of increased dietary levels of Formi™LHS.

By adding up to 1.2% Formi™LHS to diets, time to market was reduced by 4.8 days, average daily gain (ADG) was improved by 6%, average daily feed intake (ADFI) was increased by 3%, and feed:gain was improved by 2.8% (Table 1). The addition of Formi™LHS also reduced fat-% and increased lean-% in a dose-dependent manner.

Table 1. Effect of Formi™LHS in diets on performance and carcass traits of pigs.

Formi™LHS, %	0	0.6	1.2	SEM	Linear effect
Average daily gain (g)	863^{x}	886^{x}	915^{y}	10	0.0003
Average daily feed intake (kg)	2.41^{x}	2.45^{x}	2.49^{y}	1.6	0.006
Feed/gain	2.80	2.78	2.73	0.01	0.11
Carcass lean-%, GP2Q	54.1	54.1	54.8	0.4	0.18
Carcass lean-%, primal cuts	53.7	53.5	54.1	0.3	0.25
Carcass fat-%, primal cuts	19.4^{x}	19.2^{xy}	18.3^{y}	0.3	0.03

^{x,y}Values in rows with different superscripts are significantly different ($P \leq 0.05$).

The growth performance of grower-finisher pigs was significantly improved with the addition of Formi™LHS. The product Formi™LHS also significantly reduced carcass fat content and tended to increase carcass lean content. The highest performance was achieved with 1.2% Formi™LHS.

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WATER ADDITION TO PIGLET FEED: EFFECTS ON POST-WEANING GROWTH AND HEALTH

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Routine use of antibiotics in feed for farm animals was abandoned in Sweden in 1986. Since then alternatives have been sought to manage the growth checks and scours which frequently occur at weaning. From the early 1990's large doses of zinc oxide (2000 ppm) were used as a scour prevention routine in many herds. From 1998, however, inclusion of more than 250 ppm zinc oxide in piglet feed is allowed only on veterinary prescription. This decision by the Swedish Board of Agriculture has put the problems at weaning back on the agenda. Liquid feeding of piglets around weaning improves piglet performance (Pluske *et al.*, 1996; Lindberg *et al.*, 1997). The reason for this may be the feed ingredients used in liquid diets and/or the high water content. In the present study the effect on piglet performance of adding water to the diet was evaluated.

Twenty litters of purebred Swedish Yorkshire piglets were allocated at two weeks of age to either dry creep feeding (Dry) or the same diet (12.2 MJ ME and 155 g crude protein per kg) with water added to a dry matter content of 34% (Wet). Piglets were fed to appetite. For the Wet treatment feed was mixed with water in the morning and stored at room temperature until feeding, half of the ration was given in the morning and the remainder in the afternoon. Piglets were weaned at 5 weeks (± 3 days) and remained in the farrowing pen until the end of the experimental treatment at 9 weeks of age. Piglet feed intake, growth rate, health status and medical treatments were monitored. The effect of dietary treatment (Dry vs. Wet) on feed consumption and growth rate was examined using ANOVA with litter size as a covariate. The litter was regarded as the experimental unit in this analysis. The effect of diet on scouring frequency was tested with Pearson's χ^2-test with the piglet as unit.

Table 1. Effect of Dry vs Wet feeding of piglets from 2 to 9 weeks of age on feed intake, growth rate and scouring frequency (10 litters per treatment).

	Treatment		P-value[2], effect of	
	Dry	Wet	Treatment	Litter size[1]
Litter size	10.0	9.7		
Feed intake, g (air dry)/d				
3 weeks-weaning	22	32	NS	NS
Weaning-7 weeks	533	678	0.012	0.032
7-9 weeks	1010	1360	0.001	0.04
Growth rate, g/d				
3 weeks - weaning (5 weeks)	210	211	NS	0.01
Weaning-7 weeks	230	297	0.02	0.06
7-9 weeks	491	494	NS	NS
Piglet live weight at 9 weeks (kg)	20.1	21.5	0.09	
Numbers of piglets with weaning scours	43	28	0.039	

[1]Covariate. [2]NS, P>0.10.

Adding water to the piglet diet increased feed consumption, particularly after weaning, increased the growth rate for the two weeks following weaning, and decreased the frequency of weaning scours. In conclusion, addition of water to the diet of piglets may have a positive effect on their feed intake and health, thereby improving piglet weaning performance.

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CREEP FEED OFFERED AS A GRUEL PRIOR TO WEANING ENHANCES PERFORMANCE OF WEANED PIGLETS

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In theory, increasing nutrient intake prior to weaning by providing supplementary feed should increase weaning weight and enhance early post-weaning growth rate, both of which are positively correlated to subsequent performance (Miller *et al.*, 1999). In practice, supplemental milk fed prior to weaning does increase weaning weight (King *et al.*, 1998) but generally dry creep feeds have little or no effect due to low intakes (Pluske *et al.*, 1995). Liquid feeds are consumed more readily than dry feeds pre-weaning, therefore, offering creep feed as a gruel should enhance nutrient intake and improve weaning weight and post-weaning performance. The aim of this study was to test this hypothesis.

Fifteen crossbred litters (Landrace x Large White) were randomly allocated to one of three treatments; A) no supplementary feed (control); B) dry creep pellets; or C) diet B as a gruel (1:2 meal to water). Creep diets were fed to appetite from days 14 to 24. Daily intakes were recorded. Intact litters were weaned at 24 days of age into individual flat deck pens and offered the same weaner feed. Stocking density was standardised at 0.2 m^2/pig. Individual pig weights were recorded at weaning and on days 7 and 35 after weaning. In a second experiment comparing only treatments A and C, 4 piglets were slaughtered from each treatment at weaning and another 4 from each treatment 5 days later. Sections from the small intestine were examined histologically. Data were analysed by litter using the GLM procedure of Minitab 12.2. Starting weight was used as a covariate.

Table 1. Piglet weights at 14 days of age and at weaning, average daily gain for week 1 (ADGwk1) and for the first 35 days (ADG35d) after weaning, and creep feed intake (FI) between day 14 and weaning.

Supplement	Control	Dry Creep	Gruel	SEM
Start weight (kg)	4.6	5.1	5.0	0.4
Total prewean FI/pig (g DM)	0^a	90.7^b	374.4^c	15.1***
Wean weight (kg)	6.9	6.5	6.7	0.4
ADGwk1 (g)	48.6^a	58.3^a	125.0^b	21.8*
ADG35d (g)	316.5	368.3	415.9	29.7

^{a,b}Values in the same row with different superscripts are significantly different. *P≤0.05, ***P≤0.001.

Piglets that received gruel ate more creep pre-weaning (P<0.001) and grew faster post-weaning than piglets which had received dry or no supplementary food (ADGwk1 P<0.05, ADG35d P<0.1). However, there was no improvement in weaning weight. It appears that there is an advantage to providing creep as a gruel. Examination of sections from the small intestine of A and C piglets revealed that villous height in gruel-fed piglets was lower (P<0.05) than that in control pigs at weaning (319 vs 431 μm ± 18.5, respectively; mean ± SEM) but there was no reduction in villous height by 5 days after weaning in gruel-fed pigs. In contrast samples from control pigs demonstrated a dramatic decrease in villous height 5 days after weaning (245 ± 20.1 μm; P<0.05). This suggests that feeding gruel prior to weaning aids gut adaptation to solid diets.

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WEANING WEIGHT AND DAILY LIVE WEIGHT GAIN IN THE WEEK AFTER WEANING PREDICT PIGLET PERFORMANCE

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Weaning weight is positively correlated to subsequent performance (Mahan and Lepine, 1991) but not to growth rate in the week after weaning (ADGwk1) (Pluske and Williams, 1991). However, pig producers often claim that the heaviest pigs at weaning do not perform as well as some of their lighter littermates. The objective of this study was to investigate this hypothesis over a wide range of weaning weights.

A total of 612 crossbred piglets (62.5% Large White, 25% Landrace, 12.5% Duroc) were weaned into fully-slatted flat deck-pens. The piglets were weaned at 27 ± 0.3 days of age (mean ± SEM) and 7.9 ± 0.06 kg live weight (LW). Nine piglets were allocated to each pen (1.37 m x 1.43 m). All piglets were individually weighed at weaning and 7, 14 and 20 days after weaning. Food and water were provided *ad libitum* throughout the 20-day experimental period. The piglets received 1 kg starter diet per pig (17.5 MJ DE/kg, 17.5 g total lysine/kg) followed by a second stage starter diet (16.5 MJ DE/kg, 16.5 g total lysine/kg) until day 20. Data were analysed using ANOVA to determine differences among piglets in different weaning weight ranges (see Table 1) and by simple linear regression to determine relationships among weaning LW, ADGwk1 and day 20 LW.

Table 1 Average daily gain (ADG) from weaning to 20 days after weaning for piglets of different weaning weights.

LW group (kg)	≤6.5	>6.5 ≤7.5	>7.5 ≤8.5	>8.5 ≤9.5	>9.5	SEM
Overall ADG (g)	341^{a}	374^{b}	414^{c}	408^{c}	428^{c}	8.63***
ADGwk1 (g)	200^{a}	223^{ab}	249^{ab}	261^{ab}	280^{b}	25.4*

***Significant difference among groups P≤0.001; *significant difference among groups P≤0.05. ^{a,b,c}Numbers in the same row with different superscripts differ significantly from each other P≤0.05.

Weaning LW and ADGwk1 were found to be the best predictors of day 20 LW (r^2 = 0.489, P<0.001 and 0.413, P<0.001, respectively). Multiple regression analysis showed that these effects were additive; Day 20 LW = 3.73 (SEM = 0.297) + 1.25 (SEM = 0.037) weaning LW + 8.92 (SEM = 0.304) ADGwk1 (rsd = 1.209, r^2 = 0.798, P<0.001). Regression of weaning LW against ADGwk1 accounted for less than 2% of the variation in ADGwk1 (r^2 = 0.011). Analysis of performance to 20 days after weaning by weight group showed that piglets of >7.5 kg LW performed similarly whilst piglets 7.5 kg LW and below had lower weight gains.

The data confirms that weaning weight is a significant predictor of subsequent piglet performance, however, ADGwk1 is of similar importance. Therefore, management practices that promote daily live weight gain in the first week after weaning should be given equal emphasis to those that maximise weaning weight. Improving ADGwk1 by increasing feed intake and/or feed conversion efficiency should provide significant improvements in subsequent growth rate. In absolute terms, heavier piglets at weaning outperformed lighter piglets although there was no difference in weight gain for piglets above 7.5 kg LW.

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INFLUENCES OF LITTER ORIGIN AND WEANING WEIGHT ON POST-WEANING PIGLET GROWTH

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A check in piglet growth is almost invariably observed following weaning. However, the severity or duration, or both, of this response varies among individual piglets and to date has not been definitively explained. Previous work has demonstrated that growth rate in the first week after weaning affects subsequent performance but is poorly correlated to weaning weight (Miller *et al.*, 1999). Therefore what determines early post-weaning growth rate? The objective of this study was to investigate the effects of litter origin and weaning weight on subsequent piglet growth and thus to establish to what extent these factors determine post-weaning performance.

One hundred and twenty crossbred piglets (JSR Healthbred) offered creep feed from 14 days of age were weaned from 21 sows of mixed parity at 26 ± 0.4 days of age (mean ± SEM) and 7.9 ± 0.13 kg live weight. The piglets were allocated to one of four pens of 10 piglets (3 replicates over time) each balanced for litter origin, live weight (LW) and gender. Groups were housed in fully-slatted, flat-deck pens (1.99 m^2). Water and food (16.5 MJ DE/kg, 16.5 g lysine/kg) were provided *ad libitum*. Piglets were weighed at weaning and on days 7, 14 and 20 of the experiment, and average daily gains (ADG) were calculated. Data were analysed by the GLM and regression procedures of Minitab 12.2.

Table 1. Significance of weaning weight, litter origin, gender and week 1 growth rate (ADG 1-7) influences on piglet live weight and average daily gain (ADG) to day 20 post-weaning.

	Weaning weight	Litter origin	Gender	ADG 1-7
Live weight				
day 0	-	***	NS	-
day 7	*** (R^2 = 0.653)	***	NS	*** (R^2 = 0.389)
day 14	*** (R^2 = 0.563)	***	NS	*** (R^2 = 0.397)
day 20	*** (R^2 = 0.456)	***	NS	*** (R^2 = 0.406)
ADG				
day 1-7	NS	***	NS	-
day 8-14	NS	***	NS	*** (R^2 = 0.171)
day 15-20	NS	***	NS	*** (R^2 = 0.146)
day 1-20	NS	***	NS	*** (R^2 = 0.652)

NS, not significant P>0.05; ***significant correlation P≤0.001.

Regression analysis indicated that weaning weight had a significant though apparently declining association with live weight at days 7, 14 and 20 subsequent to weaning (Table 1). Piglet ADG in week 1 was significantly associated with day 20 live weight but was not influenced by weaning weight (Table 1). Piglet ADG in week 1 was found to differ significantly among litters (P<0.001). Within litter, siblings showed similar relative ADG regardless of weaning weight. Gender did not affect either weaning weight or ADG to 20 days post-weaning.

The results of the study confirm the importance of high weaning weight in determining subsequent live weight superiority but suggests that the significance of this influence reduces with time. The results presented here also confirm that weight gain during the first 7 days after weaning is a major determinant of post-weaning growth (Miller *et al.*, 1999). Weight gain during this critical 7 day post-weaning period is influenced greatly by litter origin. Genotype, lactogenic immunity and behavioural traits are possible maternal influences of progeny performance and thus require further investigation. The role of supplementary feeding pre-weaning as a modifier of gut function should also be considered.

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DEFINING THE TRYPTOPHAN REQUIREMENT FOR PIGS BASED ON PROTEIN DEPOSITION RATE: 45 TO 75 KG BODY WEIGHT

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A factorial approach for calculating amino acid requirements based on protein deposition rate (PD) has allowed more precise estimates of essential amino acid requirements across diverse genetic and environmental circumstances. The objective was to define the apparent ileal digestible tryptophan (dTRP) requirement for PD in barrows (B) and gilts (G) grown from 45 to 75 kg body weight (BW).

Pigs were F1 crosses from Camborough 15 females and Canabrid males (Pig Improvement Company, Canada). Each treatment consisted of five pigs of a single sex per pen with four replicates per treatment. Pigs were given *ad libitum* access to water and one of five pelleted diets containing 14.2 MJ DE/kg. Crystalline tryptophan was added to the basal diet to obtain the other diets. Other essential amino acids and non-essential amino acid nitrogen were calculated to be non-limiting. Whole-body protein content was estimated using real-time ultrasound; coefficients of determination were 82 and 98%, for B and G, respectively (Miller, K.D. and Wilson, E., personal communication). Tryptophan requirement for maintenance and content in whole-body protein were taken as 11 mg/kg $BW^{0.75}$/d and 0.0078 g/g, respectively (Fuller *et al.*, 1989). Requirement was determined as the intersection of lines using the two-line segment model (Kim *et al.*, 1983). Marginal efficiency of utilisation was calculated from the slope of the first segment.

Table 1. Effect of apparent ileal digestible tryptophan to energy ratio (dTRP/MJ DE) on average daily feed intake (ADFI), gain to feed ratio (G/F) and protein deposition rate (PD).

		dTRP/MJ DE					CV[1]	Effects[2]				
Variable		0.035	0.049	0.070	0.085	0.106		S	L	Q	S x L	S x Q
ADFI, g/d	B	1244	1898	2210	2152	2556	6.4	*	*	*	*	ns
	G	1466	1966	2051	2121	2165						
G/F, g/g	B	0.258	0.298	0.380	0.408	0.368	7.8	ns	*	*	ns	ns
	G	0.273	0.303	0.390	0.405	0.397						
PD, g/d	B	60	102	155	161	169	5.1	*	*	*	ns	ns
	G	57	94	142	153	162						

[1]CV, coefficient of variation (%). [2]S, sex; L, linear; Q, quadratic; ns, not significant (P>0.10); *P≤0.01.

There was a curvilinear increase (P<0.02) in ADFI for B and G as dTRP content increased. Based on PD, the requirement was 0.066 and 0.067g dTRP/MJ DE for B and G, respectively. At requirement, PD for B and G was 156 and 142 g/d, respectively. Marginal efficiency of utilisation of dTRP for PD was 46% and the requirement for dTRP was 1.68 g/100 g protein deposited, which agree with other publications (Burgoon *et al.*, 1992). Using the amount of apparent ileal digestible lysine (dLYS) needed to deposit whole-body protein as 8.98 g/100 g (Lorschy and Patience, unpublished), dTRP/dLYS was 19%. Information from this study may be used in growth simulation models to predict dTRP requirement.

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DEFINING THE THREONINE REQUIREMENT FOR PIGS BASED ON PROTEIN DEPOSITION RATE: 45 TO 75 KG BODY WEIGHT

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A factorial approach based on protein deposition rate (PD) aids in applying estimated requirements across diverse genetic and environmental circumstances. The objective was to define the apparent ileal digestible threonine (dTHR) requirement for PD in barrows (B) and gilts (G) grown from 45 to 75 kg body weight (BW).

Pigs were F1 crosses from Camborough 15 females and Canabrid males (Pig Improvement Company, Canada). Each treatment consisted of five pigs of a single sex per pen with four replicates per treatment. Pigs had *ad libitum* access to water and one of five pelleted diets containing 14.1 MJ DE/kg. Crystalline threonine was added to the basal diet to obtain the other diets. Other essential amino acids and non-essential amino acid nitrogen were calculated to be non-limiting. Whole-body protein content was estimated using real-time ultrasound; coefficients of determination were 82 and 98%, for B and G, respectively (Miller, K.D. and Wilson, E., personal communication). Threonine requirement for maintenance and content in whole-body protein were taken as 46 mg/kg $BW^{0.75}$/d and 0.0376 g/g, respectively (Fuller *et al.*, 1989). Requirement was determined as the intersection of lines using the two-line segment model (Kim *et al.*, 1983). Marginal efficiency of utilisation was calculated from the slope of the first segment.

Table 1. Effect of apparent ileal digestible threonine to energy ratio (dTHR/MJ DE) on average daily feed intake (ADFI), gain to feed ratio (G/F) and protein deposition rate (PD).

		dTHR/MJ DE					CV[1]	Effects[2]				
Variable		0.236	0.293	0.350	0.407	0.465		S	L	Q	S x L	S x Q
ADFI, g/d	B	2765	2557	2498	2552	2403	2.4	*	*	*	*	ns
	G	2235	2085	2068	2119	2178						
G/F, g/g	B	0.265	0.380	0.390	0.386	0.397	2.0	*	*	*	*	ns
	G	0.375	0.465	0.476	0.469	0.464						
PD, g/d	B	118	145	149	154	149	2.6	*	*	*	ns	ns
	G	107	134	136	136	137						

[1]CV, coefficient of variation (%). [2]S, sex; L, linear; Q, quadratic; ns, not significant (P>0.10); *P≤0.001.

There was a curvilinear decline (P<0.001) in ADFI for B and G as dTHR content increased. Based on PD, the requirement was 0.31 and 0.29 g dTHR/MJ DE for B and G, respectively. At requirement, PD for B and G was 152 and 135 g/d, respectively. Marginal efficiency of utilisation of dTHR for PD was 70%, and the requirement for dTHR was 5.42 g/100 g protein deposited, which agrees with other publications (Adeola, 1995). Using the amount of apparent ileal digestible lysine (dLYS) needed to deposit whole-body protein as 8.98 g/100 g (Lorschy and Patience, unpublished), dTHR/dLYS was 60%. These data, in concert with other information in the literature, validate the use of factorial estimates of amino acid requirements under commercial conditions.

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A REVIEW - NEONATAL AND WEANER PIG: MANAGEMENT TO REDUCE VARIATION

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Abstract

The most important difference among piglets at birth is body weight. This difference has a major impact on subsequent performance and complicates management of the facilities. The present paper is concerned with the management of the neonate and the weaner piglet to reduce variation in body weight. In the first section, the extent of variation in birthweight is considered. Emphasis is given to the effect of birthweight on body composition and performance including total mortality and growth. For a given genotype, the most important factor influencing variation in birthweight is litter size. The variation is established early in foetal life (< 30-35 days of gestation). Small piglets (<1.0-1.1 kg) have a fewer number of muscle fibres, which may affect their subsequent growth potential. Competition at the udder explains both a higher postnatal mortality in small piglets and a lower growth of the survivors. The milking ability of sows accounts for differences in growth among litters. These result in a two-fold difference in weaning weight, both among-and within-litters. In the second section, practices used to reduce variation in birth and weaning weights are evaluated. Cross-fostering within a few hours of birth is a necessity to save piglets when their number exceeds the number of teats. However, evidence that cross-fostering reduces mortality is weak, whereas minimising the practice is recommended to reduce the impact of some diseases in infected herds. Split-weaning has only a temporary beneficial effect on the uniformity of piglet weight. However, supplementation with a milk replacer to litters from low-milk producing sows and to litters with small piglets grouped on a sow appears beneficial. Grouping of piglets by weight is common practise at weaning. However, the effects on subsequent performance and maintenance of uniformity in the group are still unclear. Overall, providing supplemental milk replacer to small piglets and/or to litters of low-milk producing sows both during the nursing period and the period of post-weaning diet transition appears promising, by improving the piglets' performance thus helping them to cope with the growth lag immediately following weaning.

Introduction

A major goal of the pig producer is to maximise the number of pigs weaned. This implies reducing stillbirths and maximising survival of piglets born alive. The weaned pig must be of sufficient quality (adequate body weight and gut development, optimal humoral immunity) to ensure high levels of growth in the post-weaning and finishing periods.

It would simplify management a great deal if pigs in a litter were like "peas in a pod" at weaning (English *et al.*, 1977). As implied in this quotation, the within-litter variation in body weight is a factor relevant to pig production (Thompson and Fraser, 1986). This variation is already present at birth and is a major cause of perinatal mortality. However, the within-litter variation in birth weight is not simply a reflection of litter size. Several factors influence the growth of piglets, within- and between-litters. These mainly include competition among littermates for milk, milking ability of sows, and health of piglets and the sow.

In major pig-producing countries, the production of pigs has developed towards specialised farms with hundreds or thousands of animals. As such, pig housing has become an intensive production industry. The principle of all-in, all-out is a major control element. Therefore a short turnover rate of pigs through each facility is of major interest.

Reducing variation in piglet body weight is an important goal of the pig producer. A complete discussion of the subject is beyond the scope of a single paper and therefore

the present paper will consider two main sections. Firstly an assessment of differences in birthweight, body composition and growth potential of piglets while attempting to understand these differences, and secondly the effects of these differences on performance including mortality and growth. In the second section, practices to reduce the variation in piglet body weight, (a) within- and among-litters during the suckling period and, (b) during the post-weaning period, are evaluated.

Piglets are not equal at birth with respect to body weight

At birth, piglet body weight can vary enormously. The combination of small weight piglets and variation in body weight is often reported as a major cause of perinatal mortality. Furthermore, weight differences may influence postnatal growth and development. Therefore, it is relevant to assess the extent of variation in birthweight, the effects of birtweight on body composition and growth potential of piglets and to understand the origin of these variations.

Body weight at birth: extent of the variation.

In addition to differences in birthweight existing within litters, the differences in piglet birthweights between litters may be large. Within-litter variation is usually associated with total mortality during the sucking period. Because variation in birthweight influences weaning weight, both within- and among-litter variation in birthweight generate a problem in managing the facilities to combine piglets into different groups at weaning when the all-in all-out concept of raising pigs is followed. Thus, reducing both types of variation is a major concern for pig producers.

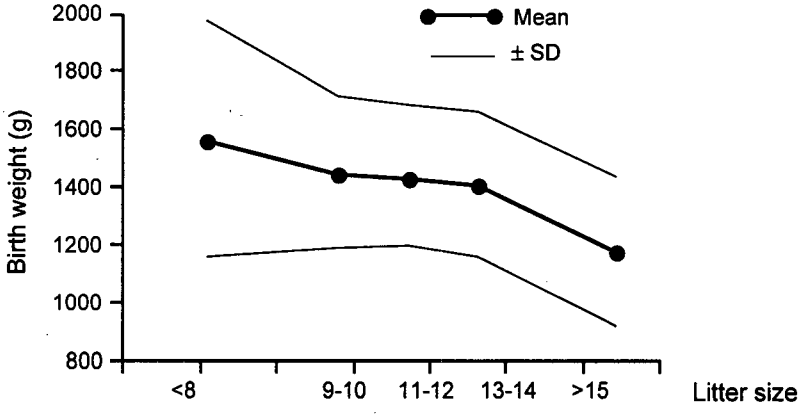

Figure 1. *Effect of litter size (total born) on within litter variation in birthweight (F. Madec, personal communication).*

Overall, the within-litter component of variance of birthweight represents 60 to 70% of the total variance, with the coefficient of variation (CV) ranging between 18 and 25% (Blackwell, 1989; Leenhouwers *et al.*, 1999; J.P. Bidanel, personal communication; F. Madec, personal communication). At weaning, CV of body weight is of the same order or higher than at birth. Variations in birthweight depend on genotype, parity and litter size. Genotype has no effect on within-and among-litters CV of birthweight. Within- and among-litter CV of 17.9 and 16.4%, respectively, are reported when four lines of specialised dam lines are compared (Leenhouwers *et al.*, 1999). A similar figure is found when the European Large White breed is compared to the Chinese Ja Xin and Meishan breeds (J.P. Bidanel, personal communication). Surveys by Rydhmer (1992) and F. Madec (personal communication) indicate that the highest CV occurs in both small (<7-8 piglets, CV=26.3%) and large (>15 piglets, CV=22.3%) litters (Figure 1). In addition, an increase

in litter size is associated with both a decrease in absolute mean birthweight amounting to 35 g for every additional pig born and an increased proportion of weak piglets (<1.0 kg). Increasing litter size from 7-9 to 13 piglets results in an increase of 0.2 small pigs for every extra pig born. For larger litter sizes (>13), 0.7 small pigs are born for every extra pig born. Furthermore, increased litter size has problems associated with mortality and, sometimes, rearing of the supernumerary animals (Figure 2).

Figure 2. *Effects of litter size on the within litter distribution of birthweight (J.Y. Dourmad, personal communication).*

Body composition.

Is the difference between a small and a large piglet at birth only a difference in weight? "Small" is an arbitrary term. It is applied to newborns weighing 75-80% of the mean birthweight of the litter, on the basis that these pigs have a greater risk of dying before weaning (Rydhmer, 1992; Léon and Madec, 1992; Herpin *et al.*, 1996; J.P. Bidanel, personal communication). As such, newborn pigs from modern genotypes weighing 1.0-1.1 kg are considered as being small. Small pigs have smaller muscles and organs, with the exception of the brain (Flecknel *et al.*, 1981). However, relative to birthweight, the length of the small intestinal is greater than in normal piglets (Xu *et al.*,1994), although the number and height of the villi is reduced in small piglets. Segmental activity of lactase and specific activity of lipase were lower in small piglets, but whether this impairs digestive capacity is uncertain. Within a large range of birthweights (0.5-1.86 kg), de Passillé and Hartsock (1979) did not observe marked differences in gross chemical composition, whereas Ojamaa *et al.* (1980) found that liver and muscle glycogen concentrations were marginally affected. However, runt pigs, i.e., pigs weighing less than 65% of the mean birthweight of the litter (Hegarty and Allen, 1978), have lower respiratory enzyme activity in the *longissimus dorsi* (Hayashi *et al.*, 1987) suggesting a decreased oxidative metabolism in this muscle. A reduced number of thyroid receptors has also been reported in skeletal muscle of small-for-gestational age piglets (Dauncey and Geers, 1990). There is a positive within-litter correlation between circulating concentrations of insulin-like growth factor I (IGF-I) and birthweight (Herpin *et al.*, 1992). However, small pigs do not differ from normal littermates in terms of circulating concentrations of growth hormone (GH), somatomedin C, triiodothyronine (T3) or tyroxine (T4), (Scanes *et al.*, 1987).

Muscles of smaller pigs have fewer total fibres as a consequence of a smaller secondary fibre population (Powell and Aberle, 1980; Wigmore and Stickland, 1983; Handel and Stickland, 1987). At 111 days of gestation, the total number of fibres in the semitendinosus muscle of the largest and the smallest littermates were 331 and 253×10^3, respectively, with a ratio of secondary to primary fibres being 14.5 and 10.5, respectively

(Wigmore and Stickland, 1983). At slaughter at 105 kg, small piglets at birth are also reported to have fewer but larger muscle fibres (Hegarty and Allen, 1978). This is presumably a reflection of a reduced potential for growth during the grower and/or finisher phases because protein synthesis in the newborn is relatively resilient to change; it is not altered by intra-uterine growth retardation induced by protein restriction of the sow (Davis *et al.*, 1997). Furthermore, since the number of fibres is fixed at or shortly after birth (Stickland and Goldspink, 1973) and this is an important determinant of muscle mass in later life (Miller *et al.*, 1975), small piglets do not have the capacity to fully catch up to larger counterparts.

Factors accounting for differences in body size and growth potential of the piglet

Foetal growth is ultimately controlled by the genetic endowment of the foetus but can be influenced by a variety of factors including nutrition. Little emphasis has been placed on the genetic improvement of birthweight and its variability. Heritability of birthweight (h^2 = 0.1, Rydhmer, 1992) and of variation in birthweight (h^2 = 0.07, Knol, 1999) are very small, thus offering little promise of genetic improvement. Environmental factors such as the position in the uterine horn may affect the development of the conceptus (Dziuck and McKay, 1986; Tarrat *et al.*, 1995), whereas uterine crowding results in a decrease in mean foetal weight (Dziuck and McKay, 1986; Père *et al.* 1997a) and more variation in foetal weight (Legault *et al.*, 1995). Interestingly, hemi-hystero-ovariectomised sows have increased ovulation rate in the remaining ovary, resulting in overcrowding in the uterine horn and a higher rate of foetal mortality (before 35 days of gestation) with both the weight of surviving foetuses and that of placentas being lower and more variable (Père *et al.*, 1997a; Legault *et al.*, 1995). As demonstrated by Chen and Dziuck (1993), a large number of foetuses in the uterus during the first part of gestation may affect subsequent embryo development, suggesting that a high ovulation rate is associated with lower and more variable birthweight.

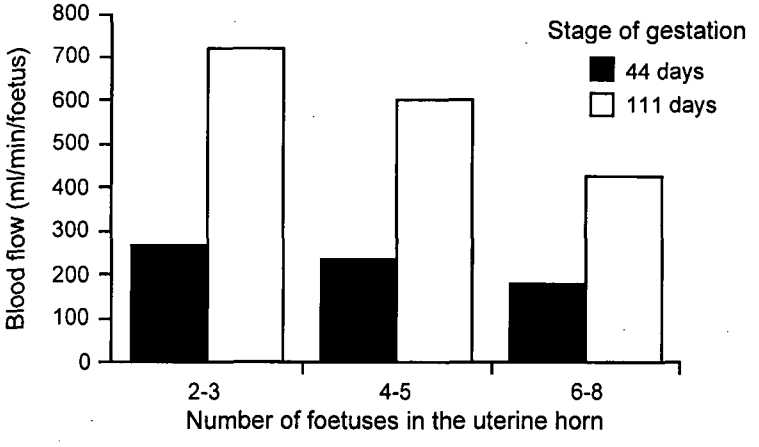

Figure 3. *Uterine blood flow in relation to the number of foetuses present in the uterine horn and stage of gestation (Père et al., 1997b).*

Foetal nutrition is primarily determined by the amount of nutrients transferred from the mother to her foetuses, which depends on both the size of the placenta and the blood flow. This in turn is a function of the number of foetuses. A study conducted in sows during late pregnancy (DeRoth and Bisaillon, 1980) reported a high correlation (r = 0.73) between the placental size and the foetal weight. Similarly, a close relationship (r = 0.83) was found between placental blood flow and foetal weight (Wootton *et al.*, 1977). Since uterine blood flow per foetus decreases with the number of foetuses (Père *et al.*, 1997b; Figure 3), this provides an explanation why piglets from larger litter sizes are lighter at

birth. It also provides some evidence that the within-litter distribution in birthweight is programmed relatively early in foetal life. This view is substantiated by the findings of Wise *et al.* (1997) that small foetuses within the litter can be identified as early as 30 days of gestation. Similarly, van der Lende *et al.* (1990) found that the within-litter variation in birthweight is already established at 35 days of pregnancy.

Foetal nutrition depends to some extent on the maternal nutritional state. The relationship between the level of feeding of the pregnant sow and the birthweight of piglets is well documented (Cromwell *et al.*, 1989; Pluske *et al.*, 1995). However, and except under extreme nutritional conditions such as fasting or protein deprivation, level of feeding of the pregnant sow has only moderate effects on foetal growth. It is estimated that foetal birthweight increases by approximately 4 and 8 g for each additional daily megajoule digestible energy intake in primiparous and multiparous sows, respectively (Henry and Etienne, 1978). However, one recent study (Dwyer *et al.*, 1994) suggests that maternal nutrition can have subtle effects on the foetal development and subsequent performance of the progeny. These authors found that doubling the recommended level of feeding immediately before fibre hyperplasia (days 25 to 50 of pregnancy) increased the production of secondary myofibres in the semitendinosus muscle by 9 to 13% without effect on average body weight. An important feature of this increase is that it caused an increase in fibre number in the small piglets in the litter with no changes in the larger piglets. These data suggest that small piglets have experienced nutritional deficiency *in utero* due to placental insufficiency and have less secondary fibres. This could be, to some extent, increased by adequate manipulation of the diet of the pregnant sow. However, further studies are needed to substantiate these results.

In summary, data indicate that, in addition to differences in body size existing between litters, the within-litter variation in birthweight is itself a factor relevant to piglet production. Differences are established early in foetal life and are associated with a significant reduction in the population of secondary muscle fibres. This, and the fact that there seems to be a physiological limit to fibre hypertrophy (Hegarty and Allen, 1978), suggests that small piglets have reduced growth potential in later life.

The effects of variation in birthweight

The effects of variation in birthweight are expressed in terms of perinatal mortality, postnatal growth, and eventually in altered carcass traits at market weight.

Total mortality

In major pig-producing countries, total mortality is commonly within the range of 15 to 22% of the total born. Approximately one-third are classified as stillbirths and two-thirds die between birth and weaning (pre-weaning mortality). Birthweight is the single greatest predictor of survival in pigs (Stanton and Carroll, 1974) with both total and relative birthweight (birthweight expressed as a percentage of the litter mean) affecting the viability of pigs. However, there is no absolute threshold for birthweight below which pigs have an increased probability of dying. This is illustrated by the fact that both stillbirths and pre-weaning mortality are similar in the Ja Xin and the Large White breeds although the average birthweight of the Ja Xin pigs is only 50% that of the Large White counterparts (0.7 vs 1.4 kg) (J.P. Bidanel, personal communication). The occurrence of a higher total mortality in piglets of low body weight in both the stillbirths and those dying before weaning, as compared to the survivors, has been reported previously (DeRoth and Downie, 1976; Spicer *et al.*, 1986; Dyck and Swiestra, 1987; Léon and Madec, 1992; Herpin *et al.*, 1996; Leenhouwers *et al.*, 1999; Figure 4). Low-birthweight piglets have an increased risk of dying by asphyxia during delivery (Herpin *et al.*, 1996). In addition, the degree of asphyxia suffered by the piglets during birth is closely related to their subsequent viability (Randall, 1972; Herpin *et al.*, 1996). In the study of Herpin *et al.* (1996), asphyxia during parturition was directly responsible for the mortality of 5.5% of the live-born pigs, which in turn accounted for 25% of early postnatal mortality (birth to 10 days, Table 1). The findings of Leenhouwers *et al.* (1999) are in agreement with the latter and indicate a positive relationship between the number of stillbirths and the pre-

weaning mortality of live-borns. However, it is still unknown why smaller piglets are more prone to experience a higher degree of asphyxia during parturition than their heavier littermates.

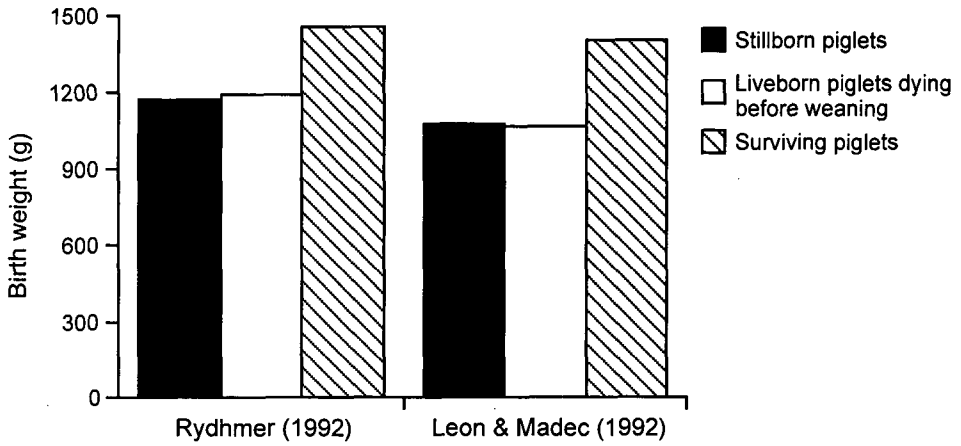

Figure 4. *Birthweight of stillborn piglets, liveborn piglets dying before weaning and piglets surviving to weaning.*

Causes accounting for the higher occurrence of mortality in small piglets born alive have been reviewed (Le Dividich *et al.*, 1998). Briefly, piglets of low body weight have less body energy stores, are more susceptible to cold (Curtis *et al.*, 1967; Le Dividich *et al.*, 1991) and, therefore, are more prone to experience deep hypothermia and be overlain by the sow. They take longer to achieve their first sucking (Hoy *et al.*, 1994), and within the litter, are less competitive at the udder than their heavier littermates. Consequently, the smaller piglets consume less colostrum (Fraser and Rushen, 1992) resulting in a reduced acquisition of protective immunoglobulin (Ig) and a reduced supply of energy necessary to sustain a high metabolic rate. Further, insufficient consumption of colostrum and hence Ig has important effects on subsequent piglet performance. This is exemplified by the fact that piglets dying between 5 days and weaning had lower amounts of IgG than the survivors at 12 (Blecha and Kelley, 1981) and at 24 h of life (Hendrix *et al.*, 1978). Edwards and Rooke (1999) found a positive correlation between passive immunity (IgG concentration at 7 days) and humoral immunity at weaning at 28 days (IgG concentration at weaning). These authors also reported that post-weaning growth was positively related to IgG concentration at weaning.

Table 1. Characteristics at birth of liveborn piglets dying during early postnatal life compared with survivors (Herpin *et al.*, 1996).

	Piglets dying before 10 days	Survivors
Birthweight (kg)	1.06	1.34
Birth - 1st suckle interval (min)	55	23
Rectal temperature at 24 h (°C)	36.5	38.6
Blood pH	7.21	7.27
Blood lactate (mg/dl)	49	32
Blood glucose (mg/l)	1020	590
Plasma noradrenaline (ng/ml)	73	42

Postnatal growth

The sucking phase

Birthweight is important in terms of postnatal growth. Weaning weight is highly correlated with birthweight (r =0.46 to 0.66; McBride *et al.*, 1965; McConnell *et al.*, 1987; Dick *et al.*, 1987; Caugant and Gueblez, 1993; Rousseau *et al.*, 1994). Overall, the weaning weight increases by 0.35 to 1.07 kg for every 100 g increase in birthweight (Caugant and Gueblez, 1993; Rousseau *et al.* 1994; Mahan and Lepine, 1991; Dunshea *et al.*, 1997b). The higher growth rate of large piglets results from a higher milk intake; heavy pigs consume 28, 30 and 30% more milk per sucking than do the small ones at day 17, 19 and 24 d of lactation, respectively (Pluske *et al.*, 1996a). This higher milk intake did not relate to a higher ingestive capacity because when fed *ad libitum*, pigs ingested the same amount of colostrum (g/kg body weight) regardless of their birthweight (Le Dividich, unpublished observations). Instead, it is likely related to both the nursing position and the demand for milk by sucking pigs.

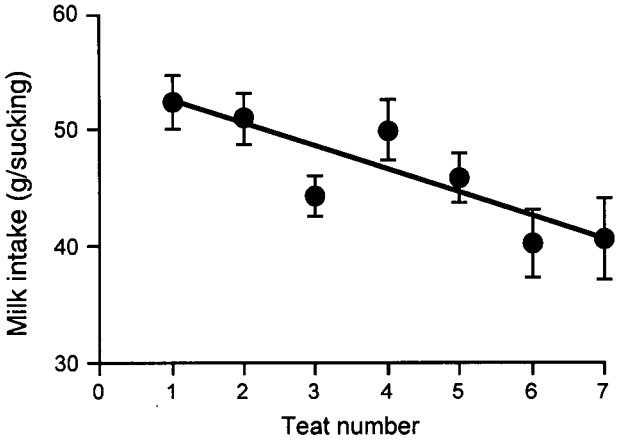

Figure 5. *Relationship between average milk intake of piglets and teat order, with teats numbered 1-7 from the anterior to the posterior end of the udder (Pluske and Williams, 1996a).*

Pigs of high birthweight tend to select the anterior teats (Hartsock *et al.*, 1977), likely because of the closer proximity of the sow's grunting (Jeppesen, 1982) and the greater ease of extracting the first colostrum from the teats (Fraser and Lin, 1984). Several studies reported a higher weight gain in pigs using the most anterior teats (Fraser and Lin, 1984; Hoy and Puppe, 1992) which could suggest that milk yield is dependent on teat position. In experiments in which the sow was hand milked, a marked decline in both colostrum and milk yield was found between the most anterior teats and the most posterior teats (Fraser and Lin, 1984; Salmon Legagneur, 1958). Pluske and Williams (1996a) found a strong negative relationship between average milk intake in piglets at day 22 of lactation and teat number (Figure 5). Similarly, Hoy and Puppe (1992) found that pigs adopting the anterior teats were 1.5 kg heavier at weaning than those adopting the posterior teats. The concentrations of IgG was also higher in the first colostrum obtained from the top of the udder than from the bottom (Bland and Rooke, 1997). Perhaps most importantly are the findings that piglet body weight itself may contribute to differences in milk intake (Auldist and King, 1995). King *et al.* (1997) reported that fostering two-week old pigs onto newly farrowed sows increased milk production of the sow by 26% during the first week of lactation, whereas fostering newborn pigs onto sows on their third week of lactation reduced subsequent milk production by 22%. It is suggested that heavier piglets are more efficient at draining their teats than the lighter, less active, piglets and may therefore stimulate greater milk flow. Low milk intake during the first postnatal

week has important effects on growth, protein metabolism, and muscle and gut development of piglets. Whole-body protein synthesis at 7 days of age is reduced by 47% in piglets fed sows' milk at the rate of 100g/kg BW/d compared with those fed 300g/kg BW/d (Marion *et al.*, 1999) (Table 2). Rate of milk intake has no effect on the total number of muscle fibres in both *longissimus dorsi* and *rhomboïdeus* muscles, but cross-sectional area of secondary fibres is dramatically decreased in both muscles of piglets fed the lower amount of milk. These piglets have also a higher proportion of fibres containing foetal myosin heavy chains in their muscles suggesting that a low amount of milk intake soon after birth delays muscle maturation (L. Lefaucheur, personal communication). Segmental activity of lactase and height of villi of the small intestine are also reduced in piglets fed the low amount of milk suggesting that the digestive capacity is lower in piglets consuming less milk (Le Huérou-Luron *et al.*, 1999). However, to what extent these effects persist when normal milk intake is established remains to be determined. Whatever it may be, it follows that the weight of a pig relative to its littermates has a considerable impact on future gain and differences in weaning weight. For example, for an average weaning weight of 8.1 kg at 25-28 days of age, 12.5% of piglets were <6.0 kg whereas, 16.0% were >10 kg (J.Y. Dourmad, personal communication). Similar results are reported by Cranwell *et al.* (1995).

Table 2. Effect of amount of sow milk intake during the first postnatal week on whole-body protein turnover in piglets at 7 days of age.

Milk intake (g/kg BW/d)	100	200	300
Ks (% per day)[1]	5.8	10.2	14.2
Whole-body protein synthesis ($g/d/BW^{0.75}$)	10.7	16.9	20.9
Protein deposition ($g/d/BW^{0.75}$)	4.5	14.6	19.7

[1]Fractional rate of protein synthesis (percent of protein mass synthesized in a day) (After Marion *et al.*, 1999).

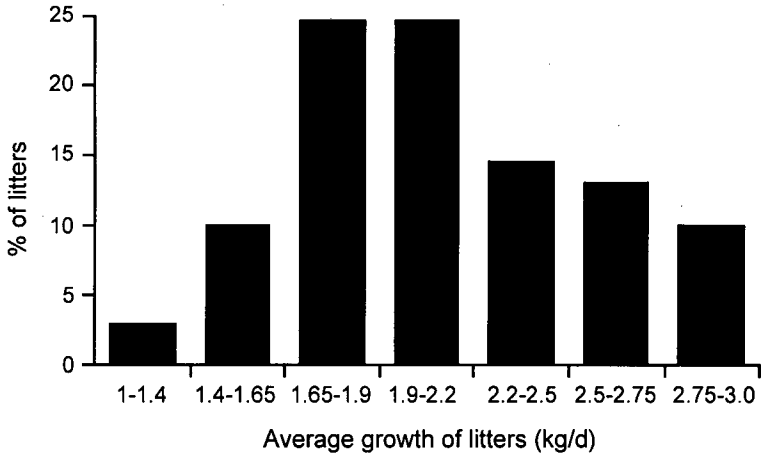

Figure 6. *Distribution of mean growth of litters during the first week of lactation. Data recorded in 69 litters (litter size adjusted to 11 piglets) from a commercial farm (M. Drillet, personal communication).*

Between-litter differences in growth may also be large, reflecting differences in sow milk production capacity that exist among breeds, crosses and among individuals of the same genotype. These variations are detected soon after birth as exemplified by the results of Thompson and Fraser (1998) that showed piglet growth ranging from 5 to 227g/day during the first 3 days of life. The distribution of litter growth during the first postnatal week recorded in a commercial farm is shown in Figure 6. The average daily gain (ADG) was 210 g/piglet. However, it is noticeable that 38% of litters achieved ADG >200g/piglet, 49% had an ADG ranging between 150 and 200 g/piglet, whereas, 13% had ADG <150g/piglet. A survey involving 756 litters (J.Y. Dourmad, personal communication) provides values of litter growth between birth and weaning ranging from 1.38 to 2.60 kg/day per litter (data adjusted to 8.4 piglets/litter at weaning). According to Thompson and Fraser (1988), approximately 45% of the variation in the mean weights of litters at day 14 is explained by the gain during day 0-2, with differences established by day 14 being perpetuated and sometimes enhanced during the remainder of lactation (Thompson and Fraser, 1986). Moreover, they suggest that low initial weight gain predisposes litters to highly variable weaning weight most likely caused by a higher competitive pressure at the udder when milk intake is low.

In summary, these data indicate (i) that a two-fold difference in piglet mean weaning weight both within- and among-litters is very common in commercial herds and (ii) the importantance of the first week of lactation, that is, the period of establishing lactation on the subsequent performance of the litters. It is suggested that more attention will have to be paid to this period.

Weaning to slaughter phase

Weaning weight has marked effects on both post-weaning growth responses and subsequent performance in the growing-finishing phase. Based on data in Figure 7, the weight of individual pigs at the end of the post-weaning phase increases by 1.6 to 2.1 kg for every 1 kg increase in weaning weight. Furthermore, every extra 1 kg at weaning saves 2.5 to 3.2 extra days to attain the slaughter weight (Mahan and Lepine, 1991; Mahan, 1993; Tokach *et al.*, 1992; Kavanagh *et al.*, 1997). The higher growth of the heavier piglets is largely attributed to a higher feed intake and is likely due to their higher capability to compete at the feeder (Graham *et al.*, 1981). However, whether low weaning weights result in fatter carcass at slaughter is still uncertain (Powell and Aberle, 1980; Hegarty and Allen, 1978; Sève and Bonneau, 1986; Caugant and Gueblez, 1993, Kavanagh *et al.*, 1997)). Nevertheless, under practical conditions, the age at slaughter is very variable. In France, for example, slaughter weight standardised to 105 kg is attained at a mean age of 174 days, with 8% being aged <160 days and 15% being aged >190 days. This variation in age at slaughter makes the management of facilities more complex.

In summary, evidence is given that small piglets are disadvantaged compared to large ones with regard to survival and performance. Now the question is, to what extent can the light piglets to catch up growth to the large piglets without assistance? According to England (1974), birth weight is not a meaningful predictor of genetic potential for future performance, as h^2 is near zero. England and Keeler (1965) reported that both runt (birthweight = 0.74 kg) and normal (birthweight = 1.24 kg) artificially-fed piglets achieved similar weight gain at 17 days of age. Lecce (1971) found that piglets weighing <1.0 kg at birth triple their body weight by two weeks of age. Indeed, piglets are reported to have higher growth potential when adequately fed than observed in sucking piglets (Benevenga *et al.*, 1990). In contrast, Ritacco *et al.* (1997) failed to show any growth response by runt piglets to an increase in artificial milk intake, suggesting that they may have a reduced absorptive capacity. However, performance of these piglets has not been evaluated past the first few weeks of life.

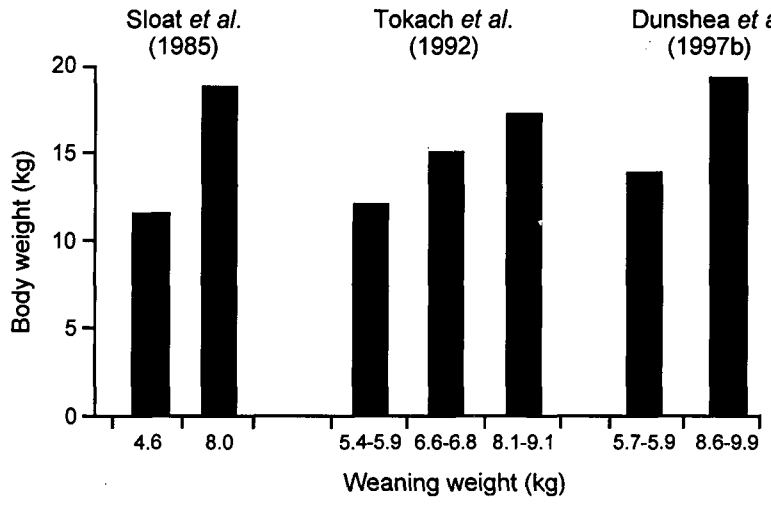

Figure 7. *The effects of weaning weight on the final weight during the post-weaning period. Weaning ages and durations of the post-weaning period were 21 and 28 days, respectively (Sloat et al., 1985; Tokach et al., 1992) and 28 and 21 days, respectively (Dunshea et al., 1997b).*

Management of the neonate to reduce variation

In batch-farrowing systems, which includes synchronisation of farrowings, management practises to maximise survival and to achieve high and uniform weaning weights within the litter are directed towards reducing competition among littermates. These practises include: (i) grouping piglets of similar body weight together within litters (cross-fostering); (ii) split weaning, that is, a permanent removal of part (the heaviest piglets) of the litter a few days before complete weaning; and to a lesser extent (iii) selective tooth clipping of the heaviest piglets; and (iv) design of the farrowing crate. Reducing differences between litters is directed towards: (i) improving sow milk production; and ultimately (ii) compensating for the insufficient sow milk production by providing adequate milk substitute to piglets.

Reducing variation among littermates

Cross fostering

Fostering piglets from one litter to another is routinely used to avoid an excessive number of piglets in a given litter while equalising weights within litters to avoid excessive competition among litter-mates. In France, for example, all sizes of litters are involved in piglet exchanges soon after birth, however small (<7 piglets) and large (>15 piglets) litter sizes are of most concern (Figure 8). Cross-fostering is assumed to improve uniformity of body size and is necessary in an attempt to save the newborn pigs in excess of the number of functional teats of the dam. It is usually practised a few hours after birth before the formation of the nursing order, but can be continuously practised up to weaning.

Some studies report that cross-fostering soon after birth reduces pre-weaning mortality. English and Bampton (1982) found that cross-fostered litters had a 40% improvement in piglet survival to weaning. Another study (Marcatti Neto, 1986) indicated that piglets with a birthweight of 800 g or less had 62.5% pre-weaning mortality when they were left with their dam, compared to 15.4% if fostered into groups with similar birthweight. In contrast, Kirkwood *et al.* (1998) concluded in their study involving 120 sows, that there was no advantage in creating litters of uniform body weight. At farrowing, these authors formed experimental groups of three sows. They grouped the lightest 50% piglets from two litters on one sow and the remaining heavier piglets on the

other. A third sow served as control (intact litter). Using this protocol, they found a higher mortality among litters of uniform light birth-weight with a lower weaning weight of the survivors (Table 3). Comparisons made within the intact litters of heavier and lighter halves provide similar trends. In fact, evidence supporting a higher mortality in litters with a high standard deviation is weak (English *et al.*, 1977; Fahmy *et al.*, 1978). Further, on the basis that small piglets are less able to extract milk from teats, improved growth of these piglets in response to cross fostering is questionable. More likely, the practice would result in an enhanced gap between litter growth. According to van der Lende and de Jager (1991), piglets of low birthweight have a higher death risk independent of their status within the litter. They suggested that the correlation between mortality rate and variation in birthweight could be due to the presence of small individuals and not to variation itself.

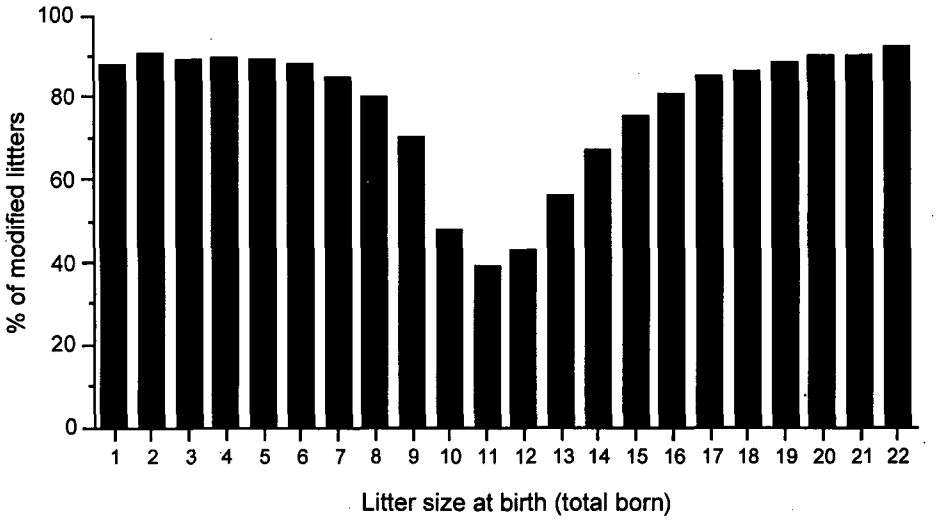

Figure 8. *Percentage of litters involved in piglet exchange at birth (J. Dagorn, personal communication).*

Table 3. Effects of cross fostering on growth and survival of piglets (Kirkwood *et al.*, 1998).

	Fostered litters		Intact litters		
	Heavy	Light	Overall	Heavy[1]	Light[1]
Litter size	10.3	10.1	9.7	9.9	9.6
Average birth weight (kg)	1.46	1.02	1.27	1.46	1.07
Average weaning weight (kg)	8.66	7.72	8.68	9.26	7.76
Mortality (birth-weaning) (%)	8.7	20.0	11.4	6.4	19.5

[1]Heavier and lighter halves of intact litters.

Cross-fostering is sometimes continuously practised up to weaning so that individuals that fall back are transferred to a smaller litter. Straw *et al.* (1998a) reported that on a third of Canadian and American (Midwestern) swine farms, 40% of piglets that are fostered are older than one week of age. However, these repeated mixings have detrimental effects on the behaviour of sows and piglets. Indeed, sucking piglets develop teat fidelity within the first days after birth and usually suck from the same teat until weaning (de Passillé *et al.*, 1988). Studies of Price *et al.* (1994) reported that only 25-50% of piglets fostered after two days, i.e., once the nursing order is usually established, have sucked within the first six hours after adoption. Similarly, studies of Robert and

Martineau (1997) indicated that fostering after four days of age was associated with teat disputes where resident and fostered piglets fought to gain access to a specific teat. Sows had increased milking frequency after fostering, but many of them were non-productive. Also, foster sows showed frequent signs of nervousness and snapped at the piglets more often than control sows, with this aggressive behaviour being directed towards fostered piglets. It follows that disruption of nursing order and fighting between fostered and resident piglets is usually associated with a reduced weight gain of the fostered piglets and sometimes of the residents. Horrell and Bennett (1981) found that the weight of piglets fostered at seven days of age was reduced at 14 days after fostering to 79% of that of non-fostered littermates. Similarly, in the study of Robert and Martineau (1998), continuous fostering reduced the weight of fostered piglets by 13% at weaning at 21 days compared with that of the control piglets (4.5 vs 5.4 kg). Compared with fostering limited to the first two days of life, continuous cross-fostering reduces the variation of body weight at weaning at 19 days by 41%, thus producing more uniform litters (Straw *et al.*,1998b). However this advantage is offset by the 20% lower weight (4.3 vs 5.3 kg) recorded at weaning in the continuously fostered piglets. In these conditions, it is doubtful that the reduced variation in weight at weaning is desirable if it is associated with a reduction in growth rate. Overall, continuous cross-fostering is stressful for the piglets and sows and may reduce the performance of piglets. These factors, and the extra labour required to continually foster piglets, may limit its use in practice.

Table 4. Piglet performance before and after split weaning.

		Split-weaned[1]		Control		References
		Heavy	Light	Heavy	Light	
Live weight (kg)	day 22	7.0	5.5	7.7	5.4	Pluske and
	day 29	8.2	7.7	8.6	6.7	Williams
	day 62	22.0	19.3	22.1	19.3	(1996a)
Live weight (kg)	day 21	6.7	5.3	5.9		
	day 28	-	7.2	7.5		Vesseur *et al.*
	day 63	21.9	-	-		(1997)
	day 70	(24.4)[2]	23.6	24.0		
Piglet mortality (%)		4.7	2.1	3.7		
Piglet treated for disorders (%)		19.7	7.8	14.2		

[1]Heavy piglets were weaned at 21-22 days of age. [2]Value estimated assuming that during the seventh week after weaning piglets achieved similar growth rate to that during the rearing period, i.e., 361 g/d.

An additional important point to recognise is the fact that cross-fostering can have negative effects on the piglet, especially on their health status. McCaw *et al.* (1996) have shown that extensive cross-fostering maintains a continuous cycle of Porcine Reproductive and Respiratory Syndrome (PRRS) transmission thereby increasing pre-weaning mortality and uncontrolled post-weaning porcine PRRS infections. More recently, Madec *et al.* (1999) described a Porcine Wasting Disease (PWD) in France that is largely similar to the Post-weaning Multisystemic Wasting Syndrome (PMWS) described in North America (Harding, 1997). Typically, the pig of 2 to 3 months is at the critical period with mortality rate being as high as 20%. A survey involving 199 litters from four herds revealed that 52% (145/281) of pigs that died came from 29 litters (Madec *et al.*, 1999). Although the mode of contamination is still uncertain, there is some evidence that both the sow, and contact between piglets, are involved in the spreading of the disease. Hence, it follows that reduced mixing of litters is strongly recommended to minimise the

impact of the disease in affected herds, thus limiting the possible benefit of cross-fostering.

Split weaning

Selective weaning of the heaviest piglets of the litter some days before normal weaning improves the growth of the lightest piglets left with the sow allowing, to some extent, for these piglets to catch up in growth to their heavier littermates at normal weaning. Indeed, with the exception of Matte and Close (1987) and Gilbertson *et al.* (1989), most authors (Edwards *et al.*, 1985; Wu *et al.*, 1985; English *et al.*, 1987; Mahan, 1993; Pluske and Williams, 1996a; Vesseur *et al.*, 1997) reported an improved growth rate in light piglets left with the sow compared to their counterparts in intact litters. This improvement ranged from 27 to 61% depending on the age at the split and on the duration of the split-weaning interval. This practise, termed fractionated, partial or split-weaning, reduces the competitive pressure at the udder at an age when the supply of milk by the sow does not fulfil the energy needs of the piglets, and results in an enhanced milk intake in the light piglets. Indeed, piglets remaining with the sow after the split are reported to exploit more teats (Sibly *et al.*, 1987), distribute themselves more evenly along the udder, and to use the more productive anterior teats previously occupied by the heavy piglets (Pluske and Williams 1996a). Measurements made by Pluske and Williams (1996a) indicated that light piglets remaining with the sow for 7 days after the split consumed 49% (64 vs 43g/suckling) more milk and grew 61% (305 vs 189g/day) faster than their counterparts in intact litter (Table 4). This, coupled with the growth check occurring during this period in weaned heavy piglets, results in light and heavy piglets being of similar weight at 29 days. However, when the light piglets in the split litters are weaned, they also suffered a growth check so that the difference that existed at the split persisted when pigs were about 9 weeks of age. Similarly, Mahan (1993) reported that the gain advantage achieved by leaving piglets with the sow for an additional week gradually diminished during the post-weaning period. In summary, this practise temporarily promotes the growth of small piglets.

Other practices

Many farmers routinely clip the teeth (canines and the third incisors) soon after birth to prevent the damage that these teeth may cause to litter-mates or to the sow's udder. Fraser and Thompson (1991) and Robert *et al.* (1995) tested the potential benefits of selective tooth clipping by leaving intact the teeth of the smallest piglets of a litter to make them more competitive. In litter sizes of 9-11 piglets or less, tooth clipping has no marked effects on survival or growth of these piglets. However, in higher litter sizes (12-14 piglets) where the competitive pressure was higher, piglets of low birthweight had lower mortality (32 vs 40%) and achieved greater weight gain (+ 7.6 to 11%) if their teeth were left intact compared with piglets which had their teeth clipped. The within-litter variance of weights at 21 days was 15% lower in experimental than in control litters making weaning weights more uniform. However, in the study of Robert *et al.* (1995), these advantages to the small piglets were offset by a slightly higher mortality and lower weight gains among the heavier piglets competing against small littermates with intact teeth, so that overall mortality and weight gains were unaffected. It follows that tooth clipping works by assisting smaller littermates with intact teeth at the expense of the larger ones, but is no substitute for fostering.

Sow crate design may also affect the within-litter variation in weaning weight. Comparing two types of farrowing crates that have either horizontal bars or angled vertical bars on both sides, Thompson and Fraser (1986) found no effect of the crate on the mean body weight of piglets at 35 days. However, the within-litter variation in piglet body weight was lower in crates having vertical bars (Fraser and Thompson, 1986), suggesting that crates with vertical bars could reduce the competitive pressure among the litter. However, Rhode Parfet *et al.* (1989) failed to demonstrate this advantage.

Supervision of farrowings, providing weak piglets with sow colostrum or colostrum and milk substitute, and positioning these piglets in a heated area are efficient tools to improve survival. For further discussion, see review by Le Dividich and Herpin (1999).

Reducing variation between litters

Improving sow milk production

As mentioned earlier in this paper, variations in growth among litters may be very large, which implies that sow milk production is highly variable and potentially a constraint to weight at weaning. Factors contributing to variation in sow milk production include the genetic potential and the provision of adequate nutrients required for production of the constituents of milk. These factors have been extensively reviewed recently in "The Lactating Sow" (Verstegen *et al.*, 1998) and therefore will not be specifically discussed here. Manipulation of environmental conditions such as the photoperiod or increasing the nursing frequency using auditory stimuli are reported to increase sow milk production. Marbry *et al.* (1982) compared the effects of a 16 h photoperiod with an 8 h photoperiod and found that piglets exposed to 16 h light suckled more often than piglets exposed to 8 h light, and consequently were heavier at 15 days of age (7.2 vs 5.8 kg). Similarly, Stone *et al.* (1974) found that auditory stimuli such as cyclic playback of feeding sounds shortened the nursing interval and increased sow milk production. However, it is doubtful that these manipulations could sufficiently improve milk production of low milk-producing sows. However, in batch farrowing systems, one can reasonably assume that these practises may improve milk production of high milk-producing sows, but this would maintain or even exacerbate the differences between litters.

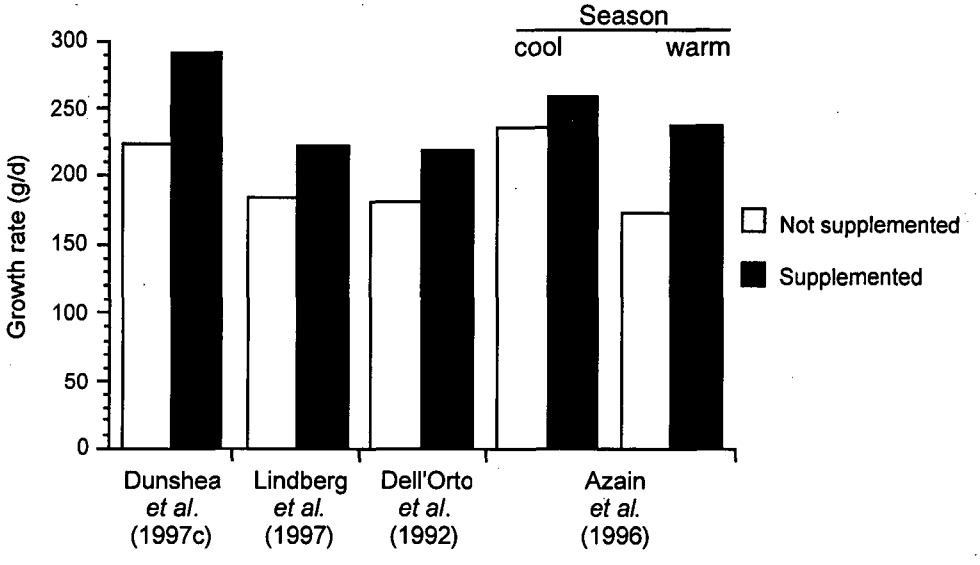

Figure 9. *Effect of milk substitute supplementation on the growth rate of piglets. Piglets were supplemented from day 10 to day 20 (Dunshea et al. 1997c), day 14 to day 35 (Lindberg et al., 1997), and day 1 to day 21 (Azain et al., 1996).*

Provision of supplemental milk substitute

Providing litters that exhibit poor growth with milk substitute could, to some extent, compensate for insufficient milk production of the sows. Advantages claimed include reduced pre-weaning mortality, heavier weaning weights and faster post-weaning

growth. Litters having access to supplemental liquid milk substitute grew 10 to 38% faster than non-supplemented litters (Figure 9). Interestingly, pigs supplemented with milk during lactation grew faster until 120 days of age than piglets suckled by the sows (Dunshea *et al.*, 1997a). This practise does not affect sow milk production before weaning at 20 days of age (Dunshea *et al.*, 1997b) whilst it improves the body condition of sows weaned at 35 days (Lindberg *et al.*, 1997). Also of interest are the findings of Azain *et al.* (1996) showing that providing litters with milk substitute during the warm season when sow feed intake and milk production are reduced, is more effective to improve litter growth (+38% higher than non-supplemented litters) than during the cool season (+10%). Therefore, providing a supplemental milk substitute to poor milk-producing sows could reduce the differences in litters growth associated with high and low milk-producing sows. This practice could also promote the growth of litters of small piglets grouped on a sow and warrants future research. However, the practice needs to be evaluated against the cost of the extra labour and the relative cost of the milk substitute.

Management of weaner pigs to reduce variation

Weaning is associated with grouping of piglets from different litters, and changes in nutrition and physical environment. Each factor potentially affects the growth of piglets. However, as subsequent performance is at least partially dependent on the weaning weight, it may be important to group pigs by weight. This practise in conjunction with a post-weaning feeding strategy could allow, to some extent, pen-mates of lower body weight to catch up in growth to the heavier pen-mates. However, physical factors such as space allowance and access to feeders also induce competition, thus affecting performance.

Grouping of pigs

Mixing of piglets from different litters is associated with vigorous fighting (Algers *et al.*, 1990) leading to the formation of a new social order. However, as in fattening pigs (Gonyou *et al.*, 1986) the aggressive behaviour is more pronounced: (i) in groups of uniform weight than in groups of heterogeneous weight, and (ii) in groups of uniform heavy weight than in groups of uniform light weight (Rushen, 1987; Francis *et al.*, 1996). This aggressive behaviour may cause injury and activate the sympathetic nervous system (Fitko *et al.*, 1992). However, the duration of fighting does not usually exceed a few hours. This is confirmed by the short-term increase in heat production which is observed after mixing piglets (Heetkamp *et al.*, 1995) and by the behavioural studies of Rushen (1987). Similarly, an increase in plasma glucose concentration, which reflects the activation of sympatho-adrenal function, is only recorded on day-1 post-mixing (Puppe *et al.*,1997).

Mixing of unfamiliar piglets did not exacerbate the set-back at weaning (McGlone *et al.*, 1987) and had no marked effects on performance at 28-30 days post-mixing (Friend *et al.*, 1983; Backshaw *et al.*, 1987; McGlone *et al.*, 1987) or even promoted growth (Pluske and Williams, 1996c). However, McGlone *et al.* (1987) found that small piglets gained faster when they were held in heterogeneous groups than in uniform groups.

The effects of grouping piglets by weight on subsequent performance are unclear. Combining the three trials of Francis *et al.* (1996), long-term performance of uniform groups was generally unaffected compared to litter or heterogeneous groups. However, the question is raised as to what extent the uniformity of the groups is maintained throughout the post-weaning period. According to McGlone *et al.* (1987) pens of uniform-weight pigs remained uniform during the post-weaning period, and Kornegay *et al.* (1985) reported that the CV in weight of pen-mates remained essentially constant during the post-weaning period. However these results conflict, to some extent, with the findings of Tindsley and Lean (1984) which show that CV of mixed-weight groups (18.0 kg (s.e. 4.5)) changed little over a 10-week period (17.0 to 17.8%), whereas CV of uniform weight groups (18.0 kg (s.e. 0.5)) increased as the trial progressed from 2.0 to 13.4%. However, it is suggested that grouping piglets by weight is beneficial to the uniformity of the group.

Restricted floor space and/or access to a feeder may create competition among pen-mates, thus affecting piglet performance. At a constant floor allowance, there is a small decrease in daily gain as the number of piglets was increased (Kornegay and Notter, 1984; McConnell *et al.* 1987). However, when space allowance is below optimum, daily gain decreases due to a reduced feed intake (Le Dividich, 1979; Giles *et al.*, 1998) most likely reflecting competition at the feeder. However, the effects of feeder access on feed intake and hence on performance are unclear. Comparing a multi-space feeder, a wet single-space feeder and a dry single-space feeder, Pluske and Williams (1996c) failed to show any effect of type of feeder on piglet performance suggesting that piglets adjust their feeding behaviour to the type of feeder. Clearly, there is a need for future research to quantify the effects of multiple stressors, including mixing, crowding, type of feeder on feeding behaviour and performance.

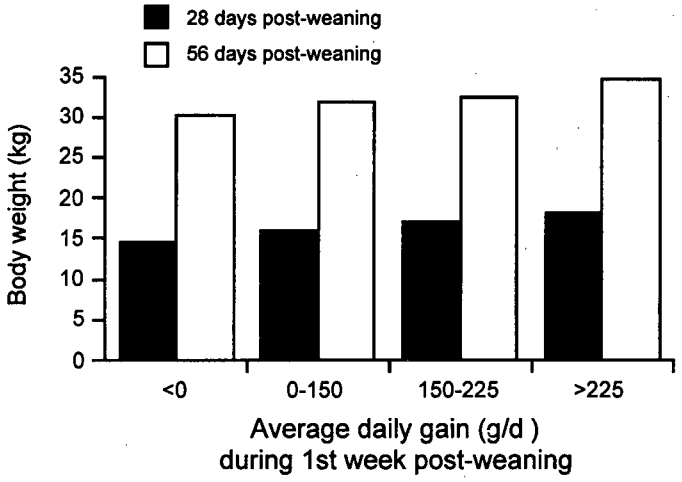

Figure 10. *Effects of daily gain during the first post-weaning week on the body weight of pigs at 28 and 56 days post-weaning (Tokach et al.. 1992).*

Feeding strategy

When offered between 2 and 4 weeks of age, the amount of creep food consumed before weaning is usually very small (Pluske *et al.*, 1995). Weaning is associated with a period of underfeeding during which piglets learn to eat dry food. It results in a growth "check", the severity of which has a major impact on subsequent performance (Figure 10). Tokach *et al.* (1992) and Azain (1993) reported that piglets gaining well (225-340g/day) during the first week post-weaning reached market weight some 10 to 28 days prior to piglets that exhibited poor gain (0-110g/day) during the first week post-weaning. Pigs of low body weight have less body fat reserves (Sloat *et al.*, 1985) and hence are less able to withstand the period of underfeeding. In addition, weight at weaning may affect the digestive capacity of the piglets. Amounts of pancreatic amylase (de Passillé *et al.*, 1989; Cranwell *et al.*, 1997) and trypsin, lipase and colipase (Cranwell *et al.*, 1997) are higher in large than in small nursing piglets. Similarly, feeding the young piglet at maintenance affects growth, morphometry and digestive capacity of the small intestine (Nunez *et al.*, 1996). All these changes would indicate that, at equal age, a heavier piglet at weaning may have a more developed digestive tract and a better ability to cope with the transition to the post-weaning diet, and explain in part, the differences in post-weaning performance observed between large and small piglets during the post-weaning period. Thus, grouping piglets by weight at weaning may allow for separate diets matched to the digestive capacity to be used for weaned piglets. However, piglets provided with a complex starter diet high in energy and dairy milk products failed to catch up to the growth of heavier piglets (Mahan and Lepine, 1991; Mahan, 1993). Nevertheless, from

data of Dunshea *et al.* (1997a) and Azain (1997), one can assume that providing supplemental milk substitute to piglets during both the sucking period and/or during the post-weaning diet transition should help light piglets to cope better with the stressors associated with weaning and improve subsequent performance of all pigs.

Conclusion: the need for research

This paper has discussed a number of practices used to reduce variation in growth of piglets, within and among litters during the nursing period and between pens during the post-weaning period. It is clear that a greater number of studies need to be conducted to substantiate the results.

However, the pig sector is constantly on the move. The development of hyperprolific breeds introduces new challenges, particularly at the piglet stage. Large litter sizes are more and more frequent, with the ensuing problems of increased intrapartum deaths, number of small piglets and variation in birthweight, and rearing of supernumerary piglets. Because of the greater competition at the udder, there is the possibility that colostrum and milk availability may impair survival and growth of the survivors. Therefore, it is suggested that more research should be focused on: (i) nutrition of the pregnant sow and pre-natal foetal nutrition to improve foetal birthweight and uniformity of the litter; (ii) birth process to improve the efficiency of farrowing; (iii) factors initiating and controlling colostrum production by the sow; and, (iv) strategies of rearing the supernumerary piglets, including adoption by a nurse sow, and very early weaning of piglets.

At weaning, one consistent finding has been a large variation, both within and among litters, in the time piglets start to eat the weaning diet. The practice of feeding piglets with liquid diet (Geary *et al.*, 1996), or providing supplemental milk substitute to piglets during the transition to the post-weaning diet appear promising in improving piglet performance. This, and the development of the immune system, a previously neglected area in relation to post-weaning performance, warrant future extensive research.

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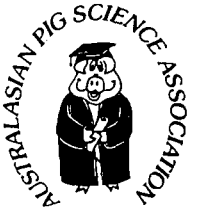

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A REVIEW- LEPTIN: A REGULATOR OF FEED INTAKE AND PHYSIOLOGY IN SWINE

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Abstract

Leptin is a small hormone produced by adipose tissue and subsequently secreted into the bloodstream. This hormone is involved in the regulation of feeding behavior, metabolism, reproduction and immunology. Review of the literature indicates that leptin may function as a regulator of overall energy balance in the normal pig. It is a signal synthesized in the peripheral energy stores (adipose tissue) in increasing quantity as the energy stores increase in volume. This signal is secreted by the adipose tissue and acts at the peripheral tissues and in the central nervous system to reduce feed/energy intake in a counter-regulatory manner. Leptin may have effects in peripheral tissues as well as in the central nervous system as receptors are present on many tissues. Thus, leptin has been demonstrated to alter nutrient partitioning through metabolic actions on muscle and adipose tissue. The onset of puberty and subsequent fertility mandate a positive energy balance for reproduction. Data suggests that leptin may contribute to the overall signal for the onset of puberty through actions both in the central nervous system and at the ovary. In addition, leptin can promote development of the immune system. Starving animals have depressed immune systems and low serum leptin levels. Leptin administration stimulates macrophage and T cell growth and activity. The potential application of leptin to swine will be dependent upon acquiring the knowledge necessary to alter these biological processes in such a manner as to improve production efficiency of the pig.

Introduction

Leptin was discovered as a result of experiments demonstrating that some mice have a genetic mutation that produces excessive feed intake and subsequent gross obesity. The actual genetic prevents the synthesis of a specific protein which has since been named leptin (Zhang *et al.*, 1994). Leptin is produced primarily in fat cells from where it is subsequently secreted into the bloodstream. Therefore, by definition, leptin is an endocrine hormone. Leptin was shown to reduce feed intake in mice and was proposed be a potential component to an effective treatment for human obesity (Campfield *et al.*, 1995; Halaas *et al.*, 1995; Pelleymounter *et al.*, 1995). Investigations into the actions of leptin have revealed numerous functions for this protein in regulating feed intake, metabolism, reproduction and immune function (for reviews see Flier, 1998; Fruhbeck *et al.*, 1998; Houseknecht *et al.*, 1998). Each of these physiological actions of leptin has direct relevance to animal agriculture (Figure 1). Proper understanding of the physiology of this peptide and the potential use of this hormone in the future may enhance the efficiency of pig production.

The majority of leptin research has been performed in rodents. Therefore, the question must be raised as to how this information, and its potential application, is relevant to the swine industry. Few studies have so far been performed using leptin in swine. Synthetic leptin is made by recombinant technology and the current technology does not permit the commercially viable production of leptin for systemic use in swine *in vivo*. However, recombinant porcine (Barb *et al.*, 1998) and ovine leptin (Gertler *et al.*, 1998) have been successfully synthesized. These recombinant leptins for domestic animals have permitted some initial analysis of the *in vivo* effects of leptin, but by administration into the CNS rather than systemically (Barb *et al.*, 1998). A single administration of recombinant porcine leptin into the lateral ventricle of the brain was demonstrated to reduce feed intake by up to 90% in gilts (Barb *et al.*, 1998). Morrison *et al.* (1998) have reported that chronic infusion of ovine leptin over a 7 day period can also produce a decrease in cumulative feed intake in ewe lambs. These studies demonstrate

that leptin has a similar function in pigs and sheep as in the rodent. Thus, for the sake of this review, the rodent studies serve as a guide to potential areas toward which leptin research may be directed within the swine industry.

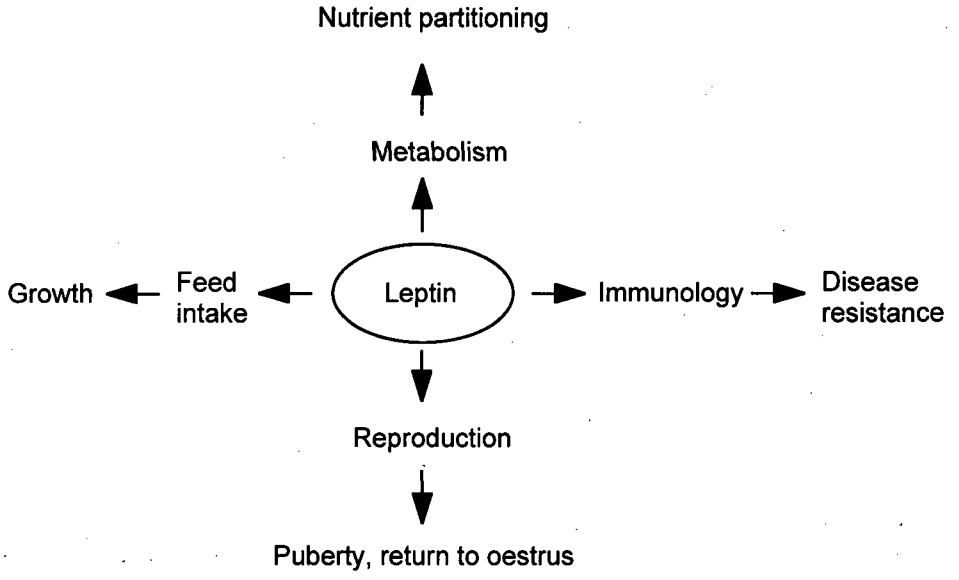

Figure 1. *Diagram of the potential impact of leptin on animal physiology and its relevance to animal agriculture.*

Leptin gene expression

A major limitation to our understanding of leptin physiology in swine is the current lack of adequate detection methods for serum leptin. Consequently, most data available for pigs relies on levels of tissue mRNA expression, rather than the actual amount of leptin protein. Ramsay *et al.* (1998) have demonstrated that leptin mRNA level in swine adipose tissue varies in proportion to changes in serum leptin, as determined by western analysis. Obese swine had greater leptin mRNA abundance and serum leptin concentrations than non-obese, crossbred pigs (Figure 2). Spurlock *et al.* (1998) have reported that leptin mRNA abundance correlates with fat mass ($r=0.74$, $P<0.0001$) and percentage of fat ($r=0.72$, $P<0.0001$) in swine, but no serum protein analysis was performed. Also, Minton *et al.* (1998) reported that circulating leptin concentrations in beef cattle were positively correlated with carcass fatness. These data provide some level of support for relating tissue leptin mRNA data, or adiposity, to physiological changes in serum leptin.

The complete sequence of the leptin gene has been characterized (Bidwell *et al.*, 1997). Several polymorphisms have been identified in the pig leptin gene, but no polymorphisms were detected within the open reading frame that can be conclusively associated with fat or lean deposition (Jiang and Gibson, 1999). This is not surprising as few polymorphisms in the leptin gene that can be associated with fat deposition or obesity have been identified in other species and these are in untranslated regions (Fitzsimmons *et al.*, 1998; Hager et al, 1998). However, further studies are necessary in swine with various breeds and genetic lines to exclude the possibility of the role of leptin gene polymorphisms as predictors of fat deposition.

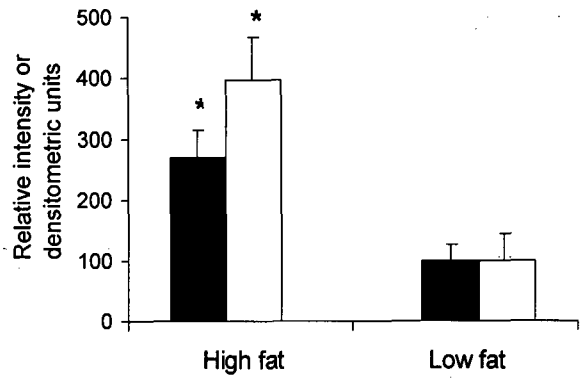

Figure 2. *Leptin mRNA abundance in dorsal subcutaneous adipose tissue (■) and porcine leptin expression in sera (□). Tissue and sera were obtained from the USDA line of pigs selected for increased backfat (High fat) versus tissue from contemporary crossbred pigs (Low fat = Landrace x Yorkshire). Northern analysis was performed with 20 μg total adipose tissue RNA and data were quantified by phosphorimager analysis and expressed as relative intensity. Leptin protein in 3 μl of sera following SDS gel electrophoresis, transfer and blotting of nitrocellulose membrane with an anti-human leptin antibody and data expressed as densitometric units (Derived from Ramsay et al., 1998). *Significantly different from crossbred values (P≤0.05, n=3).*

Regulation of leptin mRNA levels has been explored under a number of conditions using adipose tissue from swine. In general, hormonal regulation of leptin expression in pigs is similar to that in humans or rodents (Chen *et al.*, 1998). Since the expression of leptin within porcine adipose tissue is specific to the adipocyte and not other cells contaminating the tissue (Chen *et al.*, 1998b), the endocrine effects on leptin status can be assumed to be specific to the adipocyte. Glucocorticoids appear to induce the most potent stimulation of leptin mRNA expression while growth hormone is an inhibitor of its expression in swine adipose tissue *in vitro* (Chen *et al.*, 1998). Spurlock *et al.* (1998) also found that growth hormone is a potent inhibitor of leptin mRNA abundance *in vivo*. However, Portocarrero *et al.* (1998) have reported that 3 days of growth hormone treatment result in an increase in bovine leptin mRNA level. In poultry, Ashwell *et al.* (1999) have demonstrated that leptin mRNA expression is quite different from the mammal. Unlike for the pig, expression of chicken leptin is primarily in the liver. These data indicate that species specificity must be taken into consideration when assessing the potential applications of leptin to pigs.

Leptin and feeding behaviour

Leptin was originally identified as the result of genetic studies with a particular strain of obese mouse. These *ob/ob* mice became hyperphagic, consuming more than two to threefold the intake of their littermates (*ob*/+ or +/+) within weeks of birth. Genetic analysis identified this "mutation" as the result of single gene defect (Coleman, 1978). Many studies were performed over the years to characterize the obesity in these mice relative to their lean littermates. Finally, genetic mapping experiments revealed the identity of the actual mutation (Zhang *et al.*, 1994). The genetic mutation responsible for this obesity prevented the synthesis of a protein, since named "leptin". The production of recombinant leptin has resulted in a logarithmic increase in leptin research. Leptin has been reported to induce anywhere from a 5-90% reduction in feed intake depending on route of administration, dosage and ancillary treatments.

The most potent response to leptin in small laboratory animals has been observed following central administration, directly into the ventricles of the brain. In these studies administration of high doses of leptin have resulted in greater than 50% reductions in feed and water intake. Recent studies in domestic animals have also demonstrated significant

reductions in feed intake in response to central leptin administration. For example, Barb et al (1998) reported that a single injection of porcine leptin into the lateral ventricle of the pig causes a dose-dependent reduction in feed intake (Figure 3). The reduced feed intake occurred in hungry pigs that had been fasted before leptin treatment and subsequently presented with feed. The effect of a single injection of leptin was maintained through 24 hours post-injection, with recovery of intake occurring by 48 hours post injection (Figure 4). Thus, leptin is a potent inhibitor of feeding intake in swine. Also, Henry *et al.* (1999) reported that ventricular infusion of human leptin reduced feed intake in sheep by up to 60% after three days of treatment. These studies suggest that altering the CNS-leptin axis may permit changes in feed intake in domestic animals.

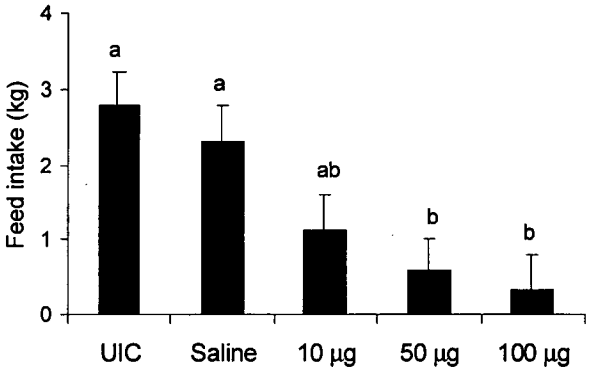

Figure 3. *Cumulative feed intake for pigs receiving intracerebroventricular injection of saline (n=3), 10 µg (n=4), 50 µg (n=4), or 100 µg (n=4) of recombinant porcine leptin. Feed intake was monitored at 20 hours after feed presentation; 24 hours after leptin or saline administration (Derived from Barb et al., 1998).* ^{a,b}*Values with different letters are significantly different (P≤0.05).*

Since central administration is not an option for humans or domestic species, current industrial research is focused primarily on producing potent synthetic analogues of leptin. However, current studies suggest that one mechanism of leptin's action is to impact on the actions of other regulators of feed intake, especially neuropeptide Y (Stephens *et al.*, 1997), corticotropin-releasing hormone (Schwartz *et al.*, 1997) and melanocyte stimulating hormone (Seeley *et al.*, 1997). Leptin can inhibit feed intake via the central nervous system in rodents following binding to leptin receptors on cells secreting these regulatory peptides. The contribution of these central regulatory peptides to leptin's regulation of swine feeding intake is unknown. However, central administration of leptin in domestic animals most likely acts by mechanisms similar to those that occur in rodents. Dyer *et al.* (1997) demonstrated that leptin receptor mRNA is present in the ovine hypothalamus in discrete neuronal nuclei associated with the regulation of feeding behaviour. Furthermore, the leptin receptor mRNA content varied with feeding state, suggesting a functional role for these receptors in the feeding response. Studies in sheep have also shown that central leptin administration reduced neuropeptide Y mRNA abundance (Henry *et al.*, 1999). Thus, central leptin administration reduces the potential expression of a hunger signal, generating a satiety response.

Since leptin is produced by the adipose tissue in the periphery, then it must cross the blood brain barrier to be effective in the CNS. Several studies have now demonstrated that leptin can bind to transporters at the blood-brain barrier and cross into the CNS effectively (Banks *et al.*, 1996; Karonen *et al.*, 1998). However, leptin may also function in peripheral tissues to regulate feed intake. Leptin receptors are present on cells of numerous tissues and organs (pancreas, intestine, etc.) involved in digestive processes (Lee *et al.*, 1996). These organs can provide an afferent feedback signal by way of the vagus nerve to the CNS. The effect of regulatory effect of leptin on feed intake has led to

the following hypothesis that leptin is a co-ordinator of overall energy balance in the normal animal. It is a signal synthesized in the major peripheral energy storage tissue (adipose tissue) in increasing quantity as the energy stores also increase in mass. This signal is secreted by the adipose tissue and acts at the peripheral tissues and in the CNS to produce a reduction in feed/energy intake. The reduced energy intake results in a decrease in energy balance and a subsequent decrease in energy stores as demonstrated by a drop in body weight, primarily a reduction in adipose tissue.

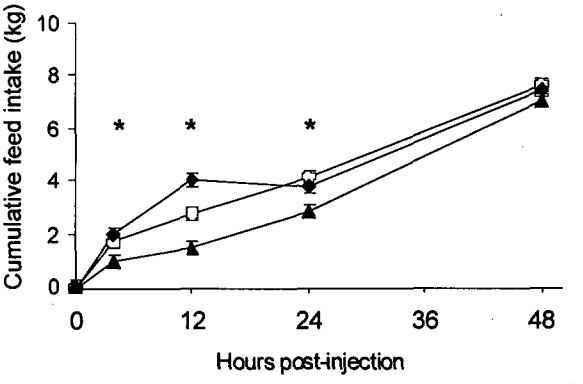

Figure 4. *Cumulative feed intake for pigs receiving intracerebroventricular injection of saline (n=4, ◆), a control peptide (n=4, pST, 50 μg, □) or 50 μg (n=4, ▲) of recombinant porcine leptin. Feed intake was monitored at 4, 12, 24 and 48 hours after ICV administration. *Leptin treatment significantly different from saline treatment (P≤0.05).*

In cases of shifts in energy balance, other factors may come into play to regulate leptin secretion. For example, fasting for 48 h results in a dramatic decrease in serum leptin levels in mice, despite the adipose tissue mass being relatively unchanged over such a short time frame. In contrast, an induced positive energy balance results in a drastic increase in serum leptin, proportionally far more than any change in body fat that could occur over such a short time. Thus, it appears that leptin secretion by adipose tissue is responsive to changes in energy balance before changes in fat mass can occur. The consequent counter-regulatory actions of leptin on energy intake suggest a close relationship between leptin secretion and energy metabolism.

Under most practical conditions, pig performance is limited by feed intake. Therefore, the interest of the swine industry is not to decrease feed intake but rather to enhance it. This will require the development of methods to inhibit the activity of leptin. One potential mechanism is to autoimmunize pigs against their own leptin, thus inactivating the leptin. However, significant methodological development is necessary to 1) inactivate leptin and increase feed intake; 2) to ensure that elevated caloric intake is utilized for protein synthesis and not fat accretion. Initial experiments have not met with great success in swine (Evock-Clover *et al.*, 1998; Wuethrich *et al.*, 1998).

Leptin and metabolism

The interaction between leptin and energy balance indicates that leptin affects energy metabolism. In fact, studies in the *ob/ob* mouse that cannot produce leptin have demonstrated that exogenous leptin administration increased oxygen consumption and body temperature, and normalized serum glucose and insulin levels (Campfield *et al.*, 1995; Halaas *et al.*, 1995; Pelleymounter *et al.*, 1995). Thus, leptin has the potential to influence many aspects of metabolism.

Perhaps the best example of the role of leptin in metabolic regulation is the response to starvation. Counter-regulatory mechanisms to oppose the large negative energy balance occur rapidly. Serum leptin concentrations and tissue mRNA abundance decrease rapidly following removal of feed (Boden *et al.*, 1996; Frederich *et al.*, 1995;

Kolaczynski *et al.*, 1996; Spurlock *et al.*, 1998). Starvation also causes an increase in serum glucocorticoids with a resultant shift from carbohydrate oxidation to fatty acid oxidation and gluconeogenesis (Ahima *et al.*, 1996), a shift in reproductive axis to an infertile state (Ahima *et al.*, 1996), and a decline in serum T_3 levels and resultant lowering of metabolic rate (Spencer *et al.*, 1983). Leptin supplementation also results in normalization or a blunting of the neuroendocrine changes associated with starvation (Ahima *et al.*, 1996). This includes activation of the hypothalamic-pituitary-adrenal axis, stimulation of the reproductive axis and the thyroid axis. The changes in neuroendocrine homeostasis suggest that the fall in serum/CNS leptin concentrations is at least a contributor to the signal for these responses to starvation and that the adaptive neuroendocrine responses are an attempt to correct the negative energy balance.

Leptin also has effects on metabolism through direct action on peripheral tissues. *In vitro* experiments have demonstrated that many of the effects of leptin do not require input from the CNS, but are the direct consequence of leptin binding to its specific receptors on these peripheral tissues. For example, leptin can bind to receptors on the pancreas and inhibit insulin secretion (Ookuma *et al.*, 1998). Insulin promotes fat synthesis and storage in the adipose tissue, the major site of leptin production. It appears that since insulin promotes energy storage, leptin is secreted to counter this process to reduce the animal's energy balance and perhaps fat deposition.

Leptin can also inhibit the secretion of glucocorticoids from the adrenal gland (Bornstein *et al.*, 1997). Since glucocorticoids promote fat/energy storage, then leptin is functioning as a feedback mechanism to regulate glucocorticoid secretion. In addition, glucocorticoids promote muscle protein breakdown. Leptin has been shown to inhibit the muscle protein breakdown induced by glucocorticoids in vitro (Ramsay *et al.*, 1998). Thus leptin inhibits the catabolic actions of glucocorticoids by inhibiting their secretion and their action on skeletal muscle.

Leptin also has actions on skeletal muscle that are independent of glucocorticoid status. For example, leptin increased fatty acid oxidation and reduced triglyceride synthesis in skeletal muscle *in vitro* (Muoio *et al.*, 1997). Furthermore, leptin stimulated glycogen synthesis in skeletal muscle *in vitro* (Berti *et al.*, 1997; Ceddia *et al.*, 1998) and in vivo (Harris, 1998.). However, effects of leptin on skeletal muscle glucose transport are equivocal. The debate may be due to the variability in leptin response between acute [direct action] and chronic treatment [indirect action] (Berti *et al.* 1997, Ceddia *et al.* 1998; Harris, 1998; Ranganathan *et al.*, 1998; Zierath *et al.*, 1998).

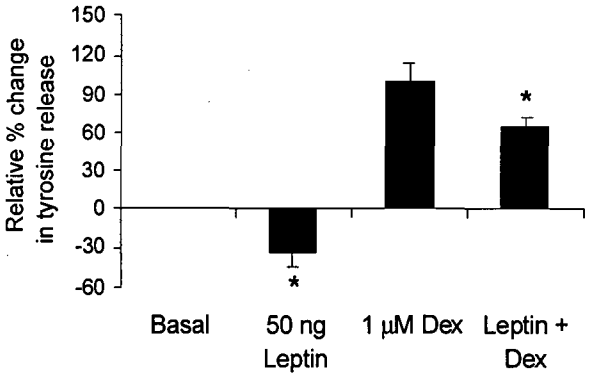

Figure 5. *Proteolysis in C2C12 myotube cultures. Cultures containing myotubes were used for protein breakdown experiments 5 days after fusion began. Protein degradation was measured by quantifying release of ^{3}H-tyrosine into culture medium under basal conditions and in the presence of leptin (50 ng/ml) or dexamethasone (Dex, 1 µM) or both following a pulse labelling with the radiochemical according to the procedures of Desler et al. (1996). *Significant leptin effect ($P \leq 0.05$).*

Exogenous leptin administration results in dramatic decreases in feed intake and live weight. Muscle appears to be conserved to some extent so that the vast majority of weight loss is adipose tissue (Pelleymounter *et al.*, 1995). The buffering of muscle from catabolism in response to a leptin-induced negative energy balance has recently been assessed (Ramsay, 1998). In that study *in vitro* treatment of C2C12 myotube cultures with leptin reduced the basal rate of protein degradation by 36% (Figure 5). While addition of dexamethasone (1 μM) to the media increased protein degradation by 100%, this was to a large extent reversed by the addition of leptin (50 ng/ml) to the media. Thus, both basal and stimulated protein breakdown can be reduced through leptin acting directly on muscle cells. The anabolic actions of leptin on muscle (reducing protein breakdown, promoting glycogen synthesis) may account for the disproportionate weight loss (adipose > muscle) during exogenous leptin treatment.

In contrast to effects in muscle, leptin's feedback effects on fat cells are more difficult to interpret. The literature is evenly divided between studies that show no effect of leptin on adipose tissue carbohydrate and lipid metabolism, and those studies that show leptin inhibits lipogenesis from glucose while enhancing glucose oxidation (Ceddia *et al.*, 1998; Harris, 1998; Mick *et al.*, 1998; Müller *et al.*, 1997; Zierath, J.R., 1998). Much of this research is confounded by an apparent interaction of leptin with insulin binding or action (Ceddia *et al.*, 1998; Müller *et al.*, 1997; Walder et al, 1997). *In vivo* experiments suggest that chronic treatment with leptin inhibits the responsiveness of fat cells to insulin (Harris, 1998) suggesting that glucose is being partitioned away from adipose tissue metabolism for use by other tissues. Furthermore, chronic leptin treatment increased adipose tissue lipolysis (Fruhbeck *et al.*, 1998; Marti *et al.*, 1998) which would provide skeletal muscle with an alternative source of energy at the expense of fat tissue.

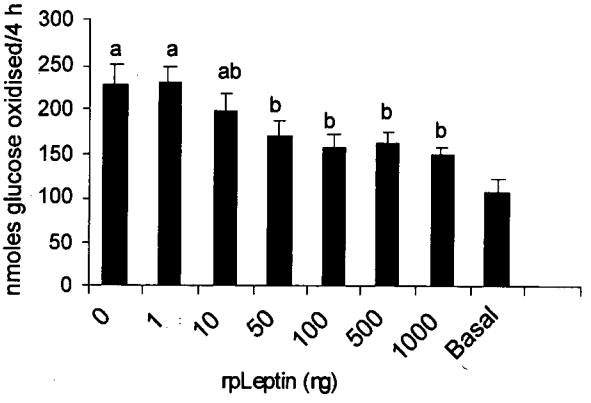

Figure 6. *Effect of leptin on insulin-stimulated and basal (no insulin or leptin) glucose oxidation in primary adipose tissue cultures. Primary cultures derived from porcine adipose tissue were differentiated in vitro according to procedures previously described (Ramsay et al., 1989). The resulting adipocyte containing cultures were changed to media containing 10 μM insulin and the newly formed adipocytes were permitted to fill with lipid for 7 days. Cultures containing lipid filled adipocytes were then washed three times with DMEM and switched to treatment media containing various concentrations of recombinant porcine leptin for 5 days before initiation of metabolic experiments. Incubation medium contained 10 nM insulin, 1 μCi U-[^{14}C]-glucose/ml, 25 mM HEPES, 10 mM glucose and 2% albumin. Three experiments were performed with triplicate flasks. Data were expressed as nanomoles glucose oxidized/flask/4 hr. Columns not sharing a common superscript are significantly different, P≤0.05.*

Studies using pig adipose tissue explants support the rodent data by demonstrating several actions of leptin on *in vitro* adipocyte metabolism (Ramsay, 1998). Acute leptin exposure *in vitro* caused no change in glucose oxidation or lipolysis in primary cultures containing *in vitro* derived porcine adipocytes. However, chronic leptin exposure in parallel cultures resulted in a decrease in glucose oxidation (Figure 6) and reduced inhibitory effects of insulin on lipolysis (Figure 7). These data demonstrate that leptin promotes the partitioning of energy away from lipid accretion within porcine adipose tissue by inhibiting glucose utilization and enhancing lipolysis, as previously demonstrated in rodents (Fruhbeck *et al.*, 1998; Harris, 1998; Marti *et al.*, 1998). Secondly, these data suggest porcine leptin functions through indirect mechanisms to alter porcine adipocyte metabolism, as chronic stimulation was necessary to identify an effect of porcine leptin.

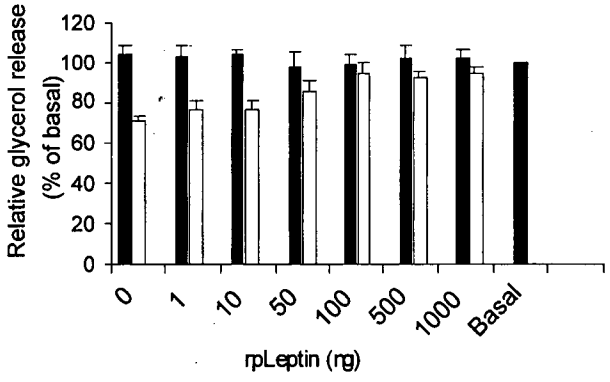

Figure 7. *Effect of leptin on isoproterenol-stimulated and basal (no isoproterenol or leptin) glucose oxidation in primary adipose tissue cultures in the presence* (□) *or absence* (■) *of insulin. Primary cultures derived from porcine adipose tissue were differentiated in vitro according to procedures previously described (Ramsay et al., 1989). The resulting adipocyte containing cultures were changed to medium containing 10 μM insulin and the newly formed adipocytes were permitted to fill with lipid for 7 days. Cultures containing lipid filled adipocytes were then washed three times with DMEM and switched to media containing various concentrations of recombinant porcine leptin for 5 days before initiation of lipolysis experiments. Lipolysis experiments were performed in the presence of 1 μM isoproterenol ± 10 nM insulin. Glycerol release was quantified 4 hours after exposure to lipolytic stimuli. Three experiments were performed with triplicate flasks. Data were expressed as relative glycerol release to correct for culture to culture variability. *Significantly different from 10 nM insulin + 0 ng leptin, (P≤0.05).*

Leptin and reproduction

The actions of leptin extend beyond metabolic homeostasis and a key role in the reproductive cycle is indicated. For example, while animals deficient in leptin (*ob/ob* mouse) are infertile, treatment of either male (Barash *et al.*, 1996; Morrison *et al.*, 1998) or female (Barash *et al.*, 1996; Chehab *et al.*, 1996) *ob/ob* mice dramatically improves fertility. In normal mice, exogenous leptin administration results in an early onset of puberty, including the onset of oestrus and cycling (Ahima *et al.*, 1997; Chehab *et al.*, 1997; Cheung *et al.*, 1997). While leptin injection has not been used in humans to manipulate reproductive function, serum leptin analysis demonstrates that leptin increases before the appearance of reproductive hormones related to puberty in females (Garcia-Mayor *et al.*, 1997). In addition, leptin concentrations are higher in the luteal than the follicular phase. These data all implicate leptin as being a permissive signal, although perhaps not a primary signal, to permit pubertal maturation to occur with attainment of energy stores

adequate for reproduction (Foster and Nagatani, 1999). This signal is so potent that leptin administration to severely food-restricted rats can induce sexual maturation despite great weight loss and malnutrition (Gruaz *et al.*, 1998), a condition that typically causes infertility.

Some of the effects of leptin on reproduction may be through alteration of gonadotrophin releasing hormone secretion (Magni *et al.*, 1999) and subsequent luteinizing hormone and follicle stimulating hormone secretion (Nagatani *et al.*, 1998; Yu *et al.*, 1997), although this has not been confirmed in swine. Thus, leptin can alter gonadal steroid secretion by an indirect mechanism mediated via the hypothalamic/pituitary axis (Yu *et al.*, 1997). Leptin may also have direct effects on the ovary. Several studies have now shown that leptin can also have direct effects on ovarian function. Following binding to leptin receptors on granulosa cells, insulin-induced estradiol and to a lesser extent progesterone production are reduced (Spicer and Francisco, 1997; Zachow and Magoffin, 1997). Leptin may also inhibit dexamethasone-induced steroidogenesis (Barkan *et al.*, 1999). These effects are in direct contrast to the systemic actions of leptin on reproductive physiology *in vivo*. This divergence may be due in part to the relatively well-controlled environment of the cell culture system versus the multiple hormonal milieu of the animal.

Some type of feedback loop may exist between the ovary and adipose tissue. Premenopausal women have higher serum leptin concentrations than postmenopausal women who in turn have higher serum leptin concentrations than men (Rosenbaum *et al.*, 1996; Saad *et al.*, 1997).. These data suggest that sex steroids can affect leptin production or secretion. Dihydrotestosterone reduced leptin expression in adipose tissue of male rats but did not affect leptin secretion by adipocytes (Machinal *et al.*, 1999). In female rats, 17β-estradiol induced an up to 1.75 fold increase in leptin mRNA abundance and a 2-fold increase in leptin protein secretion. Shimizu *et al.* (1997) found that ovariectomy reduced leptin expression in the adipose tissue of female rats, while estradiol replacement therapy reversed these effects. Both the inhibition and stimulation of leptin expression by androgens and estrogens, respectively, are mediated via the nuclear receptor and occur at the leptin gene transcription level (Machinal et al., 1999). These studies suggest that the elevated leptin concentrations in females may be the consequence of sex steroid induction of leptin expression. However, whether there exists a feedback loop between the testes and adipose tissue is still unclear.

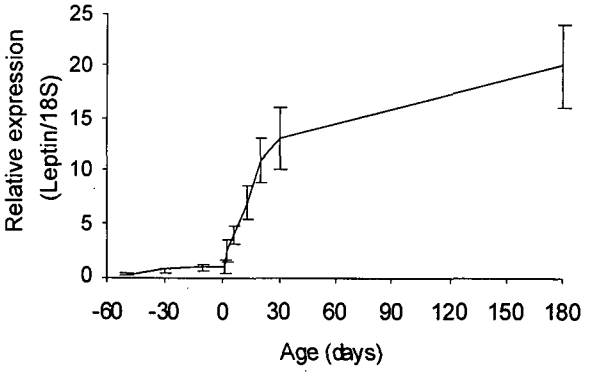

Figure 8. *Relative leptin mRNA expression (mean ±SEM) in porcine dorsal subcutaneous adipose tissue during development. Adipose tissue samples were collected at 65, 85 and 105 days of gestation and postnatally at 1, 3, 7, 14, 21, 30 and 180 of days of age. Solution hybridization and RNase protection assay were performed with 20 μg total RNA. Data were quantified by phosphorimager analysis. Leptin data are expressed relative to 18S ribosomal RNA abundance.*

In humans, serum leptin levels increase during pregnancy and decrease rapidly at parturition (Hardie *et al.*, 1997; Matsuda *et al.*, 1999). The source of the increased circulating leptin may be the placenta since the placenta can synthesize and secrete leptin (Masuzaki *et al.*, 1997) although the function of placental leptin is unclear. It has been hypothesized that placental leptin may function to assist in the partitioning of nutrients from the maternal metabolism to the fetal metabolism although this awaits confirmation (Masuzaki *et al.*, 1997). While transport of leptin across the placenta has not been demonstrated, leptin levels in fetal cord blood rise during gestation in the human fetus suggesting a role for leptin in fetal development (Matsuda *et al.*, 1997; Schubring *et al.*, 1997; Matsuda *et al.*, 1999). While neither maternal or fetal serum leptin concentrations have been reported during pregnancy in swine, there is a gradual increase in adipose tissue mRNA abundance during late pregnancy in the fetal pig (Figure 8), paralleling the accumulation of subcutaneous adipose tissue with gestational and postnatal age (Hausman, 1986).

Irrespective of the site of action of leptin within the reproductive system, a common, general hypothesis has been developed to propose that leptin functions as an afferent signal integrating information from the energy stores in adipose tissue. Subsequently, the interpretation of this signal influences reproductive function, resulting in synchronization of endocrine and behavioral components of reproductive activity during times of energy surplus to ensure survival of the potential offspring.

Leptin and immune function

A role for leptin in the regulation of immune function has been proposed to account for the well-documented immunosuppression that occurs during starvation (Chandra, 1991; Shears, 1991). Mice that are deficient (*ob/ob*) or resistant (*db/db*) to leptin have reduced T-cell function (Mandel and Mahmoud, 1978) and deficient haemopoietic activity (Bennett *et al.*, 1996), indicative of immunosuppression. Also, the structure of leptin suggests that it is a member of the family of cytokine molecules which are integral components for the proper functioning of the immune system. The homology between leptin and the cytokines led to the first studies examining the potential role of leptin in the regulation of the immune system.

Exogenous leptin administered to leptin devoid mice bound to, and promoted the proliferation of, haemopoietic cells (Gainsford *et al.*, 1996), and induced production of proinflammatory cytokines, including interleukin-6 (IL-6), IL-12 and tumour necrosis factorα (TNFα) (Loffreda *et al.*, 1998). Most importantly, Gainsford *et al.* (1996) demonstrated that exogenous leptin induced up to a 10-fold increase in macrophage phagocytic activity, an activity essential to fighting infection in the initial stages. Mikhail *et al.* (1997) subsequently demonstrated that leptin could stimulate development of macrophages and neutrophils in bone marrow from fetal or adult mice. Thus, leptin is a potent agonist for the development of cells essential for the immune response to invading microorganisms.

Inflammation induced by various methods such as lipopolysaccharide, cytokines or turpentine injection results in elevated serum leptin levels and a reduction in feed intake (Janik *et al.*, 1997; Faggioni *et al.*, 1998; Grunfeld *et al.*, 1996; Sarraf *et al.*, 1997; Zumbach *et al.*, 1997). These data suggest leptin expression during inflammation is regulated in a similar manner to the cytokine response to infection. Faggioni *et al.* (1998) demonstrated that IL-1β is one of the primary regulators of leptin expression in response to inflammation/infection. The initial conclusion from these studies was that leptin was responsible for the anorexia associated with chronic or acute inflammation. However, demonstration of an anorexic response to lipopolysaccharide in *ob/ob* and *db/db* mice that are leptin deficient or have defective leptin receptors, respectively, indicates that leptin is not essential for an anorectic response (Faggioni *et al.*, 1997). Also, injection of lipopolysaccharide did not alter leptin mRNA abundance in swine adipose tissue, suggesting that leptin does not contribute to the anorexic response to endotoxin (Spurlock *et al.*, 1998b).

Recently, Lord *et al.* (1998) have reported a different function for leptin in the resistance to infection. The *ob/ob* and *db/db* mice are also known to have impaired T cell

mediated immunity, suggesting a potential role for leptin in T-cell function. These mice have low interferon-γ and moderate IL-2 levels, both products of T cells. Leptin has now been reported to bind to its own receptor on T-cells and subsequently produce an enhanced T-cell response (Lord *et al.*, 1998). This T-cell induction appears to be selective for Th1 cells rather than Th2 cells with the consequent production of proinflammatory cytokines (Interferon-γ) and inhibition of regulatory cytokines (IL-2) by these Th1 cells. Treatment of leptin deficient, *ob/ob* mice with leptin resulted in a large increase in Interferon-γ and suppression of IL-4 production in a dose-dependent manner, demonstrating a specific role for leptin in the induction of Th1 cells and the proinflammatory response. Perhaps the most important observation by Lord *et al.* (1998) was that starvation of mice for 48 hours produces a 69% reduction in a delayed type hypersensitivity response and that leptin administration completely reversed this inhibition. These data demonstrate that leptin may an important link between nutritional status and optimal immune response to infection or inflammation.

Conclusions

While understanding the regulation of leptin expression or secretion is important to the overall physiology, the application of leptin to swine production will be dependent upon its use to alter biological processes in such a manner as to improve the production efficiency of the pig. As already mentioned, autoimmunization against leptin could be used to enhance feed intake. However, this may have deleterious effects on reproduction or immune status although the former may not be a problem in animals destined for slaughter. Alternatively, further studies are necessary to determine whether leptin treatment can potentially be used to induce an earlier onset of puberty in swine. Leptin treatment may also have the potential to substitute for nutritional flushing of gilts as a mechanism for increasing ovulation rate. Furthermore, leptin may be used to boost the immune system of the neonate swine which have extremely limited body fat and hence leptin stores Leptin could be used to promote a rapid development of T cells and macrophages in these very lean neonates.

Animal scientists are just beginning to examine the potential applications of leptin to animal agriculture. It is likely that as the biology of this hormone becomes more completely understood, additional applications will be discovered. Until then, the highest priority is to develop a better grasp of how this mediator between energy balance and physiology functions to augment growth and development of the pig.

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SERUM LEPTIN CONCENTRATION IN PIGS SELECTED FOR HIGH OR FOR LOW DAILY FOOD INTAKE

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Leptin is synthesised and secreted from adipocytes into the blood stream and transported to the brain, where it acts to cause a release of factors which can reduce food intake (Housesknecht *et al.*, 1998). There are two murine mutations of the recessive gene coding for leptin that are associated with obesity. The *Lep^{ob}* allele determines synthesis and secretion of leptin, while the *Lepr^{db}* allele determines responsiveness to leptin. Divergent selection for daily food intake (DFI) has been practiced for seven generations in a Large White pig herd (Cameron, 1994). The selection lines provide the experimental resource to determine if the correlated response in fat deposition is consistent with insensitivity to leptin or with insufficient leptin production.

In each of the high and low DFI selection lines, 20 Large White boars and gilts were penned individually and performance tested using the diet-choice procedure (Bradford and Gous, 1991) to reduce nutritional constraints on the animals' genetic merit for growth. Animals were offered two isoenergetic (14.0 MJ DE/kg) diets differing in lysine content (9.7 and 15.7 g/kg) throughout the test period from 30 ± 3 kg to 90 ± 5 kg live weight (LW). Blood samples were taken at the start and end of the test and at 50 ± 4 kg and 75 ± 5 kg LW. Serum leptin concentrations, expressed as ng/ml human equivalent (HE), were determined with a commercially available radio-immunoassay procedure (Linco Research, Missouri) using an antibody raised against human leptin which displayed 67% cross-reactivity to porcine leptin and had a detection limit of 1 ng/ml HE (Linco Research, Missouri). A commercial assay with a higher specificity was not available.

Table 1. Serum leptin and performance indices of the high and low DFI lines.

		Live weight at measurement (kg)				sed
Trait	Line	30	50	75	90	
Serum leptin (ng/ml HE)	High DFI	2.48	2.55	2.93	3.38	
	Low DFI	2.21	2.23	2.30	2.34	0.20
Backfat depth (mm)	High DFI	7.8	11.5	14.9	17.3	
	Low DFI	6.6	8.8	10.6	12.0	0.74
		Between live weights (kg)				
		30-50		50-75	75-90	
Daily food intake (g)	High DFI	1862		2362	2797	
	Low DFI	1611		1996	2394	182
Growth rate (g/day)	High DFI	867		933	902	
	Low DFI	766		840	845	49

Serum leptin concentrations in the high DFI line were higher than in the low DFI line and increased with live weight (Table 1). Backfat depth and DFI were significantly ($P<0.05$) higher in the high DFI line, from 50 kg LW. Fat content was not measured in the current study, but in the previous generation fat content of the high and low DFI lines were 249 and 190 g/kg (sed 7) at 85 kg LW. The phenotypic correlation between serum leptin and backfat depth increased from 0.33 (se 0.16) at 30 kg LW to 0.53 at 90 kg LW. In contrast, correlations between serum leptin and growth rate or serum leptin and total food intake (0.13 and 0.18 respectively, se 0.16) were not significantly different from zero. The positive response in serum leptin and backfat depth indicated that increased fat content was not due to reduced leptin production. Serum leptin could usefully be incorporated in selection criteria for genetic improvement of carcass lean content in pigs.

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PROTEIN AND LIPID DEPOSITION IN PIGS SELECTED FOR COMPONENTS OF EFFICIENT LEAN GROWTH

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Genetic improvement in carcass composition can be achieved by an increase in protein deposition or by a reduction in lipid deposition or by both routes. Fowler *et al.* (1976) proposed that selection on rate of lean growth would increase protein deposition, while lipid deposition would be reduced with selection on efficiency of lean growth. In the Edinburgh lean growth project, divergently selected lines for efficiency of lean growth (LFC), for rate of lean growth with animals performance tested on *ad libitum* (LGA) or restricted (LGS) feeding or for daily food intake (DFI) have been established with seven generations of selection in a Large White population. The LGS lines were fed a restricted ration (0.75 g/g of daily *ad libitum* food intake) in the selection phase to emphasise both rate and efficiency of lean growth. The current study measured the responses in protein and lipid deposition to test the Fowler hypothesis.

In each selection line, 10 pigs were fed *ad libitum* the standard test diet (14.0 MJ DE/kg, 10.64 g ileal digestible lysine/kg). The study also included 20 control line pigs. Two pigs from each selection line were slaughtered at 30, 45, 60, 75 or 90 kg live weight with the right hand side of the carcass and the non-carcass components homogenised and subsampled prior to chemical analysis of dry matter, protein, ash and gross energy. Lipid content was determined from: energy (MJ/kg DM) = (39.6 lipid + 23.8 protein)/100, assuming energy contents of lipid and protein of 39.6 and 23.8 kJ/g respectively, and with lipid and protein contents expressed in g/100 g DM. On average, the sum of water, protein, lipid and bone weights in the carcass and non-carcass components plus the weights of blood and gutfill proportionally accounted for 0.97 of the slaughter weight.

Rates of tissue deposition were estimated in residual maximum likelihood analyses with a model including fixed effects of selection line and sex and random effects of litter and selection group, with litters nested in selection groups. The interaction between selection line and days on test was included in the model from which tissue deposition rates were determined.

Table 1. Protein and lipid deposition and water retention rates of the selection lines (expressed as a deviation from the control line).

	Control	LFC_H	LFC_L	LGA_H	LGA_L	LGS_H	LGS_L	DFI_H	DFI_L	sed
Protein (g/day)	117	13	-18	17	-14	50	6	7	23	8
Water (g/day)	382	37	-49	73	-64	196	39	36	100	33
Lipid (g/day)	146	-18	7	13	4	38	72	69	-21	18

Subscripts denote the high (H) and low (L) selection lines

Selection for high LGA or LFC significantly ($P<0.05$) increased protein deposition and water retention. Selection on LGS increased rates of protein deposition and water retention in the high line and increased lipid deposition rate in the low line. Asymmetric responses were also detected in the DFI selection lines with higher lipid deposition in the high line and higher protein deposition and water retention in the low line.

Responses in protein and lipid deposition were not as predicted by Fowler *et al.* (1976) as responses with selection on LGA and LFC were similar. Responses in protein and lipid deposition with the LGS selection objective were consistent with the Fowler hypothesis. Selection on LGS may be preferable to selection on LGA provided a pragmatic method is available for testing animals on a restricted feeding regime.

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GENOTYPE WITH NUTRITION INTERACTION FOR PROTEIN AND LIPID DEPOSITION IN PIGS

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Testing different pig genotypes on a single diet may constrain protein and lipid deposition or reduce the efficiency of nutrient utilisation by under- or over-supply of nutrients. The current study estimated the genotype with nutrition interaction for protein and lipid deposition rates to determine if the ranking of genotypes was dependent on the diet used when performance testing animals.

The study included 320 Large White boars and gilts from lines divergently selected for rate of lean growth with animals performance tested on *ad libitum* (LGA) or restricted (LGS) feeding, efficiency of lean growth or daily food intake over seven generations. In each of the eight selection lines, 30 pigs were fed *ad libitum* one of three isoenergetic (14.0 MJ DE/kg) diets differing in lysine:energy (0.40, 0.76 and 1.12 g ileal digestible lysine/MJ DE). The study also included 80 control line pigs, which were fed the 0.40 and 0.76 ileal digestible lysine diets. Two pigs from each line-diet subclass were slaughtered at 30, 45, 60, 75 or 90 kg with chemical analysis of carcass and non-carcass components.

Rates of tissue deposition were estimated in residual maximum likelihood analyses. The model included fixed effects of selection line, diet, sex and the selection line-diet interaction and random effects of litter and selection group, with litters nested in selection groups. Days on test was fitted as a covariate for each selection line-diet subclass from which tissue deposition rates were determined.

In Fig. 1, estimated protein deposition rates for each selection line-diet subclass (X axis) are plotted against estimated protein deposition rates from a model omitting the selection line with diet interaction (Y axis). If there was no interaction, then points would lie on a straight line.

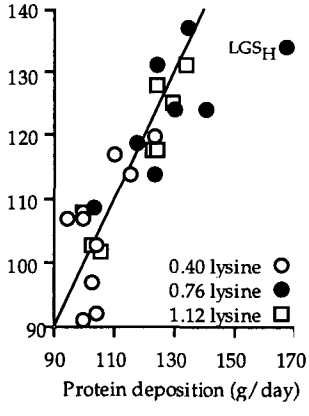

Figure 1. *Protein deposition rate.*

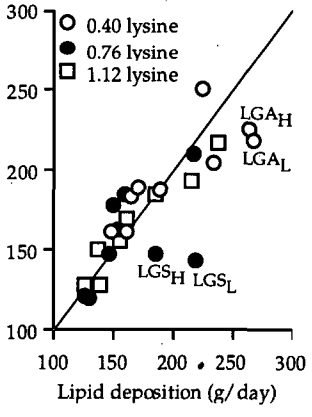

Figure 2. *Lipid deposition rate.*

A significant line with diet interaction ($P<0.05$) for protein deposition (Figure 1) was primarily due to the high (H) LGS line, which had higher protein deposition on the 0.76 lysine diet than predicted from the additive model. The interaction for lipid deposition, Figure 2, resulted from high lipid deposition in the LGA lines on the 0.40 lysine diet and the LGS lines on the 0.76 lysine diet, particularly the low (L) LGS line. When the high LGS line was excluded from the analyses, the correlation between the two sets of estimates for protein deposition of 0.85 (se 0.21) was not significantly different from unity. Similarly, the correlation between lipid deposition estimates was 0.89, excluding the LGS lines.

There was no evidence for a genotype with nutrition interaction for protein or lipid deposition when the LGS lines were excluded, such that ranking of selection lines will be similar irrespective of the diet for performance test. From the current study, the selection strategy-diet combination of high LGS and 0.76 g ileal digestible lysine/MJ DE provides an efficient and high protein deposition rate.

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SELECTION FOR EFFICIENT LEAN GROWTH UNDER RESTRICTED FEEDING: 1. GENETIC PARAMETERS

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It is expected that selection for increased growth rate on fixed feed intake over a fixed time period will exploit variation in the efficiency of food utilisation, associated with the variation in lean relative to fat deposition. This should lead to a reduction in fat, in energy used for maintenance, and an increase in lean growth rate. To test this hypothesis, two lines of 36 sows and six boars were newly established from sampling within litters of an outbred Large White population free of the halothane gene. Pigs were divergently selected for high and low growth rate over a 6 week period starting at 50 kg live weight. Over the test period, all pigs were fed the same fixed ration restricted to 80% *ad libitum* intake measured in a test on unselected foundation animals. The diet contained 14 MJ DE and 0.65 g/MJ of available lysine. Measurements were made of live weight and ultra sonic P2- backfat thickness at the end of the test.

This paper reports estimates of genetic parameters on 1,751 pigs in the two lines from the foundation to the third generation of selection. Variance and co-variance components were estimated using a restricted maximum likelihood procedure (REML). A multi-trait REML algorithm was used to allow simultaneous estimation of multiple random effects (ASREML) (Gilmour *et al.*, 1999). Fixed effects were batch and sex, and animal was the random effect. The same model was used for the analysis of average daily gain (ADG), P2 backfat thickness, and food conversion ratio (FCR). Estimates of genetic parameters are given in Table 1.

Table 1: Heritabilities (diagonal, in bold), phenotypic (in italics) and genetic correlations (in regular font) on restricted feeding.

Traits	ADG	P2-fat	FCR
ADG	**0.26** (0.04)[1]	*-0.11 (0.03)*	*-0.94 (0.00)*
P2-fat	-0.23 (0.13)	**0.33** (0.05)	*0.11 (0.03)*
FCR	-0.99 (0.00)	0.25 (0.14)	**0.27** (0.05)

[1]Standard error in parenthesis.

The magnitude of the heritabilities and the direction and magnitude of the correlations are consistent with results on restricted feeding reported by McPhee *et al.* (1988) and Nguyen *et al.* (unpublished). The phenotypic and genetic correlations between ADG and FCR were close to -1.0 and reflect a very low variation in food intake (coefficient of variation=2.5%), the numerator of the ratio. These parameter estimates are consistent with responses in growth, efficiency, and lean to selection for growth rate on restricted feeding reported so far in these lines by McPhee *et al.* (1999). Emphasis on increasing efficiency confirms that selection with food restriction exploits genetic variation in the relative rates of lean and fat tissue deposition.

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INSULIN INFUSION AND HIGH PROTEIN DIETS CAN INCREASE SOW MILK YIELD AND PIGLET GROWTH

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The protein content of sows' milk is a significant constraint to the growth of sucking pigs. McGuire *et al.* (1995) showed that simultaneous infusion of insulin and glucose to dairy cows increased milk protein content and yield by 7 and 3.5% respectively. In these experiments there was also a 60% decrease in the plasma concentrations of branched-chain amino acids (BCAA) while the other essential amino acids were decreased by an average of 25%. This may have limited the increase in protein content, and it is likely that supplements of additional BCAA may have stimulated milk protein yield even more. The aim of this study was to determine whether dietary supplementation with BCAA, and simultaneous infusion of exogenous insulin and glucose, would increase milk protein secretion in the sow.

The experiment involved 16 Large White x Landrace sows nursing litters of 11 piglets. Sows were fed either a normal lactation diet (16% CP, n=8) or a diet supplemented with additional protein and free BCAA (23% CP, valine, isoleucine and leucine, n=8). Sows were either simultaneously infused with insulin and glucose during mid (day 5-10) lactation and not infused during late (day 17-22) lactation, or were given the reverse treatment in a balanced design. Blood samples were rapidly analysed for glucose concentration and the glucose infusion rate adjusted to maintain blood glucose within 15% of pre-infusion levels. Milk production was measured by D_2O dilution in the piglets.

Table 1. Effect of protein and BCAA supplementation with and without insulin and glucose infusion on milk yield and constituents, and piglet growth. Data are the means over 5 days of treatment.

Diet (D)	16% CP		23% CP/BCAA			Significance		
Insulin & Glucose (I)	None	Infused	None	Infused	sed	D	I	D x I
Milk yield (kg/litter/d)	10.3	11.1	9.8	11.2	0.08	0.77	0.01	0.37
Milk protein (%)	5.1	4.9	5.6	5.4	0.24	0.01	0.29	0.84
Milk protein (g/d)	524	537	544	605	41.8	0.24	0.10	0.27
Milk fat (g/d)	775	788	701	734	128	0.89	0.75	0.48
Milk lactose (g/d)	645	723	612	731	120	0.80	0.01	0.42
Piglet growth (g/d)	229	245	262	285	22.6	0.05	0.11	0.73

As there was no effect of stage of lactation, results of these treatments have been pooled. Milk and lactose yield increased with insulin infusion, while milk protein content was increased in diets supplemented with protein and BCAA (Table 1). Milk protein yield was rather variable and not significantly different between treatments, but tended to increase (P=0.06) when the treatments were combined compared to untreated sows (605 g/day vs 524 g/day). Piglet growth was increased by feeding the higher protein diet (273 vs 237 g/d, P=0.05) and tended to increase with insulin infusion (245 vs 265 g/d, P=0.11). These effects tended to be additive such that the combined treatment resulted in a 24% (56 g/d, P<0.05) increase in piglet growth rate.

These data demonstrate that increasing the protein/BCAA content of the diet increases milk protein content but not milk yield. Infusion of insulin and glucose increases milk and milk lactose yields but not milk protein secretion. These effects are additive and translate to increased piglet growth.

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ENDOGENOUS PLASMA LEPTIN INCREASES WITH AGE AND IS RELATED TO FATNESS AND APPETITE

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Leptin is a hormone produced by adipocytes in pigs (Barb *et al.*, 1998). When injected into pigs it reduces appetite and increases growth hormone secretion (Ramsay *et al.*, 1998). Relationships between blood concentrations of endogenous leptin, adiposity and appetite were examined to determine whether leptin is a marker of these traits.

Blood was collected on five occasions from 73 well-fed Large White boars. Plasma was recovered by centrifugation at 4°C and stored at -20°C. Animals were group housed until they were individually penned at 16 weeks of age. Voluntary feed intake and live weight were measured weekly thereafter. Leptin was measured in plasma by radioimmunoassay using antiserum to human leptin that has broad species specificity (XL-85K, Linco Research Inc., St. Charles) and human leptin (R&D Systems, Minneapolis) as standard and radioiodinated tracer. Porcine leptin has 67% crossreactivity in this assay and the dose-responses to pig plasma and human leptin are parallel. Associations were assessed by linear regression. Effect of age was assessed by one-way analysis of variance.

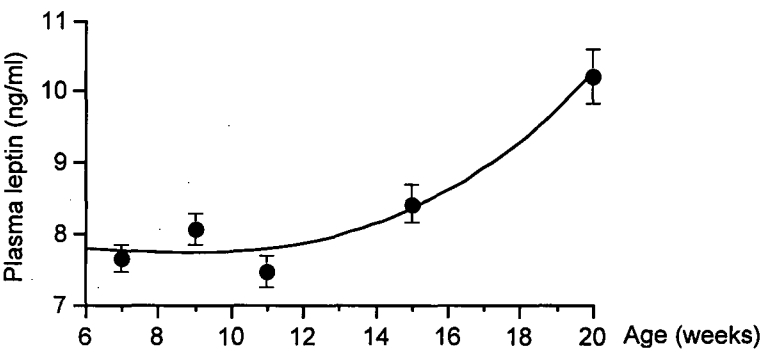

Figure 1. *Plasma leptin in boars (mean ± SEM).*

Leptin (Fig. 1) increased with age (ANOVA, $P<0.02$) which is in agreement with a recent report by Barb *et al.* (1998). Higher values of leptin observed in the present study are probably due to the use of human leptin as assay standard. Plasma leptin concentration was not related to live weight (*all* $r^2<0.03$, $P>0.3$) but tended to "track", i.e., boars with low leptin at any age also tended to have low leptin at other ages and *vice versa* (*all* $r^2>0.1$, $P<0.05$). Plasma leptin at 7 weeks was positively correlated with gain in P2 back fat between 20 and 22 weeks of age ($r^2=0.14$, $P<0.005$). At 15 weeks, leptin was positively correlated with backfat P2 at 20 ($r^2=0.12$, $P<0.025$) and 22 weeks ($r^2=0.08$, $P=0.07$). At 20 weeks, leptin was positively correlated with voluntary feed intake in the 21st ($r^2=0.10$, $P<0.01$) and 22nd weeks ($r^2=0.09$, $P<0.02$), average weekly voluntary feed intake from 16 to 22 weeks ($r^2=0.08$, $P<0.025$) and with P2 backfat depth of carcass at 24 weeks ($r^2=0.10$, $P=0.015$).

Fatter pigs have greater appetites despite their higher plasma leptin concentrations, indicating that obesity increases leptin resistance in pigs as has been suggested in humans.

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PIG RESEARCH AND DEVELOPMENT CORPORATION

INFLUENCE OF GENOTYPE AND SEX ON PORK EATING QUALITY: A CONSUMER TASTE PANEL ASSESSMENT

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The Australian pig industry has moved away from castration of boars to harness production benefits associated with entire male pigs. Recent improvements in pork production have resulted in leaner and faster growing breeds with lower production costs. However, with the use of entire males, such gains in production efficiency have been to the detriment of the eating quality of pork. Use of entire males, coupled with increased pig slaughter weights and increased leanness, have resulted in an increased incidence of boar taint, and tough and dry pork due to the lower intramuscular fat levels (Wood, 1993). The aim of this experiment was to compare the eating quality of pork from entire males with that of surgical and immunological castrates from two fast growing 'modern' genotypes.

Sixty crossbred male pigs were allocated to a 2 x 3 factorial design experiment (Mullan *et al.*, 1999) with the main treatments being Genotype (A – fast growing, 50% Duroc bloodlines, lean; and B – fast growing, <25% Duroc bloodlines with a propensity for increased fat deposition) and Sex (entire, surgically castrated and immunologically castrated male). Four pigs per treatment were randomly selected and 5 steaks per loin were used in a consumer taste panel. The pork samples were cooked using a Silex flat-plate grill and were cooked for 5 minutes at 190°C (internal temperature 75°C; medium/well done). Each steak was halved after cooking and tasted by two consumers. Each experimental treatment was tasted by 10 consumers. A total of 48 consumers (balanced for age and gender) assessed the loin steaks for eating quality attributes.

Table 1. The effect of genotype (A vs B) and sex (entire male - EM, surgical castrate male - SCM and immunological castrate male - ICM) on eating quality of pork steaks.

Genotype (G)	A			B				P values		
Sex (S)	EM	SCM	ICM	EM	SCM	ICM	lsd	G	S	G x S
Boar Odour	56.6	69.2	59.9	56.0	55.3	64.4	8.67	1.89	0.093	0.011
Flavour	60.5	70.9	63.1	56.3	52.5	69.0	9.91	0.058	0.101	0.003
Tenderness	55.1	77.5	62.2	48.4	40.8	62.7	10.52	<0.001	0.016	<0.001
Juiciness	61.7	71.8	61.3	57.4	45.8	66.6	9.97	0.005	0.304	<0.001
Overall Acceptability	60.5	76.0	64.6	55.1	48.9	68.8	9.06	<0.001	0.025	<0.001

Acceptability score (line scale) for all attributes, 0 = dislike extremely and 100 = like extremely.

Pork from Genotype A pigs was more tender, juicy and had better overall acceptability compared to pork from the Genotype B pigs. Pork from entire males was the toughest and least acceptable compared to pork from the surgical and immunological castrates. Pork from Genotype A surgical castrates had lower boar odour, and higher flavour, tenderness, juiciness and overall acceptability compared with pork from entire and immunologically castrated males. Within Genotype B, the immunological castrates had less boar odour, more flavour, better tenderness, juiciness and overall acceptability compared to the Genotype B entire and surgical castrate males. The large variability in eating quality between genotypes for surgical castrate males may be a direct reflection of the intramuscular fat levels present in the two genotypes. Hence, these data indicate that good pork eating quality is not just reliant on surgical or immunological castration, but more a complex interaction between genotype and entire or castrated male pigs.

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EFFECT OF BINDER AND BRINE FORMULATION ON SENSORY ATTRIBUTES OF PORK SHOULDER ROASTS

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In the food service sector, the use of attractive value-added products from the pork shoulder would allow chefs to provide pork meals that were competitive in price and able to meet consumer expectations of tenderness and juiciness. The objective of this study was to determine the most appropriate binder and ingredients for addition to brine to maximise tenderness and juiciness of a boneless pork shoulder product.

Twenty-four shoulders from 12 Large White x Landrace entire male pigs of 90 kg live weight were randomly allocated to eight treatments. This experiment was a 2 x 4 factorial which consisted of 2 binding treatments: (i) Pearl E (Earlee Products, Brisbane) applied to exposed meat surfaces prior to netting and injection of brine, (ii) modified starch/xanthan gum product injected into muscles with 5% added water after netting and prior to injection of brine; and 4 brine treatments: (i) 10% water with sodium tripolyphosphate (STPP) added to 0.4% of final product weight, (ii) 10% 0.3 M $CaCl_2$, (iii) 10% water, (iv) control - no added water. Brine was injected into each product at multiple sites using a 10 cm 12 gauge needle attached to a 50 ml syringe. All roasts were individually vacuum packaged and each roast was weighed prior to chilling at 2-4°C for 7 days. After chilled storage, all roasts were removed from vacuum packaging, dried and re-weighed to determine purge loss. Pork was cooked in a fan forced oven set at 180°C until an internal temperature of 70°C was attained. Sensory attributes were assessed by a consumer taste panel (n=80) using an incomplete block design.

Table 1. Effect of brine treatment on purge and tenderness, juiciness, flavour and overall acceptability of pork shoulder roasts evaluated by a consumer taste panel.

	Brine treatment					
	0.4% STPP + 10% water	10% 0.3 M $CaCl_2$	10% water	Control	sed	P
Purge loss(%)	5.22	5.65	6.39	1.77	0.475	P<0.001
Tenderness[1]	6.57	6.16	5.46	5.47	0.390	P<0.1
Juiciness[1]	6.24	5.05	4.94	5.20	0.231	P<0.01
Flavour[1]	6.38	5.34	5.34	5.64	0.258	P<0.01
Acceptability[1]	6.27	5.03	5.47	5.42	0.167	P<0.01

[1]Scale : 0 - dislike extremely to 10 - like extremely.

The modified starch/xanthan gum binder was inappropriate for use as a binder of pork as the product fell apart when sliced after cooking. In contrast, roasts bound with Pearl E did not fall apart upon slicing which allowed panellists to be presented with complete slices of product for evaluation. Pork shoulder roasts pumped with 10% water had higher purge loss compared with product pumped with 0.4% STPP + 10% water. Tenderness of pork shoulder roasts tended to be improved by the addition of either 0.4% STPP + 10% water or 10% 0.3 M $CaCl_2$ to pork shoulders compared to control roasts and roasts pumped with 10% water (Table 1). Juiciness, flavour and overall acceptability of pork roasts pumped with 0.4% STPP + 10% water was judged to be higher than pork roasts in the other treatments. In conclusion, STPP was identified to be the most suitable additive to minimise purge and enhance flavour, juiciness and overall acceptability of pork shoulder roasts compared with 0.3M $CaCl_2$.

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MAGNESIUM SUPPLEMENTATION TO REDUCE THE INCIDENCE OF SOFT EXUDATIVE PORK UNDER COMMERCIAL CONDITIONS

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Dietary magnesium (Mg) supplementation can improve pork quality by reducing drip loss, improving pork colour and reducing the incidence of pale, soft, exudative (PSE) carcasses (D'Souza *et al.*, 1998, 1999). However, before dietary Mg supplementation can be recommended to the pork industry as a viable method to reduce PSE, the beneficial effects of Mg supplementation needs to be validated under commercial conditions. Therefore, the aim of this study was to determine if Mg supplementation reduced the incidence of PSE pork under commercial conditions.

The study was conducted between July and October 1998 and consisted of 2 dietary treatments: (1) Control diet - standard finisher diet, (2) Mg diet - finisher diet supplemented with 16g Mg proteinate (Chelated Minerals Corporation, Salt Lake City, Utah) /pig/day for 2 days prior to slaughter. The study consisted of 3 replicates, with each replicate consisting of 150 Large White x Landrace finisher pigs per treatment. After the 2 day supplementation period, all pigs were transported to the abattoir where, after a two to three hour rest in lairage with access to water, they were slaughtered and the carcasses chilled according to the normal practice in the abattoir. Colour and pH of each carcass was measured in the loin at the P2 site six hours post-slaughter. Pork was classified as soft exudative (SE) if pH<5.6, dark firm and dry (DFD) if pH>6.0 and normal if 5.6<pH<6.0 (Hofmeyr *et al.*, 1997).

Table 1. Incidences (%) of pork quality categories after Mg supplementation.

	Replicate 1		Replicate 2		Replicate 3		P^1
	Control	Mg	Control	Mg	Control	Mg	Value
%SE	67	5	22	9	22	13	< 0.05
%Normal	29	54	43	55	38	37	< 0.05
%DFD	4	41	35	36	40	50	< 0.05

[1]Chi-squared goodness-of-fit test used

The changes in pH were highly significant and are reflected in the incidences of soft exudative, normal and dark firm and dry pork presented in Table 1. There were no significant differences in pork colour between diet treatments. The unusually high SE levels in replicate 1 could be a reflection of a high incidence of Halothane gene carriers in this particular slaughter batch. However, in all instances, dietary Mg supplementation significantly reduced the incidence of SE pork. There was an increase in pH with dietary Mg, which resulted in an increase in the incidence of DFD pork. This was most pronounced in the first replicate where there was an extremely low incidence in the control group. These data show that dietary Mg supplementation can reduce the incidence of SE pork and hence may have important financial and quality benefits for both producers and processors.

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A NEAR-INFRARED SPECTROSCOPIC TECHNIQUE FOR SPECIES DIFFERENTIATION OF FROZEN MEAT

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Meat from different species is not easy to distinguish visually when deboned and frozen in large blocks. There have been reports of substitution of expensive beef with cheaper kangaroo or horsemeat in commercial practice (Barai *et al.*, 1992). There is therefore a need for an instrumental or analytical technique for species differentiation of frozen meat in commercial trade. This report describes a near infrared (NIR) spectroscopic technique for differentiation of frozen meat of various species.

Samples (~200 g) of pork (30), beef (19), mutton (32), rabbit meat (32) and kangaroo meat (22) were purchased from butcher shops. To ensure diversity, no more than two samples were purchased from one shop on the same day and no samples were purchased from the same shop on consecutive days. No attempt was made to identify the anatomical origin of the samples. Following collection, samples were frozen at -30°C before being thawed at room temperature and minced with an electric kitchen mincer immediately prior to analysis. Minced meat samples were placed in a polyethylene bag within a rectangular cell, which allowed an effective scanning area of 82 cm^2 when used with a sample transport module. Scanning was performed using NIRSystem 6500 monochromator (Perstorp Analytical Inc.) over a scanning range of 400 to 2500 nm at 2 nm intervals. Spectral data were analysed using principal component analysis and canonical discriminant analysis (Gittins, 1985) with the computer software, SAS (SAS Institute Inc.). Linear discriminant function was derived from canonical discriminant analysis based on a normal distribution of each class and pooled covariance matrices. Discrimination accuracy was calculated from cross-validation.

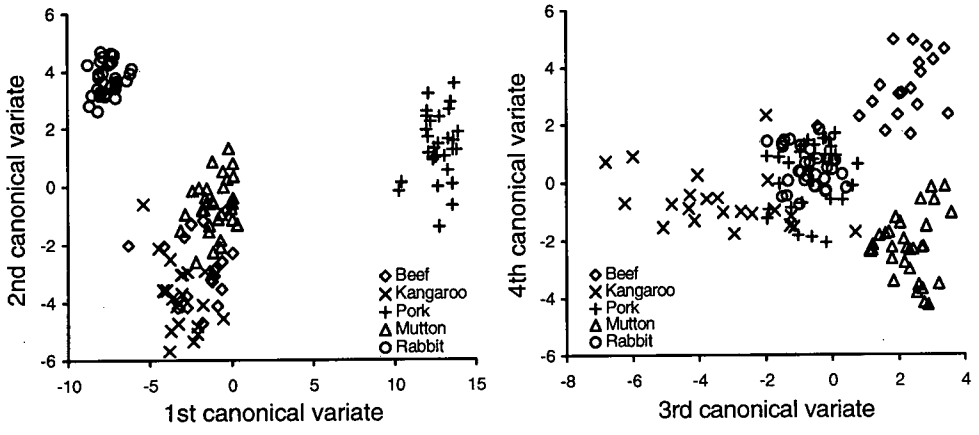

Figure 1. *Plots of canonical variates.*

The first 4 canonical variates, which summarized 100% of the inter-species variation, were derived from the first 20 principal components. Plots of canonical variates (Figure 1) visualized a clear separation of meat samples from different species. Discriminant function developed from the first 4 canonical variates separated meat samples according to the species origin with an accuracy of 99.3%. These results indicate that the NIR spectroscopic technique may be used for species differentiation of frozen meat.

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VITAMIN E STATUS IN NEWLY WEANED PIGLETS IS CORRELATED TO THE ACTIVITY OF CARBOXYLESTER HYDROLASE IN PANCREATIC TISSUE

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Vitamin E in blood plasma decreases to critically low concentrations in newly-weaned pigs even though the weaner diet contains 5-10 times more vitamin E than sows' milk. Carboxylester hydrolase (CEH) is a relatively nonspecific lipolytic enzyme, which cleaves ester linkages and is responsible for hydrolyzing ester forms of vitamin A and E. It has been shown that the activity of a number of enzymes in pancreatic tissue, including CEH, declines after weaning (Jensen *et al.*, 1997). The present experiment was performed to investigate the correlation between the activity of CEH in the pancreatic tissue and the vitamin E status of piglets during the period immediately after weaning.

An experiment was performed with 63 pigs (Danish Landrace x Yorkshire). The pigs were weaned at 28 days of age without having access to creep feed prior to weaning. The pigs were divided into seven groups and, from each group, a pig was slaughtered at 25, 28, 29, 30, 31, 33, 35 and 37 days of age. On day 31 an extra pig from each group was slaughtered for other purposes. The activity of CEH in the pancreatic tissue was measured and vitamin E status of the piglets was determined in blood samples by methods described by Jensen *et al.* (1999).

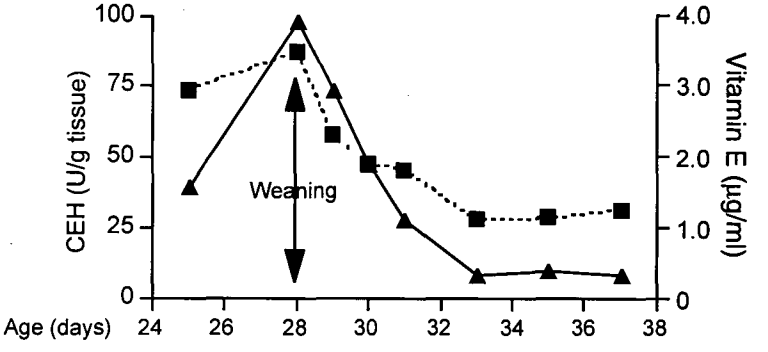

Figure 1. *The activity of carboxylester hydrolase (CEH) (▲) in pancreatic tissue and the concentration of vitamin E (■) in blood plasma of pigs. Each point represents the average of 7 piglets (14 piglets on day 31), SE_{CEH} = 50 U/g tissue, $SE_{Vit\ E}$ = 1.1 µg/ml.*

There was close agreement in the pattern of change in CEH activity and plasma vitamin E concentration, with maximum concentrations observed at weaning and minimum concentrations reached within 5 d post-weaning (Figure 1). Sows' milk primarily contains natural, unesterified vitamin E (RRR-α-tocopherol) which is readily absorbed and has a significantly higher biological activity than synthetic vitamin E (all-rac-α-tocopherol). In weaner diets vitamin E is supplied as all-rac-α-tocopherol acetate. The synthetic (acetate) form has to be hydrolysed by CEH prior to absorption. In this experiment CEH activity in pancreatic tissue and vitamin E concentration in plasma were highly correlated after weaning (r^2=0.64, P<0.001). It is therefore suggested that the low plasma vitamin E concentration during the period immediately after weaning can be explained by a limited ability to hydrolyse the synthetic vitamin E in the weaner diet.

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THE EFFECT OF STAGGERING THE STRESSORS AT WEANING ON POST-WEANING PERFORMANCE

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At weaning the piglet is normally subjected to a number of potential stressors simultaneously. These stressors include separation from their dam, changes in diet, environment and social group, and also handling, vaccination and transport. During the period immediately following these events growth rate and feed intake declines. In this experiment some of the changes during weaning were introduced over a longer period of time to determine what effect a more gradual weaning process would have on post-weaning food intake and growth.

Thirty-four mixed sex litters in a large commercial piggery were randomly allocated to either *'conventional'* (CW) or *'lag weaning'* (LW) treatments at 20 days of age and 5.8 ± 1.0 kg (mean ± SD) body weight (BW). Conventionally weaned piglets were removed from the sow at 27 days of age, placed in trolleys and transported to the weaner shed where they were placed in new social groups and introduced to a phase 1 weaner diet (15 MJ DE/kg, 0.9 g lysine/kg). In the LW treatment, piglets were provided with creep feeders at 18 days of age and the sow was removed from the crate when the litter was 20 days of age. These piglets remained as original litter groups in the farrowing crate with access to a phase 1 weaner diet until 27 days of age when they were also removed to the weaner shed and placed in new social groups. Individual live weights were measured at 20, 27, 48 and 67 days of age.

Table 1. Effect of "lag weaning" on average daily gain (ADG) growth rates and feed intake post-weaning.

	CW	LW	Significance
20-27 day ADG (g)	250	120	**
27-48 day ADG (g)	370	440	*
27-48 day feed intake (kg/d)	0.440	0.540	***
27-67 day ADG (g)	510	560	**

*P≤0.05; **P≤0.01; ***P≤0.001.

In the farrowing house, litters remaining on the sow grew significantly faster than litters where the sow was removed. However, after movement to the weaner shed, *lag weaned* piglets grew significantly faster and with a higher daily intake for the first 21 days post-weaning to be heavier at 21 days post weaning, 15.27 ± 0.78 vs 15.94 ± 1.28 kg BW (P=0.0173) and at 40 days post-weaning, 27.94 ± 3.86 vs 28.97 ± 3.87 kg BW (P=0.0275).

The gut of the piglet undergoes prolific growth immediately post-weaning (Cranwell *et al.*, 1995). In this experiment, it is possible that staggering the stressors at weaning has allowed the piglet to maximise gut development while still in the farrowing crate and to better cope with the other stressors later on. This has resulted in a higher feed intake and hence growth rate post-weaning through to 67 days of age for piglets where the stressors associated with weaning have been staggered.

These data suggest that with appropriate management changes, the impact of weaning on development of the piglet can be minimised, even in a commercial environment.

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DIETARY CHROMIUM PROPIONATE AND METABOLIZABLE ENERGY EFFECTS ON PORK QUALITY OF FINISHER PIGS

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Limited research has been conducted that addresses the effects of chromium (Cr) on pork quality. Boleman *et al.* (1995) reported no effects of Cr picolinate on water holding capacity or sensory evaluation in pork, but shear force was increased in pork of pigs fed Cr during the grower-finisher period. O'Quinn *et al.* (1998) reported that Cr picolinate decreased drip loss and marbling, and that Cr nicotinate decreased visual color and saturation index in the longissimus dorsi (LD) of gilts. The form of organic Cr may affect the response to Cr. No research has been conducted to determine if Cr propionate (CrPr) affects pork quality, as is seen with other organic Cr sources. Thus the purpose of this experiment was to determine the effects of CrPr and dietary metabolizable energy (ME) on pork quality of finisher pigs.

Cambrough 22 barrows (n=144) were allotted to dietary treatments in a randomized complete block design (RCBD; six replicates of six pigs each; average initial and final body weights were 27 and 113 kg, respectively). The dietary treatments were: 1). corn-soybean meal basal (low ME), 2). 1 + 200 ppb Cr (as CrPr), 3). 1 + 200 kcal ME/kg (4.5% added fat; high ME), 4). 3 + 200 ppb Cr (as CrPr). At the end of the trial, three pigs per replicate were killed to determine dietary effects on pork quality. Values for pH were taken in the LD between the 10^{th} and 11^{th} ribs at 45 min and 24 h post-mortem, and LD moisture was determined on the 9^{th} rib chop. After a 20 h chill, objective (Minolta) and subjective colour scores were taken on the 10^{th} rib chop. The 10^{th} rib chop was then removed, vacuum packed, and frozen for subsequent determination of purge and cook loss and shear force as previously described by Boleman *et al.* (1995). Data were analyzed as a RCBD with a 2 x 2 factorial arrangement, and the pen of pigs was the experimental unit.

Table 1. Effect of chromium and metabolizable energy on pork quality

Cr, ppb	0	200	0	200		Effects		
Item ME	Low	Low	High	High	SEM	Cr	ME	Cr x ME
45 min pH	5.89	5.91	5.88	5.94	0.06	0.49	0.87	0.72
24 h pH	5.57	5.58	5.58	5.58	0.02	0.80	0.84	0.74
Marbling	1.65	2.06	1.58	1.75	0.11	0.03	0.12	0.31
L* value	54.75	56.18	56.96	56.47	0.63	0.47	0.07	0.15
LD moisture, %	74.19	73.84	74.24	73.89	0.15	0.04	0.75	0.99
Purge loss, %	17.82	15.69	17.32	16.46	0.64	0.04	0.84	0.34
Cook loss, %	16.58	16.89	16.54	17.28	0.62	0.42	0.79	0.74
Shear force, kg	4.27	4.11	3.92	3.97	0.12	0.67	0.06	0.39

Subjective color and firmness-wetness scores and 45 min and 24 h pH were not affected by diet. Minolta colour (L*) was increased (P=0.07) and shear force decreased (P=0.06) in pigs fed high ME diets (Table 1). Subjective marbling was increased (P=0.03) and LD percentage moisture and purge loss were decreased (P=0.04) by CrPr. The decrease in purge loss by CrPr is consistent with O'Quinn *et al.* (1998), who reported a decreased drip loss in gilts fed Cr picolinate. However, the increase in marbling is inconsistent with O'Quinn *et al.* (1998), who reported decreased marbling in gilts and no response in barrows fed Cr picolinate. The improvements in purge loss and marbling indicate that dietary CrPr may benefit pork quality, and that it is possibly a biologically active organic source of Cr.

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DIETARY MAGNESIUM SUPPLEMENTATION IMPROVES PORK QUALITY

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Subjecting pigs to an acute stressor just prior to slaughter can lead to pale, soft, exudative (PSE) pork by increasing the rate of muscle acidification at slaughter and immediately post-slaughter. Dietary magnesium (Mg) supplementation in pigs has been shown to reduce the effects of stress by reducing plasma catecholamines at slaughter, reducing muscle glycogenolysis post-slaughter and reducing the incidence of PSE pork (D'Souza *et al.*, 1998). However, before dietary Mg supplementation can be recommended as a viable method to improve pork quality, the most appropriate Mg compound, Mg dose and duration of supplementation needs to be determined.

Ninety crossbred (Large White x Landrace) female pigs (live weight 70 kg ± 15 kg; mean ± SD) were used in a 2 x 2 x 2 + 1 factorial design experiment. The treatments were: (A) Diet, control diet, diet supplemented with magnesium aspartate (MgAsp) and diet supplemented with magnesium sulphate ($MgSO_4$); (B) Dose, 1.6 g and 3.2 g of elemental Mg per pig per day; and (C) Duration, 2 days and 5 days prior to slaughter. At the abattoir, a negative handling treatment comprising of 15 electric shocks from an electric goad was imposed on each pig 5 minutes prior to slaughter. Drip loss (suspended sample method) and lightness (L*) were determined on samples removed at 24 h post-slaughter. Pork was classified as PSE if drip loss ≥5% and L*>50.

Table 1. The effect of different dietary magnesium supplements, supplementation dose and duration on pork quality indicators in the *Longissimus thoracis* (LT) muscle at 24 h post-slaughter.

	Diet (D)			Dose (DO)		Duration (DU)		sed	Significance[1]
Parameter	Control	MgAsp	$MgSO_4$	1.6g	3.2g	2 d	5 d		
pH_{40min}	6.60	6.56	6.54	6.59	6.51	6.59	6.55	0.078	DO*
pH_{24h}	5.38	5.42	5.41	5.41	5.42	5.41	5.42	0.035	
Lightness (L*)	53.6	51.4	52.4	52.0	51.7	52.0	51.8	0.692	D**
%Drip loss	6.2	4.0	4.4	4.0	4.5	4.3	4.1	0.637	D***
%PSE[2]	80	13	20	15	18	18	15	-	D***

[1]*P<0.05, **P<0.01, ***P<0.001. [2]Exact contingency table test used.

Pigs fed the Mg supplemented diet had improved pork surface lightness, reduced drip loss and reduced incidence of PSE in the LT muscle compared to pigs fed the control diet. Pigs fed dietary Mg at the lower dose (1.6 g) had higher LT muscle pH at 40 minutes post-slaughter compared to pigs fed the Mg diets at the higher dose (3.2 g Mg). The marked effect of dietary Mg supplementation on drip loss and incidence of PSE, and not on muscle pH, suggests that dietary Mg supplementation may have influenced the rate and not the extent of muscle pH decline. Pigs fed the MgAsp diet had lower surface lightness values compared to pigs fed the $MgSO_4$ diet. Although $MgSO_4$ is a cheaper source of Mg, dietary MgAsp supplementation is recommended due to safety issues (potential laxative effect) associated with $MgSO_4$ supplementation in pig diets. As the Mg supplementation dose and duration had limited influence on pork quality indicators, dietary MgAsp supplementation at a lower dose of 1.6 g elemental Mg for 2 days prior to slaughter is as efficacious in improving pork quality as at a higher dose for a longer duration. From an economic viewpoint, this represents a significant reduction in the cost of dietary MgAsp supplementation to improve pork quality.

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EFFECT OF DIETARY MAGNESIUM SUPPLEMENTATION AND MIXING BOARS DURING LAIRAGE ON PORK QUALITY

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Mixing unfamiliar boars during lairage can lead to increased fighting, muscle glycogen depletion pre-slaughter, high ultimate muscle pH post-slaughter and dark, firm, dry (DFD) pork (D'Souza *et al.*, 1999). Past studies have shown dietary magnesium (Mg) supplementation in pigs reduced the effects of stress (electric prodders) by reducing plasma catecholamines at slaughter, muscle glycogenolysis post-slaughter and the pale, soft, exudative (PSE) pork (D'Souza *et al.*, 1998). However, the effect of dietary Mg supplementation in pigs subjected to prolonged stress on pork quality is unknown. This experiment investigated the effect of dietary magnesium aspartate (MgAsp) supplementation on unfamiliar boars mixed during lairage on pork quality indicators.

Forty-eight crossbred (Large White x Landrace) boars were used in a 2 x 2 factorial design experiment consisting of (A) two diet treatments, control diet and diet supplemented with 20g MgAsp/pig 2 days prior to slaughter, and (B) two lairage mixing treatments, familiar boars penned together during lairage and unfamiliar boars mixed during lairage. The pigs were blocked into four replicates of 12 pigs each and were group housed (three pigs per pen). After overnight lairage (18 h), the pigs were CO_2 stunned, slaughtered and chilled as per standard procedure. The number of skin blemishes per animal was recorded as a measure of fighting within each group. Drip loss (suspended sample method) and lightness (L*) were determined on samples removed at 24 h post-slaughter. Pork was classified as DFD if drip loss ≤1% and L*<45. Glycogen content in muscle was determined by the method described by D'Souza *et al.* (1998).

Table 1. The effect of dietary magnesium aspartate (MgAsp) supplementation and mixing boars during lairage on pork quality indicators in the *Longissimus thoracis* (LT) muscle 24 h post-slaughter.

Diet (D)	Control		MgAsp			F Probability values		
Lairage (LM)	Unmixed	Mixed	Unmixed	Mixed	sed	D	LM	D x LM
Skin blemishes	9	47	6	17	16.23	0.181	0.057	0.263
$Glycogen_{5min}$ (mg/g)	10.3	5.5	8.7	9.9	0.726	0.025	0.007	<0.001
pH_{24h}	5.47	5.48	5.45	5.51	0.039	0.874	0.419	0.270
Lightness (L*)	49.4	49.4	48.9	49.4	1.085	0.722	0.793	0.727
Drip loss%	4.5	1.8	3.9	4.4	0.325	0.002	0.001	<0.001
DFD%[1]	0	0	0	0				

[1]Exact contingency table test used.

Boars mixed during lairage tended to have more skin blemishes due to fighting compared to unmixed boars. Unfamiliar boars fed the control diet and mixed during lairage had the lowest muscle glycogen concentrations at 5 min post-slaughter and drip loss% compared to other treatment groups. The low pH_{24h} values and the absence of DFD carcasses suggest that the mixing treatment during lairage was not severe enough and may explain why the influence of Mg supplementation on pork quality was not evident in boars mixed during lairage. These data, however, indicate that dietary Mg supplementation has the potential to improve carcass and pork quality in pigs mixed during lairage to levels similar to that found in unmixed pigs.

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EFFECT OF α-TOCOPHEROL ADDITION DURING PROCESSING ON LIPID OXIDATION OF PROCESSED PORK PRODUCTS

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In processed pork products, lipid oxidation can occur during frozen storage despite the addition of phosphates and nitrite during manufacturing (Trout *et al.* 1998). Although these compounds can act as antioxidants in meat systems, they are water soluble and thus may have limited ability to prevent oxidation of triglycerides in pork fat. Previous research has identified that dietary Vitamin E supplementation to pigs can lead to a reduction in the development of oxidative rancidity in whole muscle and ground pork. The efficacy of post-slaughter addition of α-tocopherol, a fat-soluble compound, on limiting the development of lipid oxidation in processed pork products has not been as widely researched as dietary supplementation.

The objective of this study was to determine whether post-slaughter addition of α-tocopherol influenced the development of oxidative rancidity in two processed pork products during storage at -18°C for up to 37 weeks. A cured, smoked pork sausage was manufactured from coarsely minced pork trim (20% fat) and contained 2% salt, 0.3% sodium tripolyphosphate (STPP), 0.5% sucrose and 1.0% seasoning. An uncured pork roast product was manufactured from denuded and defatted pork shoulder meat (5% fat) cut into 2 cm pieces and mixed with 1.5% salt, 0.5% STPP and 0.5% sucrose. Five different concentrations of α-tocopherol (Covi-ox® T-30P), were added to each processed pork product (0, 100, 200, 500 and 1000 ppm) during mixing, with three replicates made per product for each α-tocopherol concentration. Products were cooked and then stored at –18°C for up to 37 weeks. Lipid oxidation was measured by determining the concentration of thiobarbituric acid reactive substances (TBARS) (Trout and Dale 1990) and a consumer taste panel assessed flavour and overall acceptability of both products.

Table 1. Effects of added α-tocopherol and storage at –20°C for 0 to 37 weeks on TBARS values of pork roast (Roast) and pork sausage (Saus.).

	α-tocopherol (C, ppm)					Time (T, weeks)						Significance[1]	
	0	100	200	500	1000	0	7	14	27	37	sed	C	T
Roast	0.29	0.24	0.22	0.24	0.18	0.20	0.20	0.16	0.21	0.40	0.061	0.11	0.002
Saus.	0.39	0.40	0.34	0.30	0.29	0.15	0.20	0.47	0.57	0.33	0.073	0.095	<0.001

[1]L, linear effect of Vitamin E; T, time.

Pork roast and sausage with 1000 ppm added α-tocopherol tended to have lower TBARS values compared with product containing lower concentrations of added α-tocopherol. As storage time increased, TBARS values of both sausage and roast also increased, however this was irrespective of α-tocopherol concentration. Higher TBARS values observed for the sausage may have resulted from mincing and/or the higher salt content compared with the roast product. Lipid oxidation in both products did not develop to concentrations detectable as off-flavours by the consumer taste panel, even after 37 weeks of storage at -18°C. As lipid oxidation was not a major factor influencing flavour and acceptability of the two processed pork products in this study, the inclusion of α-tocopherol during processing was not considered to offer any additional benefits.

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EFFECTS OF DIETARY SELENIUM CONCENTRATION AND FORM ON LOIN TISSUE SELENIUM CONTENT AND MEAT QUALITY ATTRIBUTES IN GROWER-FINISHER PIGS

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Selenium (Se) has recognized antioxidant properties, but sodium selenite may also act as a prooxidant whereas selenomethionine in organic sources does not possess these properties (Spallholtz, 1994). Because selenomethionine is retained to a greater degree by the pig in muscle and other selenoproteins, this experiment tested the hypothesis that the organic form might improve meat quality attributes. The attributes measured were drip loss and reflectance of loin muscle obtained from pigs fed various dietary concentrations of inorganic and organic selenium.

Crossbred pigs (n=351, initially 20 kg live weight (LW)) were allotted on the basis of sex, LW, and litter (six or seven pigs per pen with equal numbers, sexes within replicate) in a 2 x 4 factorial experiment. Eight treatment diets contained either Se yeast (Sel-Plex, Alltech Inc.) or sodium selenite added at 0.05, 0.10, 0.20, or, 0.30 ppm. A non Se-fortified basal diet (0.06 ppm Se) was fed as a ninth treatment. At 24 h post slaughter (105 kg LW) the end of the loin muscle (10^{th} to last rib) was removed to expose a fresh cut of muscle tissue, and drip loss was determined on duplicate samples (50 g) by the method of Kauffman (1965), with moisture loss recorded for 48 and 96 h post-slaughter after storage at 4^{0}C. Hunter L value was measured on the loin ends using a Minolta model CR-300 chroma meter 1 h after removal for drip loss measurement. Loin Se content was determined.

Table 1. Effect of dietary Se concentration and source on drip loss and carcass characteristics.

	Basal	Inorganic selenium				Organic selenium				SEM
Diet Se, ppm	0	0.05	0.10	0.20	0.30	0.05	0.10	0.20	0.30	
Loin Se, ppm	0.085	0.106	0.114	0.118	0.124	0.134	0.170	0.249	0.332	0.006[bcd]
Drip loss, % water										
0-48 h	3.35	3.95	3.38	3.68	4.09	3.19	3.51	2.96	2.95	0.50
48-96 h	2.33	2.52	2.27	2.25	2.44	2.11	2.46	2.56	2.40	.20
0-96 h	5.68	6.47	5.65	5.93	6.53	5.30	5.97	5.52	5.35	0.63
Hunter L value	46.5	46.9	47.7	48.6	49.8	46.6	48.6	46.5	47.4	1.21[a]

[a]Linear response to inorganic Se content (P≤0.05). [b]Se source x diet Se content interaction response (P≤0.01). [c]Quadratic response to inorganic Se (P≤0.01). [d]Linear response to organic Se level (P≤0.01).

Loin Se content linearly increased with dietary Se, but the increase was markedly higher when the organic form was fed resulting in an interaction (P<0.01). Drip loss and reflectance were unaffected by the organic Se source, but inorganic Se tended to increase drip loss (P>0.05) while values for the organic source were similar to those on the basal diet. Inorganic Se caused a linear increase in loin paleness. These results indicate that while organic Se does not appear to affect meat quality attributes, inorganic Se may have detrimental effects.

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EFFECT OF STUNNING METHOD AND ELECTRICAL STIMULATION ON THE RATE OF AGEING IN PORK

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Variations in the rate of ageing of pork may be due to differences in the rate of pH decline. Alterations in the rate of rigor development influence the activation of the tenderising enzymes, the muscle structure due to shortening, and protein denaturation. Thus an experiment was designed to determine the influence of the rate of pH decline on the rate of ageing. The rate of pH decline was altered by the use of different stunning methods and the use of low voltage electrical stimulation (14 Hz).

Twenty-four Large White x Landrace male finisher pigs of 90 kg body weight were randomly allocated to a 2 x 2 factorial design experiment consisting of two stunning methods (CO_2 or electrical (elect)) and two low-voltage electrical stimulation treatments (none or 15 seconds at 5 minutes post slaughter). Sarcomere length (SL), Warner-Bratzler shear force (WBSF), myofibrillar fragmentation index (MFI), and meat quality parameters were measured at various times post slaughter (Rees *et al.*, 1997). Analysis of variance tests were performed on all data.

Table 1. The effect of stunning (Stun) and stimulation (Stim) on meat quality measurements for pork loins at various times post slaughter.

	Stunning		Stimulation		Significance[1]		
	CO_2	Elect	None	15 sec	Stun	Stim	SED
pH 3 hours	6.11	5.89	6.20	5.80	P < 0.01	P < 0.001	0.076
Time to rigor (hr)	8.25	6.58	9.42	5.42	P < 0.05	P < 0.001	0.777
SL 4 days (μm)	1.78	1.91	1.74	1.95	P = 0.09	P < 0.01	0.067
MFI 4 days	78.4	91.2	89.4	80.2	NS	NS	8.39
WBSF rigor (kg)	6.67	5.97	6.29	6.35	NS	NS	0.457
WBSF 1 day (kg)	7.69	5.87	7.68	5.87	P < 0.05	P < 0.05	0.689
WBSF 2 days (kg)	6.77	4.85	6.60	5.02	P < 0.05	P < 0.05	0.755
WBSF 4 days (kg)	6.19	5.02	6.31	4.90	NS	NS	0.730
% decrease WBSF	5.0	14.9	-1.0	20.9	NS	P < 0.05	10.51
drip loss (%)	2.38	4.61	3.04	3.95	P < 0.05	NS	0.832
Surface lightness (L*)	43.8	46.4	43.5	46.7	P = 0.07	P < 0.05	1.33

[1]NS = not significant, Stim = stimulation, Stun = stunning method.

The time taken for rigor to be reached was reduced by both the electrical stunning and 15 sec of electrical stimulation. The SL measured at 4 days post slaughter was longer in the electrically-stimulated muscles relative to the non-stimulated muscles indicating the 0-2°C chilling conditions used was sufficient to induce cold shortening in the non-stimulated sides. Electrical stimulation and the method of stunning did not influence the myofibrillar fragmentation index at 4 days post slaughter indicating that no differences in proteolytic activity occurred. The WBSF was reduced by electrical stimulation relative to non-stimulated carcasses at 1 and 2 days post slaughter while electrical stunning reduced WBSF at 1 and 2 days post slaughter relative to those stunned with CO_2. Both electrical stunning and stimulation produced pork that would be acceptable to the consumer (WBSF <5 kg). The rate of ageing, defined as the percentage decrease in WBSF from rigor to 4 days post slaughter, was increased by electrical stimulation compared to non-stimulated carcasses but was not influenced by the method of stunning. At rigor, electrical stimulation increased the L* value (surface lightness). Drip loss was increased by electrical stunning relative to carbon dioxide stunning but was not influenced by electrical stimulation. Thus a faster rate of pH decline induced by electrical stimulation resulted in the prevention of cold shortening and improved tenderness.

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EFFECT OF PH AND TEMPERATURE DECLINE ON THE RATE OF AGEING IN PORK

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Variations in the rate of ageing of pork may be due to differences in the rate of pH and temperature decline which influence the activation of the tenderising enzymes, the muscle structure due to shortening, and protein denaturation. The decline in pH can be manipulated by the stunning method, the use of electrical stimulation (14 Hz) and the chilling temperature and thus the influence of pH and temperature decline on the rate of ageing can be determined.

Twenty-four Large White x Landrace male 90 kg body weight finisher pigs were randomly allocated to a treatment design consisting of two stunning methods (CO_2 and electrical (elect)) and three low voltage electrical stimulation treatments (none, 15 sec and 60 sec at 5 min post slaughter) and two chilling temperatures (2°C and 14°C until pH <5.8 was obtained). Sarcomere length (SL), Warner-Bratzler shear force (WBSF), myofibrillar fragmentation index (MFI) and meat quality parameters were measured at various times post slaughter (Rees *et al.*, 1997). Analysis of variance tests were performed on all data.

Table 1. The effect of stunning (stun), stimulation (stim) and chilling (chill) temperature on meat quality measurements for pork loins at various times post slaughter.

	Stunning x Stimulation				Chilling		Significance[1]		
	Elect None	CO_2 None	CO_2 15 sec	CO_2 60 sec	2°C	14°C	Stun x Stim	Chill	SED
pH 3 hours	6.24^{a}	6.27^{a}	5.83^{b}	5.59^{c}	6.03^{a}	5.94^{b}	***	**	0.075
Time to rigor (h)	9.2^{c}	8.33^{c}	5.8^{b}	3.2^{a}	7.2^{a}	5.9^{b}	***	**	0.74
SL 4 days (µm)	1.83^{b}	1.64^{a}	1.88^{b}	1.88^{b}	1.76^{a}	1.86^{b}	**	*	0.081
MFI 4 days	73.1	64.2	65.2	67.7	56.9^{a}	78.2^{b}	NS	**	9.13
WBSF rigor (kg)	7.33	7.58	7.31	6.27	7.26	6.99	NS	NS	0.747
WBSF 1 day (kg)	6.86^{ab}	8.40^{b}	7.84^{ab}	5.96^{a}	8.04^{a}	6.50^{b}	*	***	0.994
WBSF 2 days (kg)	6.48	7.63	7.55	6.21	7.79^{a}	6.14^{b}	NS	**	0.461
WBSF 4 days (kg)	6.75^{ab}	7.81^{b}	6.49^{a}	5.76^{a}	7.63^{a}	5.77^{b}	*	***	0.598
% decrease WBSF	10.9	-2.6	7.9	6.0	-9.9^{a}	14.3^{b}	NS	*	10.78
Drip loss (%)	2.0^{a}	2.6^{ab}	4.4^{bc}	5.8^{c}	3.9	3.5	*	NS	1.00
Lightness (L*)	45.8^{a}	45.1^{a}	48.2^{ab}	50.2^{b}	46.7	47.9	*	NS	1.89

[1]NS, not significant. ^{abc}Within rows and treatment combinations, values with different superscripts are significantly different (P≤0.05), * P≤0.05, **P≤0.01, ***P≤0.001.

The time taken for rigor to be reached was reduced by electrical stimulation and by chilling at 14°C which resulted in the prevention of cold shortening, with SL increasing with stimulation and chilling at 14°C. Chilling at 14°C resulted in an increased proteolytic activity post slaughter as indicated by the increased MFI values relative to chilling at 2°C. The prevention of cold shortening and the increased proteolytic activity following chilling at 14°C resulted in improvements in WBSF from 1 to 4 days post slaughter and a greater percentage decrease in WBSF from rigor to 4 days post slaughter. However the faster rate of pH decline induced by CO_2 stunning and electrical stimulation only resulted in lower WBSF at 1 and 4 days post slaughter which may be due to increased protein denaturation suggested by the increased drip loss and L* value. None of the treatments resulted in consumer acceptable levels of tenderness, that is a WBSF of less than 5 kg. However, chilling at 14°C did not induce protein denaturation. Thus increasing the chilling temperature was an effective way to improve pork tenderness due to the prevention of cold shortening and the increased proteolytic activity.

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DUAL ENERGY X-RAY ABSORPTIOMETRY TO PREDICT WHOLE BODY AND CARCASS COMPOSITION IN PIGS

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Processor grading of carcasses on the basis of P2 fat depth may not allow the end use of carcasses to be optimised because of the variability in fat distribution across the carcass. Therefore, there is a need to grade carcasses based upon total lean meat yield in the whole body. One reliable and convenient method for determining both total and regional body composition is dual energy X-ray absorptiometry (DXA) (Mitchell *et al.* 1998), and recent advances have markedly improved DXA scan speeds and accuracy. Therefore, the aim of this study was to determine whether DXA could be used to predict body composition in the whole body and carcass of pigs.

The Hologic QDR4500 DXA was used to determine body composition in pigs. Five gilts were scanned at approximately 15 weeks and 30 gilts at 23 weeks of age. Pigs were slaughtered commercially after the final *in vivo* scan, eviscerated and the empty carcass scanned before chemical analysis. Measures made by DXA, of lean, fat and bone mineral content (BMC) were compared with chemically determined measures. Fat depth at P2 was measured directly and compared with chemically determined fat in the carcass. The BMC predicted by DXA was adjusted to be 85% of total ash content (Jebb *et al.*, 1995) and carcass lean was defined as the sum of protein and water.

Table 1. Relationships between whole body and carcass composition as determined by DXA and chemical analysis of the empty body and carcass, respectively (linear model used is $y=ax+b$ where y is chemical composition and x is DXA prediction).

	a	*b*	rsd	r^2
Live Animal				
Fat (kg)	1.30	0.73	1.85	0.92
Lean (kg)	0.870	-2.31	2.40	0.95
BMC (kg)	1.04	0.49	0.16	0.85
Carcass				
Fat (kg)	1.22	1.06	1.73	0.92
Lean (kg)	1.07	-2.66	2.02	0.96
BMC (kg)	1.08	0.37	0.15	0.85

The DXA predictions of both live body and carcass fat were highly correlated with proximate analyses although the in-built algorithms underestimated fat. A positive correlation was also observed between P2 fat depth and chemical fat but the correlation was not as strong as the DXA prediction (r^2=0.68, rsd=3.38). Lean tissue predicted by DXA, in the live animal and carcass, was highly correlated with proximate measurements. While DXA slightly underestimated the lean content of the carcass, it overestimated the lean content of the live animal. At least some degree of overestimation *in vivo* arose from water in the gut lumen, which was assumed by the DXA algorithms to be associated with lean tissue. These data demonstrate the potential of DXA to determine body composition in the live animal and carcass, and to improve on current routinely used methods. Further studies utilising a greater range in live weight and body composition are required to clarify the effect of gut water on estimation of body composition.

PIG RESEARCH AND DEVELOPMENT CORPORATION

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REPEATABILITY OF MULTIPLE FREQUENCY BIOELECTRICAL IMPEDANCE ANALYSIS

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As consumer and commercial demand for lean pork increases, so does the need for a rapid, cheap and accurate means to measure body composition. Multiple frequency bioelectrical impedance analysis (MFBIA) measures the opposition to the flow of a small current (350μA) through the most conductive tissues. Multiple frequency bioelectrical impedance analysis is highly correlated with lean tissue mass, and has been used successfully in estimating human body composition. Bioimpedance has the potential to estimate carcass composition in pigs (Swantek *et al.*, 1992). The aim of this study was to determine whether MFBIA was repeatable in individual pigs.

Studies were conducted on live animals and carcasses. Study one was conducted to determine repeatability and optimal electrode location in live animals. Measurements were repeated 3 times at various sites on five anaesthetized Large White x Landrace boars (mean 83.4kg liveweight (LW)). In study two, 10 (five male and five female) whole carcasses (no head and viscera) had bioimpedance measured in the same manner. Bi-polar (one needle for each electrode) and tetra-polar (two needles for each electrode) arrangements were used. Bi-polar measurements were erratic and only data from tetra-polar measurements are reported. Whole body sites used were (1) right fore leg-right hind leg; (2) left fore leg-right hind leg; (3) left fore leg-left hind leg; and (4) right fore leg-left hind leg.

Table 1. Repeatability of bioimpedance measurements in live pigs and carcasses.

Site	Live	Hot carcass		Cold carcass	
		Male	Female	Male	Female
Midline	0.69	0.75	0.62	0.86	0.91
Whole Body 1	0.95	0.90	0.65	0.79	0.67
2	0.61	0.95	0.72	0.8	0.86
3	0.64	0.83	0.94	0.81	0.96
4	0.96	0.96	0.98	0.98	0.96
Ham	0.96	0.91	0.89	0.68	0.85
Shoulder	0.49	0.99	0.95	0.70	0.80

Repeatability is the correlation of repeated measurements on the same site on each individual. Table 1 shows that bioimpedance was highly repeatable over a range of sites. Sites with values closest to 1.0 are considered highly repeatable. Study one shows that repeatability in live animals was best at the ham and whole body sites one and four. Carcass repeatability was best at the ham, shoulder, and whole body site four in male hot carcasses. Hot female carcasses were highly repeatable at the shoulder and whole body sites three and four. In cold carcasses, sites most repeatable were the whole body site four in males, while in females the most promising sites were the midline and whole body sites three and four. It is recommended that the whole body site four be used for subsequent readings. Sex or weight did not affect repeatability. The high repeatability of measurement suggests that MFBIA, as a body composition analytic method is worthy of further study.

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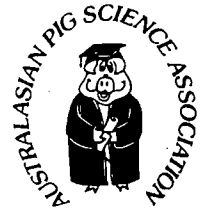

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A SYMPOSIUM – ANTIBIOTICS IN PIG PRODUCTION

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Introduction

Antibiotics are an important tool in maintaining health and production efficiency in the pig industry. With the development of intensive continuous flow production units, with large numbers of pigs of varying age sharing the one airspace, producers and veterinarians have come to rely on antibiotics not only as therapeutic agents, but as the basis of many disease control and prevention programmes.

Intensive forms of pig production developed for at least two decades before major concerns were raised within the industry about the widespread use of antibiotics to limit and reduce the impact of disease. During this period a perception developed within consumer organisations that pig and poultry meat contained significant amounts of antibiotic. Although the perception was based on largely anecdotal evidence, it lingers in the minds of sections of the consumer and welfare lobby. It is up to the pig industry to dispel such perceptions by developing more effective disease control strategies, reducing the use of antibiotics whenever possible, and explaining the continuing need for using antibiotics in defined situations.

During the past decade and a half, there have been major advances in our understanding of the role that alternative strategies have in reducing disease prevalence and limiting the impact of disease on production. Both the aetiology and pathogenesis of many of the health problems found in pig herds are multifactorial. The fact that a micro-organism or micro-organisms are involved is only part of the story. In a number of examples, such as enteric disease and respiratory disease, environment and management factors may be more important than the range of pathogens present in determining the level of clinical disease expressed in the herd. A number of reports have been published which demonstrate that by manipulating factors such as management, nutrition, housing, shed environment, pig flow and stocking density, the prevalence of disease and its impact on production can be reduced. Such tactics not only have the potential to reduce disease prevalence and increase production, but also allow for more judicious use of antibiotics. Therapy can be targeted at animals over key periods during the growing cycle, avoiding the necessity of medicating animals for long periods late into the growing period prior to slaughter. Production systems such as medicated early weaning, segregated early weaning, age-segregated rearing, all-in/all-out and multi-site all describe a new genera of production systems which have the potential to reduce the producer's reliance on antibiotics to maintain production efficiency.

Reducing disease prevalence by management also reduces the level of contamination of infectious agents in the farm environment and reduces the opportunity for infection to spread. If the pathogen load and the stress on animals can be reduced, other procedures such as vaccination, dietary manipulation and the use of probiotics have the potential to become even more efficacious.

The symposium provides an opportunity to review the current situation and to discuss alternative strategies available to pig producers. However, the solution to the problems of residues, resistance, over-use and mis-use of antibiotics may only be found in using a number of strategies in concert. For example, implementing vaccination programmes, modifying diets to control enteric disease and feeding probiotics may only reach their full potential in reducing the use of antibiotics, when housing and environment are optimal. To improve shed hygiene, stocking density and shed environment without implementing vaccination programmes or manipulating diets may result in modest improvements, whereas using all of these strategies together may result in significant gains with a significant reduction in the use of antibiotics.

There is still a long way to go in understanding the complex interactions between the physiological and immunological responses of animals to their environment. However, the data presented during the symposium suggest that there is great potential in exploring strategies that may be used in combination with, or as alternatives to, antibiotics, to reduce or eliminate the impact of disease in pig herds.

THE DOWN-SIDE OF ANTIBIOTIC USE IN PIG PRODUCTION: THE EFFECT ON ANTIBIOTIC RESISTANCE OF ENTERIC BACTERIA

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Abstract

Use of antibiotics for growth promotant, prophylactic or therapeutic purposes in pigs has the potential to result in the development of tissue residues and the emergence of populations of antibiotic resistant bacteria in treated animals. Residues may have some direct effects on some people but their effect on the development of resistant bacterial populations in consumers is debatable. Pig pathogens and commensals both demonstrate resistance to antibiotics. The extent of antibiotic resistance in pig isolates is difficult to define in the absence of systematic surveillance and monitoring. Resistance in pig pathogens can lead to less effective therapy or even treatment failure. There is also concern about the spread of antibiotic resistant strains of food-borne pathogens such as salmonella and campylobacter from treated pigs to people. There is currently much debate about the contribution of antibiotic resistant enteric bacteria in pigs (for example enterococci) to the emergence of antibiotic resistant strains of these organisms in people. While molecular studies support the view that antibiotic resistance genes can spread from animal to human isolates, the frequency and extent to which this occurs is still a controversial issue. Nonetheless, it is time to develop protocols for better and more strategic use of antibiotics in pigs.

Introduction

Antibiotics are used in pig production for therapeutic, prophylactic and growth promotant purposes. It is quite clear that such use can result in antibiotic residues in the tissues of treated animals and that exposure of bacteria in animals to antibiotics selects for populations of resistant bacteria. What needs to be resolved is the significance of these problems when balanced against the importance of antibiotic therapy in the treatment and control of infectious diseases and the economic and other benefits of growth promotants.

Antibiotic residues

In the past there has been relatively little expression of concern by animal health authorities about antibiotic resistance as a consequence of antibiotic use and any controls on antibiotic use focussed on control of residues in the tissues of treated animals. The concerns of veterinary authorities about residues have been related to their potential to disrupt import and export trade through imposition of non-tariff trade barriers. The food safety issue for residues seems to revolve around allergic reactions and to adverse effects on the flora of the human gastrointestinal tract (selecting for resistance or transfer of resistance). The Swan Report (Joint Committee on the Use of Antibiotics in Animal Husbandry and Veterinary Medicine, 1969) discussed both antibiotic residues and resistance. The report noted that penicillin residues in milk could provoke allergic reactions in sensitised individuals but concluded that there were no other adverse effects associated with antibiotic residues. Triggering of allergic reactions in sensitised individuals by penicillin residues is well-documented (Dewdney *et al.*, 1991) but these authors conclude that there is no evidence that any individual has become sensitised by food residues of either penicillins or macrolides. Dayan (1993) noted that cases of proven allergy to substances in food are very rare, although adverse reactions to antibiotics have been linked to cases of chronic urticaria. However, he concluded that allergy to antibiotic residues in food is very rare. A rare fatal blood dyscrasia in

individuals sensitised to chloramphenicol could also be triggered by chloramphenicol residues in food (Settepani, 1984).

There is little scientific data on the effect of antibiotic residues on the bacterial flora of the human intestinal tract. Clearly such studies are very difficult to carry out (Corpet, 1993; Kidd, 1994) and Gorbach (1993) points out that most human studies have used therapeutic rather than residue-range concentrations and that to date results have been confusing.

Nevertheless many developed countries have continued to monitor and survey tissues from animals (Table 1). Acceptable daily intakes (ADIs) for people based on "no effect levels" and safety have been calculated for many antibiotics, despite the lack of data. From these a tolerance level or maximum residue level (MRL) is calculated. Established MRLs are modified and often reduced as the techniques for detection of residues improve and become more sensitive. Conversely, quite a few have been increased recently as data becomes available that facilitates better risk assessment. Monitoring is largely driven by the requirements of countries importing animal products, although in Australia the National Food Authority has a significant role too. Australia, along with its major trading competitors, monitors meat and other foodstuffs through the National Residue Survey.

Table 1. Monitoring of pig tissues for antibiotic residues in Australia, Canada and the United Kingdom.

Country	Number tested	Year	Number > MRL (%)
Australia	1,469	1997	38 (2.6%)*
United Kingdom	14,517	1997	46 (0.34%)
Canada	12,103	1996/1997	88 (0.72%)

*Most Australian violations are for chlor- and oxytetracycline where the Australian MRLs are considerably lower than that of its trading partners; other residues found at a much lower frequency are sulphadimidine, neomycin and dihydrostreptomycin (J. Webber, personal communication).

Antibiotic resistance

There are problems in trying to compare results from different studies of antibiotic resistance in bacteria. A variety of techniques have been used to assess antibiotic susceptibility, but there has been no standardisation of cut-off values for ascribing sensitivity or resistance and there are differences in antibiotic treatment regimens in different countries. These issues must be taken into account when reviewing the literature about antibiotic resistance in animal isolates.

Escherichia coli

Antibiotic resistance is readily detected in bacterial isolates from pigs (and other animals exposed to antibiotics). There are more studies on antibiotic resistance in *Escherichia coli* than on any other bacteria. As early as 1957 it was found that *E. coli* isolated from pigs and poultry receiving tetracycline as a growth promotant were resistant to tetracycline (Anderson, 1968; Smith, 1967). Resistance to other antibiotics was detected as new agents were introduced for therapeutic and growth promotant purposes (Anderson, 1968; Smith, 1967). Some workers (Linton *et al.*, 1988; Lee *et al.*, 1993) also noted the occurrence of tetracycline resistance on some piggeries even though tetracyclines had not been used in those piggeries. Others have commented that resistance persists for a long time after antibiotics are withdrawn (Rollins *et al.*, 1976).

Feeding oxytetracycline to recently weaned pigs was found to lead to a rapid increase in the incidence of tetracycline resistance which was widely distributed among all strains of *E. coli* present, rather than restricted to a few selected clones (Hinton *et al.*, 1985). More recently, feeding low doses of ampicillin to chickens was shown to select for

high level resistance to that antibiotic (El-Sam *et al.*, 1993). Marshall *et al.* (1990) has described an elegant experiment that demonstrated that resistant strains of *E. coli* spread among animals (and to other species such as mice) even in the absence of on-going antibiotic treatment.

There are many papers describing various aspects of antibiotic resistance in *E. coli* isolates from pigs and all appear to agree that if pigs are treated with antibiotics, resistance emerges to varying degrees. However, there are differences between antibiotics in the time taken for resistance to be evident and in the extent of the resistance that is seen. In herds treated with tetracycline, aminoglycoside and sulphonamide, widespread resistance is seen (Rollins *et al.*, 1976; Williams Smith, 1980; Wray *et al.*, 1993a; Franklin, 1984; Nijsten *et al.*, 1993; Dunlop *et al.*, 1998a; Matthew *et al.*, 1998; Sunde *et al.*, 1998). However, resistance to other antibiotics such as ampicillin and olaquindox is less widespread (Linton *et al.*, 1988; Dunlop *et al.*, 1998a,b). Resistance to more than one class of antibiotics is the rule rather than the exception in these published studies.

There are no published studies of antibiotic resistance of Australian pig isolates. The Animal Health Committee, 1982 (unpublished report) noted the resistance patterns of *E. coli* isolates collected between 1976 and 1981. About 200 isolates were examined each year. Reasonably widespread resistance to tetracyclines and streptomycin were found and a lower prevalence of resistance to neomycin, furazolidone, ampicillin and chloramphenicol. A recent Australian study (Barton and Pratt, unpublished) found widespread resistance to nitrofurans, sulphonamides, tetracycline, streptomycin, apramycin, neomycin; less widespread resistance to spectinomycin and negligible resistance to gentamicin; moderately widespread resistance to carbadox, trimethoprim, ampicillin and augmentin; very little resistance to cephalosporins; and no resistance to fluoroquinolones. Approximately 90% of the isolates were resistant to 3 or more classes of antibiotics, with around 20% resistant to 6 or more classes.

Salmonella spp.

Because salmonella is a recognized food-borne pathogen, a number of the published reports of resistance patterns in pig isolates have been linked with studies of human isolates (Threlfall *et al.*, 1993; Seyfarth *et al.*, 1997) or with concerns about resistance to particular antibiotics (Wray *et al.*, 1986; Heurtin-Le Corre *et al.*, 1999). A number of countries have carried out surveys of resistance in salmonella which have included pig isolates or have on-going antibiotic resistance surveillance programmes (Wray *et al.*, 1993b; Seyfarth *et al.*, 1997; Commission on Antimicrobial Feed Additives (CAFA), 1997; Baggesen and Aarestrup, 1998; Fedorka-Cray *et al.* 1998). The results are not easy to interpret because some reports do not distinguish between different serovars of *Salmonella enterica* and it is recognized that some serovars such as Typhimurium are much more likely to be resistant than other serovars such as Dublin and Enteritidis PT4 (Ministry of Agriculture Food and Fisheries (MAFF), 1998). The overall conclusion is that resistance is generally less prevalent in salmonella but that resistance to tetracyclines, sulphonamides and streptomycin is quite widespread.

At this stage there is no formal surveillance of salmonella resistance in Australian animal isolates, although some information is collected by the National Enteric Pathogen Surveillance Programme. Murray *et al.* (1986) reported a survey of 346 Australian pig isolates and found moderately widespread resistance to tetracycline and streptomycin and a lower prevalence of resistance to neomycin, furazolidone, ampicillin and chloramphenicol.

Campylobacter spp.

Thermophilic campylobacters are not pathogenic in pigs so any reports on antibiotic resistance are derived from specific surveys. It is recognized that *Campylobacter coli* is more frequently resistant to some antibiotics, including erythromycin and fluoroquinolones. Erythromycin-resistant campylobacters have often been reported in pigs (Elharrif and Megraud, 1984; Hariharan *et al.*, 1990; Moore *et al.*, 1996), with the latter reporting more frequent resistance in C. *coli* isolates. Aarestrup *et al.* (1997) found

that most *C coli* isolates from pigs were either co-resistant to tylosin and erythromycin or sensitive to both. Barton and Pratt (unpublished) in an Australian study of 116 isolates found significant levels of resistance to erythromycin, clindamycin, ampicillin and tetracycline, but no resistance to fluoroquinolones (which are not registered for use in livestock in Australia).

Enterococcus spp.

Enterococci are not pathogens in pigs. However, in recent years there has been much interest in the resistance of pig isolates to avoparcin because of concerns about transfer of glycopeptide resistance from pig isolates to human strains (Danish Veterinary Laboratory, 1995; Klare *et al.*, 1995; Aarestrup *et al.*, 1996; Jensen *et al.*, 1998). These reports all associate feeding avoparcin to pigs with the appearance of avoparcin/vancomycin resistant enterococci in the treated pigs. Other antibiotic resistance reported in pig enterococci includes macrolide-lincosamide-streptogramin group (Dutta and Devriese, 1982), tylosin (Aarestrup and Cartensen, 1998) and virginiamycin resistance (Hammerum *et al.*, 1998). An Australian study (Barton and Pratt, unpublished) found widespread resistance to macrolides (erythromycin and tylosin), clindamycin, tiamulin and virginiamycin; aminoglycosides (streptomycin, gentamicin, apramycin); spectinomycin; tetracycline; monensin; moderately common resistance to bacitracin, a low prevalence of resistance to ampicillin and no resistance to vancomycin or teicoplanin.

Human health concerns

There is worldwide concern about the spread of antibiotic resistance from animal isolates of bacteria to human pathogens. In October 1997 the World Health Organisation (WHO) organized a meeting on this topic (WHO, 1997), followed by another WHO meeting on the use of fluoroquinolones in animals (WHO, 1998). At much the same time the Swedish Commission on Antimicrobials reported on the importance of growth promotant antibiotics and emergence of resistance problems in human isolates (CAFA, 1997) and MAFF in the UK issued their report (MAFF, 1998). There was also a report from the House of Lords in the UK on antibiotic resistance (House of Lords, 1998) and a committee of enquiry in the USA (Committee on Drug Use in Animals, 1998). In the meantime some of the Scandinavian countries had been taking action to restrict the use of growth promotant antibiotics and to prohibit totally the use of some agents. More recently, the European Union has withdrawn approval for the use of some growth promotants and this is likely to have implications for other countries exporting meat and other animal products into Europe. All of these reports have advocated tighter controls and restrictions on the use of some classes of antibiotics in animals.

Australia responded by establishing a Joint Expert Technical Advisory Committee on Antibiotic Resistance (JETACAR) which released a draft report in April 1999. It would seem that the recommendations are likely to be directed towards better controls on the use of antibiotics, encouraging prudent use of antibiotics and reviewing the use of some antibiotics as growth promotants and for long term prophylactic use in animals.

The question of whether or not antibiotic use and antibiotic-resistant isolates of bacteria from animals have an impact on human health has been under scrutiny since the Swan report was published (Joint Committee on the Use of Antibiotics in Animal Husbandry and Veterinary Medicine, 1969). Some of the issues have been recently reviewed (Barton 1998, 1999; Witte, 1998). Not everyone accepts that there are links between antibiotic use and resistance in animal isolates and resistance in human pathogens. However, scientific evidence is mounting, for at least some organisms and some antibiotics. For example, salmonella is a well-recognized food-borne pathogen, so resistant strains of salmonella, including *Salmonella* Typhimurium DT104 do spread from animals to people via the food chain (Threlfall *et al.*, 1993; Wall *et al.*, 1995; Glynn *et al.*, 1998) and by direct contact (Fone and Barker, 1994). Apramycin resistance in human strains of salmonella provides evidence of transfer of resistance from animal to human isolates, since apramycin is not used in human medicine (Wray *et al.*, 1986).

Campylobacter is another food-borne pathogen. Fluoroquinolone-resistant strains of *Campylobacter jejuni* were isolated from people soon after enrofloxacin started to be used in poultry (Jacobs-Reitsma *et al.*, 1994; Velaquez *et al.*, 1995). A recent paper from the USA documents not only a temporal association between use of fluoroquinolones to treat chickens and resistance in human isolates but molecular sub-typing indicated that resistant strains from chickens were very similar to resistant strains from people (Smith *et al.*, 1998). A further concern with fluoroquinolone-resistance is resistance in animal isolates of salmonella (Griggs *et al.*, 1994) or transfer of this resistance from campylobacter (Herikstad *et al.*, 1997) or *E. coli* to multi-resistant salmonella (Heurtin-Le Corre *et al.*, 1999). There is observational evidence from case studies indicating direct spread of resistant commensal enteric bacteria from animals to people (Nijsten *et al.*, 1994, 1996; Van den Bogaard *et al.*, 1997). Although it has been easy to find resistant bacteria in animals and similar resistance patterns in isolates from people, until recently few isolations have been made from food (Quednau *et al.*, 1998; Klein *et al.*, 1998). The early evidence for transfer of glycopeptide resistance from animals to people came from observational studies (e.g., Danish Veterinary Laboratory report, 1995; Aarestrup *et al.*, 1996) but scientific evidence based on molecular analysis of the resistance genes is increasing (Jensen *et al.*, 1997). Molecular studies have helped clarify the link between avoparcin resistant enterococci and VREs in people: to date there is only evidence for transfer of *van*A resistance from animal isolates to human isolates and some suggestion that use of avoparcin does not select for *van*B resistance (CAFA, 1997). *van*A mediated resistance is the commonest form of vancomycin resistance in human isolates in Europe whereas *van*B resistance is the form most commonly seen in the USA (where avoparcin is not used in animal feeds) and Australia. Molecular studies are also providing supporting data linking transfer of resistance with other antibiotics such as virginiamycin (Hammerum *et al.*, 1998).

Conclusions

So how can residue and resistance problems associated with antibiotic use in piggeries be controlled? One of the first requirements is a system for detecting and monitoring the problem. While there is a programme for detection and trace-back of residues, there is no mechanism for monitoring and surveillance of antibiotic resistance in any livestock in Australia. There has been some surveillance of salmonella in the UK and Sweden since the 1970s (MAFF, 1998; CAFA, 1997) and more recently in France, (Martel and Coudert, 1993) but there has been no systematic coverage of either animal (pig) pathogens or animal commensals of concern to public health authorities. The emergence of the vancomycin resistant enterococci (VRE) problem has led to some changes, particularly in Europe (CAFA, 1997; Aarestrup *et al.*, 1998a,b). Recently the USA commenced monitoring of resistance of salmonella isolated as part of their abattoir Hazard Analysis Critical Control Point (HACCP) programme (Fedorka-Cray *et al.*, 1998).

Farmers as the end-users of antibiotics must be made more aware of the antibiotic resistance problem and it is crucial that on-farm Quality Assurance programmes address responsible use of antibiotics more broadly than simply trying to reduce the incidence of residues in tissues. Clearly there is a case for reducing antibiotic usage on farms. Some of this reduction can come from development of and adherence to guidelines for prudent use of antibiotics. It would be also worthwhile considering segregating antibiotics into those for human use and others for animal use. It is difficult to justify using potentially valuable antibiotics as growth promotant agents, and long-term use of antibiotics for prevention of disease clearly contributes to the antibiotic resistance problem. Alternatives to antibiotics include improvements in management systems and piggery design and layout, development of more vaccines, probiotics and competitive exclusion products and perhaps bacteriophages or other novel approaches to killing microbes. Some of these alternatives are discussed in other papers in this symposium.

Consumers are concerned about the wholesomeness and safety of foods. Increasing litigation based on the premise that people should not suffer any harm from food must alert the pig industry to the need to ensure pork is a safe, healthy product for consumers. The aim should be to produce a product with no residues and with minimum levels of

bacteria which have no acquired antibiotic resistance. It could be argued that most antibiotic resistance problems in people are associated with inadequate controls and misuse of antibiotics in hospitals and in the community generally. There is, however, sufficient evidence to support calls for reduction and improved controls over the use of antibiotics in animals because antibiotic resistant strains of food-borne pathogens such as salmonella and campylobacter do spread from animals to people. Molecular evidence indicates clearly that resistance genes transfer readily among bacteria so there is likely to be spread from resistant animal strains to human strains and probably vice versa. What is not clear at this stage is how often this transfer occurs in people and animals.

Controls on animal usage will not resolve the current problems in human medicine but may well help extend the useful life of any new classes of antibiotics if and when they are introduced.

Symposium continued on next page

THE ROLE OF MANAGEMENT AND HUSBANDRY IN PIG HEALTH, WITH EMPHASIS ON POST-WEANING ENTERIC DISORDERS

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Abstract

The paper deals with the role of management and husbandry in pig health. Emphasis is placed on multifactorial enzootic diseases because they are commonly present in herds. Because a microbial agent is frequently involved, these diseases are the reason for most of the antibiotic usage in the pig industry. The case of post-weaning digestive disorders is examined to illustrate the usefulness of a technical approach to disease prevention, as an alternative to using antibiotics. The approach included a preliminary study to demonstrate that the environment has a major influence on disease expression on the farm. This was followed by a survey of 106 farms to extract the principal risk factors associated with enteric disorders. Preliminary work suggested a major role for the environment on disease expression. The validity of the results was then tested by measuring the risk factors identified in the survey on four severely affected farms and designing a technical programme to reduce the risks identified on each farm. The programmes were designed in agreement with the farmers and the health status of the herds monitored. Following modification of the farm risk factor profiles, by changing husbandry practices and improving temperature and hygiene, disease severity declined dramatically. The relevance and requirements of the approach are discussed.

Introduction

The production of pigs, and livestock in general, has undergone considerable changes in recent decades. In many countries, more changes have probably occurred over the past 50 years than during the previous five centuries. Scientists promoting the new animal production systems defined technical rules and standards for animal husbandry, but their main objective was to increase financial income as a result of better technical efficiency. Intensive systems of animal production, which take advantage of human, economical and geographical resources, have been developed (Dijkhuisen and Davies, 1995). These have led to an overall increase in the volume of goods produced. As far as pig production is concerned, these changes have resulted in a progressive increase in herd size. There is now a clear move to establish a limited number of large units in specialized areas rather than a large number of small units. The people involved in pig production now have a well-defined job and managerial jobs are of increasing importance. In many cases the situation looks like a juxtaposition of independent technical competences rather than a true organisation of tasks with a common goal. A similar partitioning of disciplines has also been the rule in animal science. Geneticists, nutritionists, ethologists go their own way, as do the microbiologists in their laboratories and veterinarians on the farm. In many cases there has been a trend to introduce antibiotics, not only to treat sick animals, but also to avoid re-occurrence of disease. Antibiotics have also been used as feed additives to enhance performance.

At the farm level, the facts are that illness, health deviations or poor performance occur when the zootechnical situation is inadequate. The terms "animal hygiene", "preventive veterinary medicine" and "ecopathology" describe the scientific knowledge derived from different disciplines that are concerned with animal health and performance (Madec and Tillon, 1986; Ekesbo, 1988). The fundamental rule is that "prevention is better than cure". There is increasing pressure from the public, consumer organisations and legislators for a better usage of antibiotics in animal production (Barton 1998; Anonymous, 1999). Such messages clearly merit attention. The present paper deals with an experience in pig disease prevention in France.

The different classes of pig diseases and their consequences in prevention

Pig diseases can be classified into three general categories, major monofactorial diseases, other epidemic viral diseases and enzootic health disorders.

Major, monofactorial diseases

Major, monofactorial diseases have common characteristics which include viral aetiology and a severe impact on the herd. They can be reproduced by simple inoculation of the virus into immunologically naïve pigs, kept under experimental conditions. The clinical signs are usually significant and unequivocal, and laboratory methods of diagnosis are available. They are known as monofactorial diseases because of the exclusive role of the aetiological agent, and are notifiable in many countries because of their severe impact on the herd, ability to spread and their possible danger to other animal species. Most of these diseases appear on list A of the OIE (Office International des Epizooties). Swine fever (African and classical), pseudorabies and foot and mouth disease belong to this category. Although many areas are currently free, such diseases can provide a real threat to the pig industry.

Other epidemic viral diseases

The principal feature of other epidemic viral diseases is an ability to spread rapidly and a tendency to exhibit fluctuations in densely populated pig regions. They can easily be reproduced in experimental facilities but they have a milder impact than those of the previous group. The severity of clinical signs in a herd often depends on a number of factors, which includes the herd health status at the time of infection. The archetype is swine influenza. Other diseases such as Porcine Respiratory Coronavirus (PRCV), Transmissible Gastro-enteritis (TGE) and Porcine Reproductive and Respiratory Syndrome (PRRS) currently in Europe, can also be included in this group.

Enzootic health disorders

Enzootic health disorders, unlike those of the previous groups, may or may not involve an infectious agent. Their herd severity is dependent on specific management and housing conditions. When an infectious agent is involved, its presence in the herd is not enough to give rise to the typical clinical manifestations of the corresponding disease. This means that such diseases are strongly dependent on environmental conditions. Sufficient infection pressure is needed for full disease expression. They commonly exhibit a wide range of severity but usually exist in a chronic form on most farms. Respiratory tract disorders (e.g., Enzootic Pneumonia, Actinobacillosis, Atrophic Rhinitis), digestive problems (e.g., white scour in the sucking pig, post-weaning diarrhoea, colitis), reproductive disorders (e.g., Metritis-Mastitis-Agalactia syndrome, cystitis), locomotor disorders and behavioural deviations fall into this third group of diseases. They represent by far the most important group that farmers and veterinarians have to face in daily practice. Due to their multifactorial aetiology they are usually difficult to reproduce experimentally in their common chronic form, even in *Specific-Pathogen-Free* pigs, without resort to specific inoculation procedures. Such specific procedures are far from the spontaneous natural means of pig contamination that occurs on the farm. It has been calculated that the financial losses attributable to this latter category of diseases accounted for about 80% of the total losses caused by disease in French pigs (Tillon *et al.*, 1980). Programmes designed to control these diseases also account for the majority of antibiotics.

Several strategies can be developed to control or reduce the impact of enzootic diseases on a herd. The purchase of pathogen free pigs is one of the best solutions. It is especially relevant when populating a new herd. On a routine basis, in a farm infected with such pathogens, the challenge is to adapt the management strategy so that the pathogen population remains at a low level. Direct drug control of the pathogens cannot be recommended as most of the microbes involved are ubiquitous and/or not always harmful. In the case of a well-defined microbial causal agent, vaccination might help, but the financial return will require evaluation. On the other hand the conditions in which the

pigs are raised are of paramount importance. On-farm management procedures, which reduce the infection pressure of potential pathogens and maintain a proper immune balance at the herd level, need to be implemented and remain the most secure solution in the long run. In consequence, efforts need to be directed primarily towards housing, feeding, husbandry and stockmanship (Kovacs *et al.*, 1967; Muirhead, 1983; Nicks and Dechamps, 1986; Thomas, 1984). In the following section the example of post-weaning digestive disorders will be examined to illustrate the relevance of such a technical or ecopathological approach to enzootic diseases.

An ecopathological approach to post-weaning digestive disorders

As post-weaning digestive disorders are an important problem in many herds, this condition was used to demonstrate an ecopathological approach to solving health problems. The study was carried out in close association with the pig industry. A preliminary study was designed to investigate the role of diet and general housing on the development of enteric health problems in growing pigs. In a subsequent study, a survey was designed to identify the risk factors associated with the onset of post-weaning disorders. A herd health programme based on the results of the survey was implemented in phase 3.

The preliminary study

Five severely affected farms were chosen for the preliminary approach. The main objective was to determine the best way of investigating the problem (Madec, 1994). It included on-farm measurements and experimental trials so that epidemiological descriptions could be set-up and subsequently used in the survey.

A series of five experiments of similar design were carried out. A sample of piglets was randomly selected on the day of weaning and divided into paired groups. One group was transferred to the experimental facilities at Ploufragan and one group remained on the farm. All pigs were fed *ad libitum* with the same diet (first phase and second phase diets), and were observed daily for clinical signs indicative of enteric disorders. The experimental facilities were totally isolated rooms. Strict biosecurity measures were taken such as air filtration and showering. Inside the rooms the pigs were kept on flat-decks with perforated floors raised (40 cm) on a concrete solid floor that was thoroughly cleaned every day. Space per pig was 0.38 m^2. Temperature and ventilation were strictly controlled. Climatic and hygienic conditions were considered to be of a high standard. The prevalence of diarrhoea was much higher in the pigs weaned on the farms and the growth rate was considerably reduced compared with pigs weaned into experimental facilities $P<0.05$, (Table 2). None of the pigs transferred to our facilities died whereas an average mortality of 3.3% was recorded on the farms. As the microbial flora could be assumed to be the same in both groups of pigs at weaning, it was concluded that the environmental conditions provided from weaning onwards played a major role in the clinical expression of post-weaning digestive disorders.

Table 2. Growth rate and mortality in paired groups of pigs under different environmental conditions from weaning to 28 days post-weaning.

Farm	Number of pigs		Diarrhoea (% d)[3]		Growth rate (g/d)		Mortality (%)	
	Farm[1]	Exp. F[2]	Farm	Exp. F	Farm	Exp. F	Farm	Exp. F
Farm A	120	30	72	3.5*	326	430*	4.2	0
Farm B	116	28	68	0*	320	445*	2.6	0
Farm C	155	30	75	7*	284	429*	4.5	0
Farm D	142	30	53	0*	346	515*	2.1	0
Farm E	98	30	57	3.5*	351	510*	3.0	0
Mean	126	29.6	65	2.8*	325	466*	3.3	0

[1]Farm: Pigs weaned on their home farm. [2]Exp. F: Pigs transferred to standard experimental facilities at the Veterinary Research Institute. [3]Diarrhoea: the faeces were examined daily, the percentage of days where diarrhoea was detected was calculated. *Significant difference, Farm vs Exp. F ($P \leq 0.05$).

The role of the composition of the first phase diet was also investigated. This diet was prepared in our own feed mill (Table 3) and tested on three farms concerned with post-weaning diarrhoea. The diet, which was given during the 4th week of suckling and then during the first 14 days post-weaning, did not contain any antibiotic. The prevalence of diarrhoea was assessed daily by examining the faecal consistency of each individual pig. A standard scale was used to score faecal consistency (ranging from 1: dry-pelleted faeces to 5: watery). Faeces scored as 4 and 5 were considered diarrhoeic. The contrasting results obtained are presented in Figure 1. Whereas diarrhoea occurred early on Farm 1, it was not apparent during the first fortnight on Farm 3. It was concluded that interactions might exist between diet composition and several other factors present on the farms, which resulted in different patterns of disease expression.

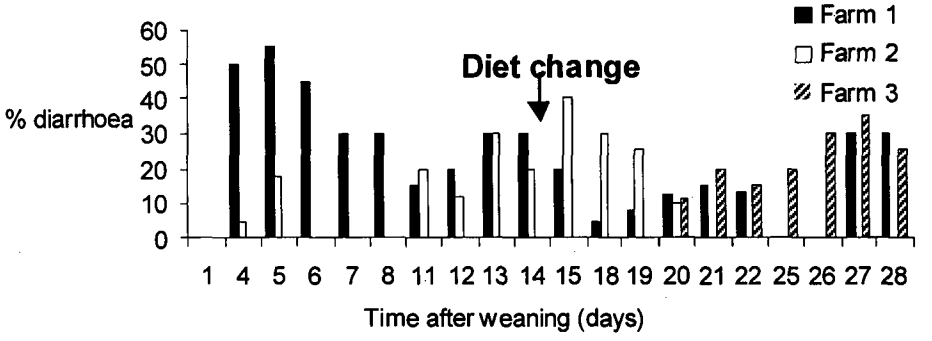

Figure 1. *Daily prevalence of diarrhoea in piglets after weaning from three different farms using the same first-phase diet with ad libitum feeding.*

Table 3. Composition of the first-phase diet.

Raw material composition	%	Analysis	
Heat-processed corn (flakes)	21	Energy (kcal DE/kg)	3965
Heat-processed wheat (flakes)	11	Crude proteins (%)	20.6
Wheat	12	Fat (%)	6.9
Skimmed milk powder	24	Total Lysine (g/kg)	15
Fish meal & fish-derived products	8.9	Cu (mg/kg)	40
Corn-derived feed	5	Zn (mg/kg)	151
Dextrose	5		
Soya bean meal	3.2		
Potato-derived proteins	4.5		
Minerals	3.7		
Lysine + methionine	0.4		
Soya bean oil	1		
Premix	1.3		

The farms

One hundred and six herds were included in the survey (Table 4). The herds were selected on a voluntary basis by veterinarians and technicians. Selection was based on the presence or absence of enteric health problems to obtain sufficient diseased and non-diseased herds for a statistical comparison. The sample of herds enrolled in the survey was retrospectively compared with a broad data base. The average herd size was 146 sows. The weaning age of the piglets was 27.2 ± 1.4 days (mean ± SD) and pigs weaned per sow per year averaged 23.3. Post-weaning facilities housed an average of 130 ± 30

pigs per room with 19.4 ± 9 pigs per pen. Pigs in pens came from an average of seven litters and 83% of rooms had fully slatted floors. Pigs were fed the first phase diet over the final week of the suckling period and during the first two weeks post-weaning. The diet was medicated in 94% of the cases, with colistine used as the base. The zinc content of the diet remained within the recommended level and was in every case below 300 ppm.

Table 4. The data set obtained from the survey.

1 - Data obtained from each piglet (n=5 variables) Dam, litter, live weight at weaning, pen at weaning, survival
2 - Data obtained during suckling phase (litter stage , n=15 variables) . litter size, parity, farrowing disorders in the sow . in the piglets: diarrhoea, locomotor disorders (e.g., arthritis), others . medications . feed distribution to the piglets (weight given), water flow at the nipples . body condition of the sow (farrowing, weaning) . intra-litter weight spread at weaning
3 - Data obtained from weaning onwards, from each pen (n=46 variables) . pen size . pen population: number of piglets, of litters concerned . pen fittings: e.g., type of drinkers, water flow, type of feeders, space per pig . feed intake per week . health status: daily recording (farmer), weekly (veterinarian)
4 - Data obtained at the farm stage (n=213 variables) . size, type, productivity, general health level (standard index) . description of farrowing facilities . description of post-weaning facilities . working order of farrowing facilities: hygiene procedure, air quality . working order of post-weaning facilities: hygiene procedure, air quality, temperature . recordings (daily recordings) . feed composition: starter diet (30 variables), weaner diet (30 variables) . water quality (8 variables) . piglet health, other than digestive, respiratory signs (counts)
5 - Others : only in case of submission of piglets to the laboratory (n=54 variables) standard forms were used for laboratory recordings

The results of the survey

The average live weight of 12,034 piglets at weaning was 8.1 ± 1.7 kg. The mortality rate during the 28 days post-weaning was 1.9%. The average daily gain was 283 g and 489 g for weeks 1+2 and weeks 3+4, respectively. Diarrhoea peaked on days 7-10 post-weaning when 35% of the 616 pens investigated were affected. The specific strategy used to investigate each data set and extract the risk factors involved descriptive multivariate statistical methods, such as correspondence analysis, followed by a logistic regression at the end of the process (Greenacre, 1993; Grange and Lebart, 1993). The risk factors obtained at the end of the multivariate screening phase at the farm level and those obtained at the pen level are listed in Tables 5 and 6. Although it was clearly apparent that several conditions of risk occurred before the piglet was weaned, most of the risk factors occurred from weaning onwards.

Development of herd health programme

Method

The principle was again a follow-up study. Four farms were chosen on a voluntary basis. The size of the herd and the housing system were typical of the area. The

farrowing and post-weaning rooms had fully slatted floors. Although all four farms had been concerned with post-weaning digestive disorders for several months, the clinical picture was different, depending on the farm.

The risk factors listed in Tables 5 and 6 were measured for the first batch of piglets selected on each farm. The profile obtained was then used to plot the farm on a risk factor map (Madec *et al.*, 1998) and to assess the degree of risk present on the farm. The farm situation, with respect to each risk factor, was then discussed with each farmer and decisions made regarding the factors listed in Tables 5 and 6. Emphasis was obviously placed on the most "negative" parameters of the profile. The response of individual farmers varied. On some occasions the farmer was easily convinced of the main points that had to be improved. In other cases greater effort was required to convince farmers as they did not see any direct causal relationship between the risk factor and the disorders. Practical considerations including financial aspects meant that the strategy had to be suitably adapted to each situation. Different trajectories could be simulated by calculating the corresponding coordinates of the farm on the risk factor map. Farmer compliance was sought in every case. A second batch of piglets was then selected and raised in accordance with the decisions made and the 13 risk factors were measured again so that a new profile could be calculated.

Table 5. List of risk factors to post-weaning digestive disorders (PWD; at the farm stage).

	Class order and limits				Sense of increasing risk
Risk factor	1	2	3	4	of PWD
Hygiene status in post-weaning room at the arrival of the piglet[1]	<8	8 – 10	11 – 12	>12	←
Air quality in post-weaning room[1]	<7	7	8	>8	←
Overall farm health disorders (excluding digestive post-weaning)[1]	>16	12 – 16	8 – 11	<8	←
Creep feed intake/piglet, in week prior to weaning (g)	<190	190 – 300	300 – 470	>470	←
Available manpower (sows/person)	>110	80 – 110	< 80	/	←
Feed intake/piglet during 1st week post-weaning (kg)	<1	1.0 - 1.36	1.37 – 1.72	>1.72	←
Level of concurrent respiratory disorders in the weaned piglet[1]	>5	4 – 5	< 4	/	←
Age at weaning (days)	<26.5	26.5 – 28.2	> 28.2	/	←

[1]These are scores combining up to 10 variables, see Appendix 1 to this paper.

Results

The severity of post-weaning digestive disorders at the first check is given for all 4 farms in Table 7. Diarrhoea and low growth rate were the main symptoms on Farms I and II. Diarrhoea was particularly severe on Farm II where it was recorded on 23 of the 28 days of observation. Mortality was critical (14%) on Farm IV. It had been 12% on average over the six months before the trial.

The real technical task started at the end of the first check. The changes that occurred in the profiles are shown in Table 8. The 1st and 2nd checks were performed 1 to 2 months apart. A special effort was made on Farm I to improve the housing conditions offered to the piglet at their arrival in the post-weaning rooms. These rooms, including the pit beneath the slatted floor, were thoroughly cleaned. The temperature was raised to

24°C when the piglets entered (instead of 15°C previously). The ventilation system was rectified to prevent noxious gases from rising up from the pit. The ammonia level remained below 10 ppm and the relative humidity within the range of 60-80%. Creep feed intake was increased by changing the feeders and the feeding procedure. The number of pigs per pen at weaning and the stocking density were considerably reduced. The feeding system in the post-weaning rooms was also changed and the post-weaning feed intake was increased, probably as a result of all these modifications. The focus on Farm II was on stocking rate, space at the feeder and creep feed intake. Although the space per pig in pens and at feeders was increased, the hygiene level on this farm could not be modified to a satisfactory level. The Farm III profile was close to that of Farm 1, except for weaning weight, which was lower. In this case the emphasis was placed on hygiene, climate and creep feeding procedure. On Farm IV where the piglets were weaned earlier, special attention was paid to climatic conditions, which were initially poor, and to hygiene.

Table 6. List of risk factors to post-weaning digestive disorders (PWD; at the pen stage).

Risk factor	Class order and limits				Sense of increasing risk of PWD
	1	2	3	4	
➢ Average weaning weight of the piglets in the pens (kg)	<7.2	7.2-8.1	8.1-9.0	>9.0	←
➢ Number of piglets/pen	<13	13-16	17-23	>23	→
➢ Number of litters involved/pen	<4	4-6	7-9	>9.0	→
➢ Space (cm) at the feeder/pig at weaning	<4.5	4.5-5.7	5.8-8.4	>8.4	←
➢ Floor surface/pig at weaning (m^2)	<0.25	0.25-0.30	0.31-0.37	>0.37	←

The health level of the piglets in the second check is shown in Table 7. A considerable improvement was observed in the herds. A third check was made on the farms to verify these changes (data not presented). The results were very similar to those obtained at the second check.

Table 7. Severity of post-weaning disorders on the farms at the 1st and 2nd checks (i.e., before and after implementation of a health programme).

Farm check	Farm I		Farm II		Farm III		Farm IV	
	Ia[1]	Ib[1]	IIa	IIb	IIIa	IIIb	IVa	IVb
Number of piglets involved	118	132	153	128	122	97	115	117
Daily gain (g, D0[2] – D28 PW[3])	314	452	323	345	351	376	299	354
Diarrhoea score[4] (D0–D28 PW)	10.5	1.4	23	12	6.8	0.8	0.9	0
% mortality (due to PWD)[5]	0.8	0	4	0	0.8	0.8	14	1.8

[1]Ia - first check on Farm I; Ib - second check. [2]D0, day of weaning. [3]PW, 28 days post-weaning. [4]Diarrhoea score: 0=absence; >0=presence. [5]PWD, post-weaning digestive disorders; scoring/pen/day; average calculation for the pens.

Discussion

While there is no doubt that toxigenic *E. coli* is involved in the pathogenic process of post-weaning digestive disorders (Beers-Schreurs *et al.*, 1992, Imberecht *et al.*, 1994), these pathogens can also be found in healthy piglets raised on farms with no history of post-weaning disorders (Fairbrother *et al.*, 1994). Hence it can be concluded that additional conditions are required for the onset of clinical signs of disease (Hampson and Kidder, 1986). The conditions on many farms are such that harmful profiles combining different risk factors can easily occur (Skirrow *et al.*, 1997). Therefore prevention should be

primarily directed towards a provision of zootechnical profiles that reduce the risk of the disease. A logical explanation for the disappointment felt by the farmers, veterinarians and other professionals involved in health maintenance lies in the multifactorial character of the disorders observed during the post-weaning phase. First, the use of drugs as a sole measure supposes the primary-causal role of bacteria in this process. There is clear evidence that this is not the case as regards post-weaning digestive disorders. Although *E. coli* bacteria are a necessary contributing factor to digestive disorders, they are not the initiating agents. Housing and management conditions before and, above all, just after weaning, are important in preventing toxigenic strains of *E. coli* from proliferating in the gut, and producing their toxins beyond the limit required to cause clinical disease. Secondly, as demonstrated on affected farms (Table 8), numerous risk factors associated with clinical disease may be present. In such situations, the correction of a single factor cannot result in a perceivable health improvement. Unfortunately, the usual approach is to work on a single parameter at a time and then look at the consequences of its modification. As no visible positive impact on health is obtained, such action is rapidly abandoned and another direction taken with a similarly predictable frustrating result. When confronted with multifactorial enzootic diseases in veterinary practice, it is wise to construct a programme that aims to change more than one factor, and that can be followed step by step. The aim at this stage is not to evaluate the impact of a single isolated factor, which is the role of experimentation. It has to be kept in mind that the animal's response to any change in one factor will strongly depend on the values of the other factors. Such heterogeneity probably explains the discrepancies of the results obtained in experiments, which in turn add to the confusion.

Table 8. Farm profiles on the risk factors.

Farm	I		II		III		IV	
	Ia[4]	Ib[4]	IIa	IIb	IIIa	IIIb	IVa	IVb
Number of piglets	118	132	153	128	122	97	115	117
Hygiene score[1] (PW room[2])	6	13	5	8	8	14	11	14
Climate adequacy[1] (PW room)	6	8	6	8	6	9	4	9
Herd health level[1]	9	9	14	14	13	13	14	14
Available man power (sows/man)	116	116	83	83	83	83	68	68
Creep feed intake (g, week BW[3])	150	192	281	365	56	234	180	285
Feed intake 1st week PW (kg)	0.64	1.33	1.2	1.23	0.67	1.17	1	1.32
Respiratory clinical signs [(1)], PW	1	3	2	3	1	2	3	2
Live weight at weaning (kg)	8.8	9.3	8.9	9.1	7.1	8.8	6.2	6
Pigs/pen (PW room)	39	25	22.7	12	15	15	12	11.5
Litters concerned/pen (PW room)	10.3	8.6	9.3	7.2	2.3	3.1	7.2	8.3
Trough space/pig (PW room, cm)	5	8	4.6	8.3	8	8	6.7	6.9
Space/pig (PW room, m^2)	0.22	0.33	0.19	0.37	0.33	0.37	0.35	0.35

[1]These were scores summarizing up to 10 initial variables (Table 6). [2]Post-weaning room. [3]Before weaning. [4]Ia: farm I, 1st check; Ib: farm I, 2nd check; IIa: farm II, 1st check ...etc...).

Obviously the relationships between environment and health are not limited to post-weaning digestive problems. Several papers have been published over the years about the role of housing and internal climate on respiratory tract damage. In Scandinavian countries these aspects received special study during the seventies (Linqvist, 1974; Aalund *et al.*, 1976). Several other surveys followed in different countries and a major association between farm management and the immediate environment on respiratory disorders in the fattening pig was established (Tielen *et al.*, 1978; Backstrom and Bremer, 1978; Pointon *et al.*, 1985; Madec and Tillon, 1986; Done, 1991; Hurnik *et al.*, 1994).

An investigation in which piglets were housed either on their home farm, or transferred to improved accommodation in a schedule similar to the one described in the present paper, showed that housing had an effect on performance and to a lesser extent on health (Straw, 1991). It is worth noting that whereas most of the studies of epidemiological type conclude in a significant impact of environment and hygiene on

health, narrowly designed experiments targeted on a single factor can have difficulty in demonstrating the role of a single factor in the pathogenic process. Concentrations as high as 100-150 ppm of ammonia applied continuously for 4 weeks were needed to produce an inflammatory reaction in the broncho-tracheal epithelium of pigs (Drummond *et al.*, 1980). Similarly, diarrhoea was produced in weaned piglets inoculated with *E. coli* when they were subjected to cold stress (Wathes *et al.*, 1989).

The difficulties involved in solving enzootic disease problems stem not only from the fact that several factors are involved, but also because they interact and intervene in series or cascades. When the signs of illness actually appear, several events will have taken place upstream, most of them remaining undetected. For most enzootic multifactorial diseases in which a microbial agent is involved, disease severity seems to depend on the size of the pathogen population (microbial pressure). Methods of management and husbandry, which aim to reduce this pressure, should therefore result in improved health. Although it is common practice to use combinations of antimicrobials to control enzootic diseases, especially respiratory diseases (Hewett, 1993), the use of antibiotics as a means of reducing microbial pressure in a prevention scheme is questionable. On the other hand, the conclusions of numerous reports indicate that under adequate environmental conditions, and sometimes with the help of vaccination in the case of a well identified pathogen, the impact of enzootic diseases can be reduced. It is unfortunate that only some of the factors and conditions that have an effect on health are precisely known at present. Knowledge of others needs to be updated and several factors remain to be discovered. Collaboration between scientific disciplines is essential if these factors and conditions are to be identified and hence a broad field is opening for applied epidemiology. Not only can the level of the clinical signs and damage to the tissues be considerably reduced by providing pigs with adequate conditions, but there is also growing evidence of the possibility of eliminating infectious agents through specific techniques even without drug usage. Segregated early weaning is one of them (Dritz *et al.*, 1996). Building design is also important with respect to hygiene (Boon and Wray, 1989). On the other hand, strict hygiene associated with strategic movement of the pigs has also been successful in the elimination of *Salmonella typhimurium* (Dahl *et al.*, 1997). While the flexibility of using antibiotics in animal therapy must be safeguarded, the emphasis should be placed on prevention through better care of the animals, while keeping production costs acceptable. Just like infectious agents, certain management and husbandry conditions encountered on farms can be considered as pathogenic in the sense of "generating pain". The first step is to gain an understanding of the precise risk factors involved in the different health problems encountered. Both experiments and field surveys are needed to provide a correct insight into the problem. Then, and only then, can relevant herd health programmes and health monitoring schedules be set up.

Appendix 1: Scales used for scoring four variables on 106 farms (Madec *et al.*, 1998).

1 - Scale used to assess hygiene status on arrival of piglets in the weaning facilities

1. Days between cleaning (after previous batch) and the arrival of the piglets followed for the survey. Score=0 (< 2 days), 1 (2-4 days), 2 (5-7 days), 3 (> 7 days)
2. Disinfection: Score=0 (no disinfection), 2 (yes)
3. Dryness of the floor at the arrival of piglets: Score=0 (humid, more or less), 2 (dry)
4. Cleanliness of the floor and of the walls between pens: Score=0 (dirty), 1 (± clean), 2 (clean)
5. Distance between the level of the slurry (in the pit below the floor) and the floor surface: Score=0 (< 40 cm), 1 (40-70 cm), 2 (70-90 cm), 3 (>90 or empty)
6. Hygiene of the pit (after removal of the previous batch of piglets): Score=0 (no emptying + no washing + no disinfection), 1 (emptying + no washing + no disinfection), 2 (emptying + washing ± no disinfection)
7. Ambient temperature at the arrival of the piglets: Score=0 (<18°C), 1 (18-20), 2 (21-24), 3 (>24)

Index value=Total of scores obtained

2 - Scale used to assess overall farm pig health status for 12 months prior to survey

1. Influenza-like syndromes (clinical episodes): Score=0 (absence), 1 (one episode), 2 (two episodes)
2. PRRS infection (serology, Porcine Respiratory and Reproductive Syndrome): Score=0 (negative), 2 (positive)
3. Sow mortality (%, one-year period): Score=1 (<1 %), 2 (1.5 – 2.5 %), 3 (2.5 - 5 %), 4 (>5 %)
4. Mortality during fattening phase (30-105 kg live weight, one-year period): Score=1 (<1.5 %), 2 (1.5 – 2.5 %), 3 (2.5 – 3.5 %), 4 (>3.5 %)
5. Mortality during post-weaning phase (from weaning to 30 kg live weight, one-year period): Score=1 (<1.5 %), 2 (1.5 - 3 %), 3 (>3 %)
6. Outbreak of neonatal diarrhoea: Score=0, (no), 1 (yes)
7. Outbreak of non haemorrhagic diarrhoea in fatteners: Score=0, (no), 1 (yes)
8. Haemorrhagic syndromes in fattening pigs: Score=0, (no), 1 (yes)

Index value=Total of scores obtained

3 - The scale used to assess ventilation quality within the post-weaning rooms

Agreement of the air flow inside the room to the building design
- air inlet: Score=0 (no agreement), 1 (yes)
- air outlet: Score=0 (no agreement), 1 (yes)

Air presently flowing out of the slatted floor : Score=0 (yes), 1 (no)
Turbulence of the flow : Score=0 (yes), 1 (no)
Falling down of cold air onto the lying area : Score=0 (yes), 1 (no)
Average air speed on the lying area : Score=0 (≥0.4 m / sec), 1 (<0.4 m / sec)
Draughts : Score=0 (yes), 1 (no)
Level of NH_3 : Score=0 (≥10 ppm), 1 (<10 ppm)
Level of CO_2 : Score=0 (≥0.15 %), 1 (<0.15 %)
Relative humidity : Score=0 (≥85 %), 1 (<85 %)

Index value=Total of scores obtained

4 - Scale used to score clinical signs of respiratory disorders in the piglets

Coughing				
Average coughs counted during observation*	0	0.1-5	>5	
Score	1	2	3	
Sneezing				
Average sneezes counted during observation*	<2	2-4	4.1-8	>8
Score	1	2	3	4

*Counts over 2 minutes duration each, n=3 counts on days 7 and 21 post-weaning. The figures are given for 100 pigs. Example: a farm obtaining an average of 4.6 counts fell into level 2 for coughing.

The whole batch of piglets was counted.

Symposium continued on next page

CAN DIET BE USED AS AN ALTERNATIVE TO ANTIBIOTICS TO HELP CONTROL ENTERIC BACTERIAL INFECTIONS OF PIGS?

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Abstract

In response to a need to develop alternative methods to control the major enteric bacterial infections of pigs, studies have been undertaken to investigate whether or not it is possible to reduce susceptibility to colonisation by pathogens through the use of specific diets. These diets are intended to alter the intestinal environment, including the resident microflora, such that conditions are no longer conducive to growth of the pathogens. In the case of swine dysentery, caused by the spirochaete *Brachyspira (Serpulina) hyodysenteriae*, it has been shown that diets with very low levels of soluble non-starch polysaccharides and resistant starch offer complete protection against experimental disease. The only fully protective diet identified to date comprises cooked white rice and animal protein, but substituting the rice with steam-flaked maize or sorghum, or finely ground sorghum, gives a diet that tends to reduce susceptibility to experimental infection. To date the application of exogenous enzymes to standard pig diets has not produced intestinal conditions that inhibit colonisation by the spirochaete. In the case of the related intestinal spirochaete *Brachyspira (Serpulina) pilosicoli*, the aetiological agent of porcine intestinal spirochaetosis, feeding the rice-based diet retards the rate of colonisation of the large intestine but does not completely prevent either colonisation or the development of disease in pigs after experimental challenge. The same rice-based diet reduces colonisation of the small intestine by enterotoxigenic *Escherichia coli* after experimental infection of weaners, whilst addition of a source of soluble non-starch polysaccharide to the rice diet results in greater colonisation by the *E. coli* strain. Finally, the stomachs of weaner pigs fed a finely ground wheat-based diet have been shown both to have severe ulceration of the *pars oesophagea* and to be colonised by *Helicobacter* spp. Feeding finely ground extruded wheat resulted in no ulceration and an absence of the bacteria. It is possible that the *Helicobacter* spp. are involved in the aetiology of the ulceration, and that their presence is influenced by the treatment of the diet consumed.

From these studies it is clear that a number of important enteric bacterial infections can be modified by the use of specific diets. The main challenges are to understand how these effects work and to develop cost-effective diets that can be applied in the field to help supplement current control measures for these and other enteric infections of pigs.

Introduction

Antibiotics are an important management tool for the pig industry. They are used to treat overt bacterial diseases, to provide a level of prophylaxis in situations where disease is liable to occur, and, to a lesser extent, to improve growth rates in the absence of disease. Unfortunately the pig industry faces mounting difficulties over its use of antimicrobials. The reason for this is that their long term use eventually selects for the survival of resistant bacterial species or strains, and furthermore there are a variety of mechanisms whereby the genes encoding this resistance can be transferred to other bacteria, thus also making them resistant. This scenario has resulted in a situation where a variety of important bacterial pathogens of pigs are showing resistance to a range of antimicrobial drugs. On an immediate practical level this drug resistance is reducing the number of available antimicrobials that can be reliably used to control diseases caused by these bacteria in pigs. Although this is an obvious problem, it is unlikely to induce legislative intervention. In contrast the potential risks to human health that may arise are leading to mandatory changes being imposed on the industry. These risks include the transfer of multidrug resistant zoonotic pathogens such as *Salmonella* spp. and

Campylobacter spp. from pigs to humans, the direct or indirect transfer of resistance genes from members of the porcine intestinal microflora to human bacterial strains, and the presence of antimicrobial drug residues in pig meat. Public concern about these issues is being translated into reduced meat consumption, tightening of restrictions on antimicrobial residues in meat, and legislation reducing the availability of antimicrobial agents for use in animal production. Consequently there is a clear and urgent need to investigate alternative means both of controlling bacterial infections and promoting growth in pigs.

There are a number of common and important enteric bacterial infections of pigs, and much of the antimicrobial usage in the pig industry is directed at controlling these infections. Clearly alternative methods of control that do not require antimicrobials would greatly reduce the need to use these drugs. Accordingly efforts are being directed at modifying management practices so as to limit exposure to pathogens and to minimise stress, at improving vaccines for enteric infections, at selective breeding of animals for resistance to infectious diseases, at improving the pig's local immune responses, and at strategies to inhibit or kill pathogenic bacteria in the gut by the use of organic acids, inorganic chemicals such as zinc oxide, bacteriophages or bacteriocins. A slightly different approach being developed focuses on excluding the growth of pathogens in the gut by encouraging the growth of other bacteria which are believed to take up microniches in the intestine and compete with the pathogens at those sites. The idea of "competitive exclusion" of *Salmonella* serovars by components of the normal intestinal microflora has long been accepted (Nurmi and Rantala, 1973), and the principle has recently been extended to *Yersinia* spp. infecting pigs (Asplund *et al.*, 1996). One form of this approach is to feed specialised strains of certain bacteria, especially *Lactobacillus* spp. and *Bifidobacterium* spp., that are selected because they are considered to promote gut health and exclude pathogens. The use of these so-called probiotic bacteria is reviewed elsewhere in this volume. Probiotics probably have most promise for use in controlling infections in young pigs, for example in the period immediately after weaning, when the resident intestinal microflora is not yet stable enough to exclude probiotic strains.

An extension of the probiotic approach to control of enteric infections has been to feed specific dietary components that act as substrate for natural populations of "protective" bacteria, such that these proliferate and more effectively exclude pathogens. For example, in the case of *Clostridium difficile* infection, different dietary fibre sources have been investigated to optimise inhibition by the resident microbial flora through its production of specific short chain fatty acids (May *et al.*, 1994). Similarly so-called "prebiotic" dietary supplements such as fructose-containing oligosaccharides have been used to selectively increase numbers of *Bifidobacterium* spp. in the large intestine, the presence of which in turn results in an inhibition of colonisation by certain pathogens (Gibson *et al.*, 1995). Recently specific metabolites from plants have been identified which when fed may interact with short chain fatty acids to create inhibitory conditions for pathogens such as *E. coli* O157 (Duncan *et al.*, 1998).

The resident intestinal microflora of the pig is extremely diverse and complex, and varies quantitatively and qualitatively at different intestinal sites and at different stages in the life of the pig. The main bacterial biomass is found in the large intestine, where dietary fibre serves as the major bacterial growth substrate. Dietary fibre is defined as plant materials that are not digested in the small intestine, and can be broadly divided into material that is fermented rapidly and material this is fermented slowly. There have been relatively few detailed studies on the intestinal microflora of pigs (e.g., Robinson *et al.*, 1981, 1984), and because of a lack of appropriate culture techniques suitable for many of the fastidious anaerobic microorganisms that inhabit the large intestine, the exact composition of the intestinal microflora of the pig is not known. There is a body of literature to show that different forms of fibre in the diet can broadly influence the composition and metabolic activity of the large intestinal microflora in pigs (Varel *et al.*, 1982; Varel and Pond, 1985; Bach Knudsen *et al.*, 1991; Jensen and Jorgensen, 1994; Reid and Hillman, 1999). Unfortunately little is known about how the different groups of resident bacteria which are stimulated to proliferate in this way themselves interact with pathogenic species of bacteria. This lack of information makes it difficult to predict how a given dietary component could be used to indirectly influence a given enteric pathogen.

It should also be borne in mind that the diet could have other influences on colonisation by pathogens, for example by modulating the amount of specific substrate available at a given site for the pathogen itself, by altering intestinal viscosity and hence intestinal motility, and by direct or indirect effects on the intestinal mucosa such that changes occur in specific colonisation sites or receptors on enterocytes. For example, different cereal types and particle size have been shown to alter epithelial cell proliferation and lectin binding patterns of the epithelium in the large intestine of pigs (Brunsgaard, 1998).

Despite a lack of detailed knowledge, the general contention that dietary components can in some way influence colonisation by pathogens is consistent with reports from the field, where it is often observed that changes to the diet result in either increased or decreased enteric disease. For example, it has been reported that units adopting liquid feeding of by-products or using fermented wet feed have a lower incidence of Salmonellosis than herds using dry feed (Stege *et al.*, 1997; van der Wolf *et al.*, 1999). Information from such field studies, although obviously important because it reflects conditions in the field, does need to be confirmed by careful experiments conducted under controlled conditions. This is because other issues apart from specific dietary components may be contributing to the effects. In the case of Salmonellosis the relative hygiene of the various diets may be influencing the infectious dose presented to the pigs.

In view of the effects that diet can have on the intestinal environment, including its resident microflora, and because of the need to develop new means for control of specific enteric bacterial pathogens, attempts have been made to select or manipulate pig diets under experimental conditions so as to help control some of these important infections. The approach assumes that the diet to be used has the correct optimal balance of macro- and micro-nutrients to support normal pig growth, and does not contain any toxic components (e.g., mycotoxins) which may increase susceptibility to pathogenic bacteria.

It should be borne in mind that many different sorts of pathogenic bacteria infect pigs, and these have different physiological requirements for colonisation and growth. Different pathogens also colonise different sites within the gastrointestinal tract, and the environmental conditions generated at these sites in response to the diet vary enormously. Given these complexities it would be optimistic to seek one simple dietary solution that could be used for the control of all enteric infections at different stages of a pig's life.

This review focuses on four important enteric bacterial diseases of pigs: swine dysentery, porcine intestinal spirochaetosis, post-weaning colibacillosis and ulceration of the *pars oesophagea* of the stomach. These conditions involve different sorts of bacteria that colonise different areas of the intestinal tract. These diseases have been selected for study both for their economic importance to the pig industry and because they illustrate some of the general principles involved in using diet to help control enteric bacterial infections.

Swine dysentery

Swine dysentery (SD) remains a major recurrent problem for the pig industry in many parts of the world. The disease is caused by infection with the anaerobic intestinal spirochaete *Brachyspira (Serpulina) hyodysenteriae,* which colonises the crypts of the large intestine and induces a severe mucohaemorrhagic colitis and dysentery (Hampson *et al.*, 1997; Ochiai *et al.*, 1997). Experimental infection studies in gnotobiotic pigs have shown that colonisation by the spirochaete and lesion formation are enhanced by the presence of other species of anaerobic bacteria, particularly certain *Bacteroides* spp. and *Fusobacterium* spp. (Meyer *et al.*, 1975; Whipp *et al.*, 1979). Vaccines developed to prevent the infection have been relatively ineffective, and control of SD on infected piggeries mainly is achieved through the prophylactic and/or therapeutic use of antimicrobials, or by eradication through depopulation and/or medication (Hampson *et al.*, 1997).

According to Harris and Lysons (1992), in the era before antimicrobials were developed, pigs with SD were treated by feeding them oats soaked in salt water or sodium hydroxide solution, with the assumption that the high fibre content of the diet was beneficial. Prohaszka and Lukacs (1984) reported a field study where the diet of pigs with SD was changed from one based on corn to one based on corn silage. This

change lowered the pH of the digesta in the large intestine, and resulted in an inhibition of growth of *B. hyodysenteriae* and protection against disease. These field observations led Siba *et al.* (1996) to test the hypothesis that diets rich in highly fermentable fibre would generate an acidic environment in the large intestine which would protect from colonisation by *B. hyodysenteriae*. Groups of weaner pigs were fed either a typical Australian wheat/lupin diet that contained rapidly fermentable fibre sources, or an experimental diet composed of cooked white rice and animal protein that contained low levels of dietary fibre. Judging from the pH of the digesta in the large intestine, the cooked rice diet apparently reduced the extent and rate of bacterial fermentation in the large intestine compared to the standard diet. Contrary to expectations nearly all the pigs fed wheat/lupin developed SD after experimental challenge, whilst none of the pigs fed the rice diet were colonised by the spirochaete or developed SD. When either the cooked rice or the animal protein were mixed with either lupin or wheat, disease occurred after challenge (Table 9). This indicated that neither the rice nor the animal protein contained specific components that inhibited growth of the spirochaete. The authors considered that the protective effects of the rice diet were most likely due to reduced fermentation in the large intestine, and associated suppression of members of the resident microflora which normally facilitate colonisation by the spirochaete. The authors did not exclude the possibility that the protection was associated with other physical changes in the large intestine associated with reduced fermentation (e.g., drier digesta). In a subsequent study of pigs fed the "protective" rice-based diet, an analysis of the bacterial flora in the large intestines showed some evidence for there being reductions in populations of bacteria which have been reported to act synergistically with *B. hyodysenteriae* - notably *Fusobacterium necrophorum* and *F. nucleatum* (Durmic *et al.*, 1998b).

Subsequent work showed that parboiled rice was not a suitable substitute for cooked rice, and that it was important for the rice to be fully cooked with a ratio of two volumes of water per volume of rice in order for fermentation in the large intestine to be minimised (Siba, 1997). Recently, workers in Canada (R.N. Kirkwood, personal communication) and Denmark (R.H. Lindecrona, personal communication) have reported difficulty in reproducing the protective effect of the rice diet. This apparent failure to obtain protection may be associated with the way the rice was processed, or alternatively it may be a reflection of differences in the intestinal microflora of pigs at the study sites. If the latter is the case it adds a further dimension of complexity to the use of diet to control SD.

Table 9. Pooled results displaying shedding of spirochaetes and the incidence of swine dysentery in pigs fed different diets following oral challenge with *B. hyodysenteriae* (after Siba *et al.*, 1996).

	Diet Type[1]			
Item	RA	RL	WA	WL
No. of pigs challenged	16	6	6	16
No. of pigs shedding *S. hyodysenteriae* in faeces	3	6	5	13
Mean duration (days) of shedding in faeces	4.6	5.4	5.6	8.5
No. of pigs that developed SD	0	5	3	10
Incidence of disease, %	0	83.3	60	62.5

[1]Diets: RA: 77% cooked white rice + 18% animal protein; RL: 64% cooked white rice + 13% animal protein + 15% dehulled Australian sweet lupins; WA: 75% wheat + 17% animal protein; WL: 62% wheat + 11.5% animal protein + 15% dehulled Australian sweet lupins.

In most parts of the world it is neither practical nor economically viable to feed pigs cooked rice, except for example for a short period during an attempt to eradicate SD from

a piggery. A study therefore was undertaken to examine a variety of different cereal grains either heat-processed or not, in order to identify practical alternatives to feeding rice, and to help identify components of the diet which might predispose to colonisation by the spirochaete (Pluske *et al.*, 1996). Two cereals, maize and sorghum, were identified which when steam-flaked reduced the incidence of disease amongst groups of pigs after experimental infection with *B. hyodysenteriae*. An analysis of all the diets tested identified soluble non-starch polysaccharide (sNSP) and resistant starch (RS) as being two important dietary components that promoted fermentation in the large intestine and were associated with a high incidence of SD.

The importance of sNSP and RS in generating conditions that allowed the spirochaetes to proliferate were then investigated by adding them in pure form either by themselves, or together, to the original protective cooked rice diet (Pluske *et al.*, 1998). Retrograde maize starch was used as a source of RS, and guar gum was used as a source of sNSP. Pigs fed these supplemented diets became colonised and developed dysentery, whilst those on the base rice diet did not. Consistent with the hypothesis that rapid fermentation associated with sNSP and/or RS is required to facilitate colonisation and disease, when a source of predominantly insoluble NSP (oaten chaff) was added to the protective rice diet, this diet remained protective. Based on measured fermentation parameters (pH of digesta, gut weight and VFA and ATP content) the chaff was poorly fermented such that the correct predisposing conditions for colonisation by *B. hyodysenteriae*, for example VFA concentrations and balance of microflora, were absent.

As wheat remains an important cereal grain for feeding pigs in Australia, attempts have been made to increase the digestibility of wheat in the small intestine by the addition of exogenous enzymes and/or cereal grain extrusion (Durmic *et al.*, 1997, 1998a). Dietary enzymes used with wheat included xylanase to reduce the viscous effects of sNSP, and various mixtures of amylase and/or protease to help increase digestion of starch in the small intestine. Extrusion was used to assure complete starch gelatinisation and near complete digestion of starch in the small intestine. These manipulations failed to reduce the fermentability of the diet such that it prevented colonisation by *B. hyodysenteriae*. In this case addition of enzyme actually resulted in an increased rate of fermentation in the caecum, with reduced fermentation only being recorded in the distal part of the colon. The increased caecal fermentation presumably was a result of liberation of smaller highly fermentable oligosaccharides from the NSP in the wheat. Extrusion of the wheat also failed to significantly reduce the expression of SD with a 57 ± 7 % (mean ± sem) expression in pigs fed either extruded or hammer-milled raw diets (8 mm screen) formulated with wheat and animal protein. This high incidence of disease occurred despite the presence of reduced indices of fermentation in the large intestine of pigs fed extruded diets. One possible explanation may be that the relatively high temperatures associated with extrusion may have strengthened bonds within the starch granules, making them less susceptible to enzyme action. Alternatively, heating may have decreased the ratio of insoluble NSP : soluble NSP (Pluske *et al.*, 1996) that exacerbated expression of the disease.

The effects of grain processing and addition of dietary enzymes to sorghum-based diets currently are being investigated in our laboratory. Sorghum grain has a very low sNSP content but a relatively high starch content, so treatments aimed simply at increasing starch digestion in the small intestine might cause reduced fermentation in the large intestine and result in a reduced expression of SD. In preliminary experiments the inclusion of dietary enzymes based on xylanase, amylase and protease have had no effect on fermentation parameters in the large intestine nor on the expression of SD in infected pigs. Extrusion of sorghum actually increased the expression of SD ($P<0.05$) with 59 ± 8 vs 19 ± 2% of pigs (n=24) expressing the disease when fed diets based on extruded versus raw sorghum (with animal protein as the protein source). For these experiments the sorghum grain was finely milled through a 1.2 mm screen using an air assisted hammer-mill compared to an 8 mm screen in a traditional hammer-mill. Further analysis comparing different experiments showed a response to fine grinding ($P<0.05$), with 63 ± 8 vs 35 ± 8% of infected pigs (n=54) expressing SD when fed coarse versus finely ground wheat or sorghum. The basis of this response is still to be investigated, but the known improvement to digestibility in the small intestine of finely ground diets (Wandra *et al.*,

1995; Mavromichalis and Hancock, 1999) should result in the reduced availability of substrate for fermentation in the large intestine. Certainly in these experiments, various fermentation indices (weight of the large intestine, pH and ATP, VFA and starch content of the digesta) suggested that finely ground sorghum resulted in rates of fermentation as low as those found with extruded sorghum.

Collectively, how do these data improve understanding of the relationships between diet, fermentation and SD? Simple linear regression analyses were conducted between dietary characteristics and indices of fermentation recorded in the large intestine and the percentage of pigs showing clinical expression of the disease (Table 10). This was based upon data from 204 pigs, 102 of which were slaughtered for measurement of fermentative indices after being fed different diets or dietary treatments (described above), and 102 of which were identically fed, but then inoculated with a virulent strain of *B. hyodysenteriae* and monitored for SD. A multiple regression model was also developed.

The percentage of variation in SD in the infected pigs explained by individual indices of fermentation in the uninfected pigs was generally low (0.3 to 18%) (Table 10). However, a multiple regression model incorporating these individual indices of fermentation explained 51% ($P<0.001$) of the total variation in the percentage of experimentally-challenged pigs that developed SD, as follows: -

Percentage of experimentally-challenged pigs that developed SD=151.4 + 7.1 (sNSP in diet, %) + 39.3 (weight of full caecum, % body weight) – 21.7 (pH caecum) + 3.8 (ATP in proximal colon, nmol/g digesta) – 0.14 (total VFA in large intestine, mmol); $r^2=0.51$. NB Indices of fermentation were derived from the uninfected pigs.

Table 10. Individual correlations between diet characteristics and fermentative indices in the large intestine, the multiple regression model, and the percentage of experimentally-challenged pigs that developed SD.

	Individual correlation with % disease expression		Multilinear regression	
Model component	Correlation (r^2)	P value	% contribution to final r^2	P value for model
Dietary sNSP	0.17	0.001	42	<0.001
Full caecum	0.18	<0.001	17	<0.001
pH caecum	0.16	<0.001	11	0.001
ATP proximal colon	0.04	0.04	5	0.029
Total [VFA] in large intestine	0.003	NS	25	<0.001

The resultant model is heavily driven by the sNSP level of the diet (Table 10). Given that dietary interventions designed to counter some of the effects of sNSP were not effective in protecting against SD, the model clearly points to the selection of grains (diets) inherently low in sNSP to achieve protection against the disease. The other components of the model mainly relate to fermentation indices in the caecum and proximal colon (Table 10). It would appear, therefore, that controlling or reducing fermentation in the first part of the large intestine is critical for reducing the expression of SD. Reducing fermentation in this part of the large intestine is difficult, however, since even small amounts of undigested material passing from the terminal ileum will be available for fermentation.

Although a cheap and easy means of treating all common cereal grains to make them "protective" against SD has not been identified, some progress has been made, and sorghum and maize have been identified as being the best grains on which to concentrate investigation. Further work will be needed to verify the effects on large intestinal fermentation and disease of grind size with these and wheat-based diets. The cooked rice diet stands out in that it can completely protect infected pigs from expressing SD. In addition it clearly results in the lowest rates of fermentation in the large intestine when compared to all other diets that have been examined so far. Although expensive, it could

be used prophylactically in outbreaks of SD, or to reduce shedding of *B. hyodysenteriae* prior to medication and/or destocking, as part of a SD eradication programme.

Porcine intestinal spirochaetosis

Porcine intestinal spirochaetosis (PIS) is a chronic diarrhoeal disease of weaner and grower/finisher pigs, resulting from colonisation by the anaerobic intestinal spirochaete *Brachyspira (Serpulina) pilosicoli* (Trott *et al.*, 1996; Ochiai *et al.*, 1997). As with the closely-related *B. hyodysenteriae, B. pilosicoli* colonises the caecum and colon, but unlike *B. hyodysenteriae,* which is chemotactic to mucus and moves deep into the crypts, *B. pilosicoli* remains largely in the lumen of the intestine, or may attach by one cell end to the epithelium adjacent to the intestinal lumen (Hampson and Trott, 1995).

For some time before the description of PIS and its association with *B. pilosicoli,* certain cases of what was almost certainly PIS were described as "grower scour/non-specific colitis" (Smith and Nelson, 1987). This condition was reported to be influenced by diet, with pelleting of the diet being said to predispose to the condition (Connor, 1992). Finely ground pelleted food is also believed to predispose to Salmonellosis (Schwartz, 1999), and ulceration of the *pars oesophagea* (Friendship, 1999).

In view of the close similarity between *B. pilosicoli* and *B. hyodysenteriae,* their very similar habitats in the large intestine, and reports from the field of dietary influences on PIS, an investigation was made into whether the cooked rice diet that protects from SD might also protect from PIS (Hampson *et al.*, 1998). In this study two groups of weaner pigs were fed either a standard commercial wheat-lupin weaner diet (n=8), or the rice-based diet described above (n=6) for three weeks after weaning. All pigs were then challenged orally over three days with 10^{10} active mid-log phase cells of a Western Australian field strain of *B. pilosicoli* (strain 95/1000). The pigs were killed 3-4 weeks post inoculation (pi).

All animals became colonised with *B. pilosicoli* strain 95/1000, but this occurred significantly later (mean of 14 days pi compared to 3.6 days), and lasted for significantly less time (mean of 13 days compared to 20 days), in the pigs fed rice compared to those fed wheat. One pig fed the wheat diet developed an acute and severe erosive colitis with severe watery diarrhoea within three days pi, and was euthanased. *B. pilosicoli* cells were observed attached to the colonic epithelium adjacent to the areas of erosion. All the other pigs on both diets developed a mild transient diarrhoea, lasting only 2-3 days. At post-mortem examination small areas of mild patchy colitis were observed grossly, but no spirochaete attachment to the epithelium was detected.

This study demonstrated that, as with *B. hyodysenteriae,* colonisation by *B. pilosicoli* can be influenced by diet. In this case the rice-based diet did not prevent colonisation, but only retarded the process. From this finding it seems that the two spirochaete species have different environmental requirements and constraints on their ability to colonise the large intestine of pigs. The details of these differences are completely unknown, and may or may not be associated with the different micro-niches that they occupy in the large intestine, with differences in their metabolism and physiological requirements, or with other factors. The generally mild nature of PIS, together with the lack of complete protection using the rice-based diet, means that the prospects for an economically-viable means of controlling PIS in piggeries by using the rice-based diet alone are poor. Given the differences between the response of the two spirochaete species, however, it might still be worth investigating whether other dietary changes have a relatively greater influence on the proliferation of *B. pilosicoli,* perhaps acting via different mechanisms than those postulated for *B. hyodysenteriae.*

Post-weaning colibacillosis

The occurrence of growth checks and diarrhoea in the first 5-10 days after weaning remains a serious problem in many piggeries. Colonisation of the small intestine with enterotoxigenic strains of *E. coli* in this period results in a severe secretory diarrhoea (post-weaning colibacillosis: PWC), and, besides mortalities and the requirement for

antimicrobial medication, the associated growth checks can result in overall increases in time taken to reach market weight (Hampson, 1994).

It is well-established that PWC is a multifactorial condition, and that there are dietary influences on the disease (Hampson, 1987). For example, "high" concentrations of dietary protein (21%) have been shown to predispose to the condition (Prohaszka and Baron, 1980). Some highly digestible and milk-based weaner diets have been associated with reduced post-weaning diarrhoea (English, 1981), whilst conversely it has been suggested that the inclusion of fibre sources to weaner diets will reduce the incidence and severity of PWC (Bertschinger *et al.*, 1978; Bolduan *et al.*, 1988).

To investigate these contradictory observations further, the growth rates and recovery of haemolytic *E. coli* from the faeces of pigs fed a highly-digestible cooked rice/animal protein diet, or the same diet supplemented with guar gum as a source of additional sNSP, were examined (McDonald *et al.*, 1997; 1999). Pigs fed the basic rice diet for two weeks after weaning were heavier, and had lighter large intestines and less fermentation at this site, than the pigs fed the diet containing guar gum. When pigs on the two diets were challenged with enterotoxigenic *E. coli*, significantly more of these organisms were recovered from the small intestine of the pigs on the rice diet supplemented with sNSP than on the other diet (Table 11). Pigs fed a commercial wheat-lupin based diet had significantly more of the pathogens isolated than did the pigs on the basal rice diet (data not shown).

Table 11. The recovery of haemolytic *Escherichia coli* from the intestinal tract of infected weaner pigs (after McDonald *et al.*, 1999).

	Diet Type[1]		
Item	RA	RAGG	Statistics[2]
Mean CFU[3]/g in small intestine	1.3×10^4	8.0×10^9	*
No. of positive sites in SI[4]	0.53	1.63	**
CFU/g in colon	1.4×10^7	3.7×10^{10}	NS
No. of positive sites in colon	1.71	1.69	NS

[1]Diets: RA: 74% cooked white rice + 20% animal protein + 4% soya bean meal; RGG: 64% cooked white rice + 10% guar gum + 20% animal protein + 4% soya bean meal. [2]*P< 0.05; **P<0.01; NS, not significant. [3]CFU: colony-forming units. [4]Maximum number of possible positive sampling sites in small intestine (SI) was 3.

This experiment suggests that the presence of sNSP in weaner diets is detrimental in terms of both piglet growth and proliferation of enterotoxigenic *E. coli* in the small intestine. It also points to the benefits of feeding a highly-digestible, rice-based diet to weaners. The mechanism(s) involved in the protection from PWC is not certain, but may be related to the reduced availability of substrate for the bacteria in the small intestine of pigs fed the rice-based diet. Apparently it is not necessary to feed sources of fibre to stimulate the development of the large intestine as a means to promote good health and production in the immediate post-weaning period. Furthermore, diets that promote development of the large intestine do this by diverting growth away from the rest of the carcass. If dietary sNSP is confirmed as a predisposing factor in PWC, then careful selection of ingredients to minimise these components and/or treatment with exogenous enzymes may be helpful in controlling the condition.

Ulceration of the *pars oesophagea*

Ulceration of the *pars oesophagea* of the stomach is a common finding in pigs at slaughter, with advanced lesions being associated with reduced weight gain (Ayles *et al.*, 1996). The condition is well known to be responsive to diet, with many factors such as the fineness of dietary grind, pelleting, cornstarch content, gelatinisation, and unknown factors in wheat all having been implicated in its aetiology (Friendship, 1999). More recently, workers in Brazil have demonstrated a link between the presence of *Helicobacter*

heilmannii in the stomach and the occurrence of stomach ulcers (Barbosa *et al.*, 1995; Queiroz *et al.*, 1996) - a situation which parallels the involvement of *Helicobacter pylori* in human stomach ulceration.

To study possible interactions between *H. heilmannii* and diet in the aetiology of ulceration, a weaner model of stomach ulceration was developed (Accioly *et al.*, 1998). In this model, weaners fed finely ground wheat developed quite severe ulceration after 2-3 weeks, and there was evidence of a urease-producing helical bacteria being naturally present in the lesions (these bacteria were thought to be *H. heilmannii*). In this model, pigs fed wheat that had been extruded remained healthy, and there was no evidence of the urease-producing organisms being present. By some as yet unknown mechanism, extrusion of the wheat resulted in the absence of the organism. At this stage it is unclear whether *H. heilmannii* is causal, and the possibility that the organism proliferates secondarily to the presence of an ulcer cannot be excluded. For example a recent study in gnotobiotic swine failed to produce ulceration of the *pars oesophagea* when the animals were inoculated with *H. heilmannii* and fed a carbohydrate-enriched liquid diet (Krakowa *et al.*, 1998). In contrast pigs fed this diet and inoculated with *Lactobacillus* and *Bacillus* spp. developed ulcers. Fermentation by these bacteria was most likely encouraged by the presence of readily available dietary substrate, and the acidic short chain fatty acids produced as end-products of the fermentation were damaging to the epithelium. Whether or not *H. heilmannii* is a primary pathogen in the stomach, or whether other bacteria may contribute to damage to the epithelium of the stomach, both possibilities provide links between diet, enteric bacteria and disease. Knowledge about such links provides new opportunities for the control of ulceration of the *pars oesophagea* in pigs.

Conclusions

The studies described above provide four examples where there are connections between the pig's diet, and the presence and/or extent of proliferation of pathogenic enteric bacteria. These pathogens inhabit very different parts of the gastrointestinal tract (stomach; small intestine; large intestine), and are themselves quite distinct. Nevertheless, in each case feeding diets that are low in soluble NSP and/or resistant starch can reduce their proliferation. Such dietary effects on infection may not be restricted to bacterial enteric pathogens, as there is evidence that carriage of the parasitic nematode *Oesophagostomum dentatum* in pigs is enhanced by diets rich in insoluble fibre (Petkevicius *et al.*, 1997).

The basis of the protective effects that have been observed is not known, nor is it clear whether different mechanisms with the same outcome are involved in the different infections in different parts of the gut. The protection may be related to reduced availability of substrate for the pathogen, or physicochemical changes in the intestinal environment, such as reduced viscosity of the digesta. Under these changed circumstances the exogenous pathogenic zoonotic bacteria may face increased constraints in colonising and proliferating. Even though no clear explanation has been found for the protection achieved, this work suggests the possibility of new approaches to the control of enteric pathogens. The principles involved may also apply to other bacterial populations in the gut, for example, by reducing sNSP levels in the finisher diet it may be possible to minimise the subclinical intestinal carriage of pathogenic bacterial species which can contaminate meat, and which present a threat to human health. The work may also be relevant to intestinal infections in other monogastric species, particularly poultry and human beings.

In summary, to answer the question posed by the title of this paper, there is evidence that the occurrence and severity of certain enteric infections can be influenced to a greater or lesser extent by the use of diet. The cost-effectiveness of this approach for direct control of diseases in production animals does however require careful consideration. Whilst it may be economically viable to feed highly refined diets to pigs for a short period after weaning, or during a disease clean-up operation, it is not something which can be universally applied. Current efforts are directed at adding exogenous enzymes to the diet or fermenting the diet before it is fed as relatively cheap methods of

reducing sNSP concentrations in the diet. The expectation is that this will help limit colonisation of the intestinal tract by pathogenic bacteria.

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Symposium continued on next page

SPECIFICALLY SELECTED PROBIOTICS CAN IMPROVE HEALTH AND PERFORMANCE OF PIGS

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Abstract

The concept of probiotics is far from new, however progress in this research area was slowed by the availability of antibiotics since satisfactory disease control was achieved with antibiotics. With the emergence of antibiotic resistant pathogens in pig raising units, there is a need to consider alternatives for improving health and performance of pigs. There are many conflicting statements made about the efficacy of probiotics in pigs, however, it is now accepted that with careful attention to the criteria used to select the particular probiotic strain, one can improve piglet health and hence performance. This requires that the target be identified and that a probiotic strain be selected which is biologically active against that target and which will be able to colonise and be metabolically active *in situ*. When this approach has been applied to post-weaning piglet diarrhoea, a strain of *Lactobacillus* was selected which both inhibited growth and adhesion of enteropathogenic *E. coli* K88. When the strain was evaluated in a field trial, it was shown to protect against post-weaning diarrhoea and improve weight gain compared to controls. It is envisaged that by selection of a number of strains which are biologically active against a range of digestive tract pathogens, it will be possible to develop a mixture of strains which provide broad spectrum protection against digestive tract disease.

Introduction

Description of probiotics

The term probiotics is used to refer to preparations of live microbes that are added to feeds and feed supplements to improve the health of the host by beneficially influencing the indigenous microbes (Fuller, 1992). Lactic acid bacteria, which are used as starter cultures for yoghurt production, have been commonly used as probiotics for man and animals. The concept dates back to the time when Metchnikoff proposed that the longevity of the Bulgarian people was attributable to the fact that they consumed fermented milk products containing microbes that could prevent the establishment of the putrefactive microbes in the bowel. Since that time there have been many studies investigating this hypothesis in both man and animals. Unfortunately these often produced conflicting findings and no conclusive proof of the value of probiotics was forthcoming as discussed in more detail below (Conway, 1989).

The emergence of the antibiotics delayed the development of probiotic usage since effective disease control could be achieved with antibiotics. It is ironic that with the development of antibiotic resistance in pathogenic microbes, probiotics are now being re-evaluated for use in reducing gastrointestinal disease. In the late 1980s and early 90s the potential for disease control in pigs using probiotics was examined in detail (Conway, 1989; Jonsson and Conway, 1992; Stewart *et al.*, 1993). It was concluded that if one used a stringent selection criterium for choosing the particular probiotic strain, it would be possible to develop efficacious probiotic preparations for improving gastrointestinal health. Such preparations would lead to a reduction in disease and hence a reduction in antibiotic usage.

Mode of action

The probiotic strains may directly or indirectly protect the host from disease inducing microbes. The direct protective action of the probiotic microbes can be

attributed to their capacity to outcompete the ingested pathogenic microbes. This has been referred to as colonization resistance (van der Waaij *et al.*, 1971). There is well documented proof of the *in vivo* function of colonization resistance (Fuller, 1986). Another direct protective action of probiotic strains involves the production by the probiotic microbe of compounds which inhibit the growth of the pathogen (Lindgren and Dobrogosz, 1990). A number of compounds have been implicated and these include lactic and acetic acid, hydrogen peroxide, peptides, fatty acids, reuterin and proteinaceous bacteriocins. While inhibition of the growth of the pathogen has been extensively studied *in vitro*, evidence of *in vivo* function of the compounds is lacking. It has also been reported that probiotic strains can produce components *in vitro* that prevent enteric pathogens from adhering to the intestinal mucosa, an initial step in invasion by the pathogen (Blomberg *et al.*, 1993). While there is no *in vivo* evidence of inhibition of adhesion of enterotoxigenic pathogens, it is interesting to note that pathogenic *E. coli* K88 adhere more poorly to mucosa from pigs with high natural levels of lactobacilli colonising the mucosal surface (Blomberg and Conway, 1989).

The indirect protective effects of probiotics can occur by either (a) the probiotic strain allowing the establishment or restoration of desirable indigenous microbes, also referred to as microbiota, such that harmful microbes can't establish; or by (b) the fact that the probiotic microbe may stimulate the immune system for the benefit of the host.

Gastrointestinal microbiota of the pig

The gastrointestinal tract of the pig is sterile at birth (Barrow *et al.*, 1977; Conway, 1995a) and is then successively colonized by microbes until a very complex stable microbiota develops (Moughan *et al.*, 1992). This population contributes to health and growth performance in a number of ways including providing protection against pathogens (Barrow *et al.*, 1977; Muralidhara *et al.*, 1977; Cargill, 1982; Blomberg and Conway, 1989) and contributing to the production of some essential vitamins and short chain fatty acids (Leavitt *et al.*, 1978). The gastrointestinal tract is a dynamic ecosystem with equilibrium existing among the various components, including the diet, the host physiology and the microbes. It is well established that an alteration in the diet can influence the microbes, which in turn influences the health of the host (Lawrence, 1983; Moore *et al.*, 1987; Conway, 1995a). Furthermore, it is known that external environmental conditions such as unfavourable temperature and humidity can influence the stability of the gut microbiota and in turn the susceptibility to colonization by pathogens (van der Waaij *et al.*, 1971).

A well documented example is the situation at weaning when piglets are more sensitive to enteropathogenic *E. coli* (Barrow *et al.*, 1977; Blomberg and Conway, 1989; Jonsson and Conway, 1992). The process of weaning represents a multitude of physiological shocks: the piglet is deprived of protective factors in the sow's milk; there is a slow host response to the altered diet which results in undigested material being available to pathogens; the removal of the sow removes a major source of body heat thus exposing the piglet to low temperatures which can be detrimental to mucosal antibody production by damaging the lymphocytes; the piglet suffers nutrient deprivation at weaning since they often do not eat for 1-2 days. The dynamics of the microbial population are further influenced by the changing management strategies such as segregated early weaning (SEW), the introduction of creep feeds and an increasing reliance on the use of antibiotics both as growth promoters and in the control of respiratory and intestinal diseases. Alterations in the stability and protective nature of the normal microbiota may be minimised by the oral administration of specific beneficial probiotic microbes.

Selection criteria for evaluating probiotic potential of strains

A new generation of probiotic strains is emerging since it is now recognized that one must apply stringent selection criteria for the probiotic strains (Havenaar *et al.*, 1992; Conway and Henriksson, 1994). It is agreed that the probiotic strain must be metabolically active in the gastrointestinal tract and targeted to be inhibitory to particular

pathogens. Furthermore, the specific strain must be characterised, since not all members of a particular bacterial species are identical. To ensure the probiotic strain will be able to grow within the tract, a number of *in vitro* parameters are evaluated and these include the survival of the strain at low pH, in bile acids and digestive enzymes and the capacity to associate with the epithelial mucosa since adhesion favours colonisation by reducing the washout of the introduced strain. It has been suggested that the probiotic strain would have an ecological advantage if it originated from the pig digestive tract. Furthermore, the site of origin within the host tract could be important since the lactobacillus distribution throughout the tract is site specific for some strains (Henriksson *et al.*, 1995). Furthermore the viability of the probiotic strain prior to administration needed to be investigated since some potential probiotic strains survived poorly with low levels of viable cells in final products.

Probiotics usage in pigs

Historical perspective

Oral administration of lactic acid bacteria to piglets to improve their health was used as early as 1947 when Mollgaard (1947) dosed piglets with a lactic acid bacillus of host origin and reported improved health and improved skeletal formation. He proposed the phytic acid in ungerminated seeds in the diet interfered with absorption of calcium and phosphorus, an effect which he showed could be inhibited by lactic acid (Mollgaard, 1946).

The prophylactic use of lactic acid bacteria for piglets received little interest until the eighties when regulatory authorities and the public became concerned with the increasing need for the use of low dose antibiotics to facilitate good growth rates in intensive commercial pig units. This is particularly evident from the number of reviews of the topic over the last 2 decades (e.g., Wolter and Henry, 1982; Jonsson, 1985; Fuller, 1986; Sogaard, 1987; Conway, 1989; Fuller, 1989; Sissons, 1989; Fuller, 1992; Jonsson and Conway, 1992). Lactobacilli are the most commonly used probiotic bacteria for piglets (Table 12), with *Lactobacillus acidophilus* being the most studied species. *Enterococcus faecium* has also been extensively used for piglets and a limited number of studies have reported using bifidobacteria and mixtures of several species of lactobacilli.

Table 12. Lactic acid bacteria (LABs) utilized as probiotics for piglets (summarized from Jonsson and Conway, 1992).

Lactobacillus spp	*Bifidobacterium bifidus*
L. fermentum	*Bif. pseudolongum*
L. acidophilus	*Bif. thermophilus*
L. murinus	*Enterococcus faecalis*
L. lactis	*Ent. faecium*
L. reuteri	
Mixed LABs prepared from the following:	
L. plantarum, L. casei, L. fermentum, L. brevis, L. acidophilus, Ent. faecium, Streptococcus salivarius, L. delbrueckii	

The efficacy of oral administration of lactic acid bacteria to piglets can be studied in terms of their influence on the stability of the digestive tract microbiota and pathogen invasion as well as the performance and health of the pig. Because of ease of measurement and the commercial interest in the performance and health of the animal, these parameters are most commonly monitored. Some studies report beneficial effects from the prophylactic use of lactic acid bacteria for piglets while others found no effects, or even detrimental effects such as the decreased weight gain and lower feed conversion noted with dosage of an *L. reuteri* (Ratcliffe *et al.*, 1986) and in one case higher mortality following the use of *Ent. faecium* strain M74 (Kluber *et al.*, 1985). It is possible that detrimental effects may be the result of over-dosing since there has been one report that overdosing humans can have a laxative effect (Gordon *et al.*, 1957). In contrast to the

single study of Kluber *et al.*, (1985) using strain M74, there have been at least six studies reporting improved performance with administration of strain M74, e.g., Moen (1982). Oral administration of a strain of *L. reuteri* different from that used by Ratcliffe *et al.*, (1986) resulted in improved mucosal morphology (Jonsson and Henningsson, 1991). *Enterococcus faecium* strain C68 dosage to pigs has resulted in less disease (Krarup, 1987) and improved performance (Maeng *et al.*, 1989). Inconsistent findings have also been observed when *L. acidophilus* was used, with some workers reporting no effect (Kornegay, 1985) while several other groups noted improved performance (e.g., Redmond and Moore, 1965). A number of factors could contribute to the reported inconsistent findings and these include variations in the age of the animal, environmental conditions, sensitivity of the gut to disturbances, diet, as well as variation in the strain and in the viability of the preparation (e.g., Wolter and Henry, 1982; Jonsson, 1985; Fuller, 1986; Sogaard, 1987; Conway, 1989; Fuller, 1989; Sissons, 1989; Fuller, 1992; Jonsson and Conway, 1992).

A case study of a targeted probiotic preparation

The recommendations that probiotic strains should be specifically selected such that they are metabolically active in the digestive tract and are biologically active against an identified target were investigated for pigs in an environment where the major cause of post weaning diarrhoea was K88 fimbriated enterotoxigenic strains of *E. coli* (Conway, 1995b). Potential strains were screened for biological activity against enterotoxigenic *E. coli* bearing K88 fimbriae. Measurements included inhibition of the growth of, and inhibition of the adhesion of, the pathogen to gut mucosa. Inhibition of adhesion of the pathogen was investigated using dialyzed and fractionated spent culture in an *in vitro* adhesion assay (Blomberg *et al.*, 1993). Furthermore since *E. coli* K88 colonize the distal ileum, lactobacillus isolates from the piglet digestive tract were selected for the capacity to colonize the stomach and hence be continually seeded into the distal ileum as viable cells. Survival of the isolates in low pH, bile acids and enzymes was evaluated by appropriate additions to buffered saline and growth media (Conway and Henriksson, 1994; Gibson and Conway, 1994).

Piglets in a commercial piggery were given *ad libitum* access to the selected *L. fermentum* strain via creep feed and weaning feed since the strain was added as a freeze dried supplement to the powdered feed. Prior to weaning an increased dose of the *L. fermentum* strain was given by addition of more freeze dried supplement. The incidence of diarrhoea and weight gain over the 9 week period were monitored in the negative (no additions to the feeds) and positive (antibiotics added to feeds) control piglets and the test piglets all housed in the same building (8 litters per group) plus additional positive and negative control pigs (7 litters per group) which were housed in a separate building. These latter controls were compared with those housed with the test groups to detect any contamination of the controls that may have occurred when controls were housed in the same building as the test animals.

Table 13. Body weights at 0, 37 and 63 days of age of piglets orally dosed with lactobacillus (test group) relative to that of control animals (positive control receiving antibiotics) housed in the same building. Piglets were weaned at 37 days. Results expressed as the mean ± SE (8 litters per group; approximately 80 piglets per group) (Conway, 1995b).

Group	Weight (kg)		
	0 days	37 days	63 days
Negative control	1.71 ± 0.07	6.59 ± 0.42	11.26 ± 0.81
Positive control	1.49 ± 0.06	5.79 ± 0.29	12.42 ± 1.20
Lactobacillus dosed	1.42 ± 0.07	6.99 ± 0.43	14.53 ± 0.70

Table 14. Weight gain of piglets orally dosed with lactobacillus (test group) relative to that of control animals housed in the same building. Weight gains of the test group are expressed as the percentage by which they are greater than that of the negative and positive controls (the positive controls received antibiotics). Piglets were weaned at 37 days and the results are presented for the entire period of study (0-63 days), pre-weaning (0-37 days) and post-weaning (37-63 days) (Conway, 1995b).

Group	Weight increase of test group (%)		
	0-37 d	0-63 d	37-63 d
Negative control	35*	72*	55*
Positive control	32	28	4

* Test group significantly different from control, $P < 0.05$. No. of litters per group = 8.

As can be seen in Tables 13 and 14, the piglets receiving the *L. fermentum* (test group) significantly gained more weight than piglets in the negative control group which received no supplements and also gained more weight than those in the positive control group receiving an antibiotic supplement. This increase compared to the positive controls was not statistically significant. No post-weaning diarrhoea was detected in the litters receiving the *L. fermentum*, while 80% of the negative control litters either housed with the test animals (8 litters) or in a separate building (7 litters) suffered diarrhoea (Table 15). It was concluded that the criteria used for strain selection yielded isolates that had the capacity to colonize the digestive tract and the results provide indirect evidence that the strains also exerted biological activity on the pathogenic *E. coli* K88. Additional work with this strain has also shown lowered levels of pathogenic *E. coli* in pigs dosed with the strain (Chandler, personal communication).

Table 15. The incidence of diarrhoea in piglets before and after weaning at 37 days for negative and positive controls (the positive controls received antibiotics) as well as the test group (orally dosed with lactobacilli). Negative controls and positive controls were both housed with the test animals (8 litters per group) and in a separate building from the test animals (7 litters per group). Results are expressed as the number of litters with diarrhoea as a percentage of the total number of litters (Conway, 1995b).

Group	No. of litters	Before weaning	After weaning
Negative controls	15	47 %	80 %
Positive controls	15	33 %	27 %
Test group	8	25 %	0 %

Scope and potential

It is apparent from the case study reported above that by careful strain selection, efficacious probiotic preparations for usage in the pig industry can be developed. To increase the spectrum of activity a number of probiotic strains could be combined such that most pathogens could be targeted. Because of the mode of action of the probiotics, the problems with emergence of resistant strains such as occurs with antibiotic usage would not be anticipated with probiotics. The efficiency of the probiotic preparations could be further enhanced by selecting prebiotics, which are carbohydrates such as oligosaccharides, that are not degraded by host enzymes but are selectively utilized by beneficial bacteria such as probiotic strains. The inclusion in the piglet diet of both probiotic strains and prebiotics which give a selective advantage to the probiotic strains will favour establishment of the probiotic strain in the digestive tract of the piglet and enhance probiotic performance.

Symposium continued on next page

CAN VACCINES REPLACE ANTIBIOTICS?

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Abstract

Antibiotics are used to treat animals infected with bacterial pathogens. In addition, over half of all antibiotics used in the animal industries are added to feed for growth promotion effects. Vaccines are used prophylactically to control infectious disease. There would appear to be two main ways that vaccination strategy could contribute to reducing the use of antibiotics in the livestock industries. Firstly, improve the cost, efficacy and repertoire of available vaccines to increase adoption of vaccination as a standard practice. Secondly, and related to the first point, attempt to predict which diseases may become problematic if antibiotics in feed are withdrawn and ensure that vaccine solutions are available. Improvement to existing, and development of new, vaccines is likely to require significant expenditure on research and development. The challenge will be to balance the costs of new product development with the returns possible from a particular vaccine market because if this cannot be achieved, vaccine manufacturers will be reluctant to invest.

Introduction

This paper aims to address the role of vaccines in disease control and their potential to reduce antibiotic use. The vast majority of pig vaccines are designed to control bacterial or viral infections. Since antibiotics act directly against bacteria there is a clear need to consider how vaccination against bacterial disease may be used to reduce the need for antibiotics. Certain viral infections however, may predispose pigs to bacterial disease so that viral vaccination may also be an important component within a strategy to reduce the use of antibiotics. Most of the major viral diseases of pigs are exotic to Australia and the only one recognized as important, by virtue of a registered vaccine, is parvovirus. Here, therefore, the discussion will focus upon pig diseases caused by bacteria.

Vaccination can be defined as a deliberate attempt to raise an immune response against a designated disease agent. Successful vaccination implies raising a *protective* immune response. There are, however, different degrees of protection and this is referred to as vaccine efficacy. These statements are obvious yet fundamental to a discussion on the role of vaccines in disease reduction. At one end of the spectrum it could be argued that if fully efficacious vaccines for every pig disease agent were possessed and properly used, antibiotics would not be needed. Although this is highly unlikely to occur, it provides one clear path toward decreasing antibiotic use. Vaccine development programmes can be designed to produce more efficacious, cost effective, user friendly vaccines against the most relevant diseases. These factors provide a complex matrix, all of which need to be considered within an economic framework. Some of these issues will be discussed below.

Role of vaccines in disease reduction

It is useful, when considering trying to improve vaccine efficacy, to have some understanding of the host immune response. Broadly, the immune system is divided into two main parts characterised by antibody and cellular responses. The major antibody responses involved in infectious disease are systemic (prototype, circulating ImmunoglobulinG (IgG)) and mucosal (prototype, surface IgA). Cellular immune responses are often directed toward intracellular pathogens such as *Salmonella* .

Bacterial pig pathogens mainly cause enteric or respiratory diseases (Table 16) and thus affect the host at mucosal surfaces. It would therefore be reasonable to expect mucosal immune responses (e.g., IgA) to play an important role in protecting pigs against

many bacterial infections. Unfortunately, predicting required protective immune response(s) cannot always be done by analysing a pathogen's mode of infection and life cycle within the host. First, it is probably overly simplistic to view the immune system, and therefore its responses, separately rather than as an interactive continuum and secondly, there may be insufficient information about the pathogen.

Having said that, there are vaccines that work by eliciting a particular immune response against a single, major bacterial virulence factor. This type of vaccine can work when the immune response can both see and be directed toward one or more *invariant* sites on a single major virulence factor (e.g., dermonecrotic toxin (DNT) produced by Type D *Pasteurella multocida* (Table 16)). Although type D *P. multocida* infects pigs at the respiratory mucosa, protection is highly correlated with humoral anti-DNT IgG responses.

Table 16: Major bacterial diseases of pigs.

Pathogen	Disease	Type of infection	*Commercial vaccine available	*Vaccine registered for use in Australia
Actinobacillus pleuropneumoniae	Pleuropneumonia	Respiratory	Y	Y
Pasteurella multocida Type D	Atrophic rhinitis	Respiratory	Y^{x}	N
Bordetella bronchiseptica	Atrophic rhinitis	Respiratory	Y	N
Pasteurella multocida	Pneumonia	Respiratory	N	N
Mycoplasma hyopneumoniae	Enzootic pneumonia	Respiratory	Y	Y
Streptococcus suis	Meningitis	Respiratory/ Systemic	Y	N
Haemophilus parasuis	Glasser's disease: arthritis, pneumonia	Respiratory/ Systemic	Y	N
Campylobacter sp.	Enteritis	Enteric	?	N
Lawsonia	Enteritis	Enteric	?	N
E. coli	Scours	Enteric	Y•	Y
Leptospira sp	Leptospirosis	Enteric	Y	Y
Serpulina hyodysenteriae	Dysentery	Enteric	Y	N
Erysipelas rhusiopathiae	Arthritis	Enteric	Y	Y
Clostridium perfringens	Enteritis	Enteric	Y	N
Salmonella cholerasuis	Scours, pneumonia, septicaemia	Enteric	Y^{+}	N

*Bacterin. •subunit. ^{x}r-subunit. $^{+}$live, gene deleted. ?none on product list of companies reviewed. Intervet International and Pfizer Animal Health Web sites and the National Registration Authority (NRA) product database Web site.

Raising protective immune responses against some other bacterial pathogens is not so simple. Several pathogens possess variant (e.g., *E. coli* pili, *Actinobacillus pleuropneumoniae* (APP) toxins), multiple, undefined (e.g., *Erysipelas rhusiopathiae, Mycoplasma hyopneumoniae*) virulence factors, some of which are not accessible to the immune system. This situation is clearly problematic and requires highly sophisticated approaches (e.g., discovery of new vaccine antigens, delivery systems, multivalent formulations) to provide a solution (Table 17). These approaches however come at some considerable cost. Therefore, the simpler, cost effective, bacterin vaccine formulation has been widely adopted (Tables 16 and 17). These vaccines are a necessary compromise between efficacy and cost of production. Generally, as the level of technology needed to provide a new vaccine solution increases (Table 17) so too does the cost of research and product development.

From a technical point of view vaccines have a large potential role in controlling bacterial disease thus in reducing the use of antibiotics. This role could be increased by improving vaccine efficacy and by expanding the repertoire of available vaccines. Ways of increasing the number and type of available vaccines include improving existing vaccines, developing vaccines for diseases for which there is no current vaccine and developing vaccines for diseases predicted to be a problem in the future. The latter category relates most specifically to the effect of reducing or withdrawing some uses of antibiotics and identifies, perhaps, the most valuable but difficult exercise.

Table 17. Main advantages and limitations of various vaccine formulations.

Type	Characteristics
Bacterins	Adjuvanted, killed whole cell mono or multivalent. Complex cellular proteins increase the chance of including relevant protective antigens without needing information about bacterial pathogenesis. Important *in vivo* expressed antigens will not be present unless the bacteria are grown in special media. The complex antigen "load" can elicit strong, irrelevant immune responses. In some cases this can result in poor efficacy and/or side-effects such as auto-immunity, immune depression, site reactions, fever, inappetence and weight loss. Injected.
Sub-unit (including recombinant sub-unit)	Adjuvanted, number of defined bacterial components (usually proteins) obtained either from fractionation or recombinant DNA technology. Costly R&D needed to define efficacious sub-units. Usually more expensive to prepare than bacterins. Formulations can have lower side effects and higher efficacy than bacterins. Injected.
Modified live (including recombinant modified live)	Bacterial pathogen rendered avirulent using for example (A) passage, (B) chemical mutagenesis or (C) by introducing a defined mutation(s) using recombinant DNA technology. These types of vaccines are usually more costly but potentially more efficacious than bacterins and sub-units. (A) and (B) possess undefined mutations so additional safety trials need to be conducted. Live recombinant vaccines need costly R&D to define the gene(s) and introduce the mutation(s). In addition they will be subject to more extensive regulatory approval. Live vaccines usually need to be lyophilised. Presentation *in vivo* of full spectrum of bacterial proteins, thus potentially enhancing efficacy. Deleterious antigens however will also be present. No adjuvant needed. Oral, nasal, or delivery by injection possible. Potentially single dose.
Live vector vaccines	Bacterial pathogens rendered avirulent can be used to carry vaccine antigens from other bacterial pathogens (recombinant sub-units), thus producing live, multivalent vector vaccines. This approach combines the advantages and disadvantages of 2 and 3 above and adds the advantage of providing multivalence to the modified live vaccine.

Technology and cost increases down the table. Application of new technologies to vaccine development can provide new solutions to disease control. For general review see Hodgson and Radford (1993).

Potential to reduce antibiotic use

The above discussion has identified that vaccination has an important role in disease reduction and therefore may be one strategy used to reduce the use of antibiotics. Further, through improvement in the type and variety of available vaccines, there is considerable scope to increase the impact of vaccination in this area. Consideration of the way in which antibiotics are used within the industry together with a look at animal health markets, can provide some clues to the potential magnitude of the impact that vaccination may have in reducing the use of antibiotics.

Antibiotics have a broader spectrum of activity than vaccines. They are used therapeutically to remove or reduce disease, and prophylactic use appears to "promote growth". It seems clear that at least in principle, vaccines could substitute for antibiotics that are used therapeutically to control particular diseases. The situation is not so clear for prophylactic use of antibiotics. The precise mechanism for the growth promotion effect is unclear but one component is likely to be the general inhibition or removal of susceptible populations of pig-borne bacteria. The fact that resistance to in-feed antibiotics emerges within enteric pig bacterial populations indicates that the antibiotics are indeed used at doses that inhibit bacterial growth. It could be concluded that such inhibition results in growth promotion by reducing the overall metabolic load imposed upon the pig by the bacteria. Under this scenario it is likely that both commensal and pathogenic bacterial species are involved in the effect. Given this, it is unlikely that vaccines could become a general substitute for antibiotics as growth promoters because the identity of the target bacteria to achieve the effect is not known. Additionally, not all of the possible bacterial targets are accessible to the immune system. It would appear therefore, that the main role of vaccines in reducing the use of antibiotics would most likely be to control bacterial *pathogens* that are currently controlled therapeutically or prophylactically by antibiotics.

Pigs are treated therapeutically both against bacterial diseases for which vaccines are available in addition to those for which there is no suitable vaccine. As identified in the previous section, this leads to two main opportunities for decreasing antibiotic use in favour of vaccines: improved adoption (used properly, improve the product) of current vaccines and development of new vaccines.

The fact that many bacterial diseases are prevalent in the face of available vaccines and in-feed medication, suggests that where vaccination is failing, in-feed medication is not controlling disease. Examples from Table 16 include pleuropneumonia, atrophic rhinitis, scours, leptospirosis, enzootic pneumonia and erysipelas. It is difficult to say whether in-feed antibiotics are responsible for reducing the incidence of these diseases. On one hand if isolates of these bacteria were found to be resistant to antibiotics commonly used in feed, then you could probably conclude that removal of antibiotics would have little effect on the incidence of that particular disease (*E. coli* scours is a likely example). If bacterial isolates from *clinical disease* were sensitive to the same groups of antibiotics then you may conclude that the bacterial population was not exposed to a sufficiently high concentration of antibiotics. If they were they would either be inhibited to an extent to prevent disease or the population would be resistant. Again, withdrawal of antibiotics would therefore have little effect on the incidence of that particular disease (pleuropneumonia is a likely example). This line of reasoning oversimplifies the situation because it does not account for one or more bacterial infections predisposing the pig to another. It does however provide a lead.

As mentioned before, determining which bacterial diseases are likely to become more prevalent if antibiotics are withdrawn is a key issue. Given the above discussion, priority could be given to sub-clinical or relatively low incidence diseases caused by bacteria that are likely to be exposed to inhibitory doses of antibiotics. Lead candidates would be enteric diseases such as swine dysentery and porcine proliferative enteritis (PPE). Although bacteria in the respiratory tract are less likely to be exposed to inhibitory concentrations of feed antibiotics, Glasser's and *Streptococcus suis* meningitis could be good targets for future vaccine development.

It is interesting to note that animal producers currently spend about the same on injected antibiotic therapeutics, in-feed antibiotics and vaccines (Table 18). If the use of

these antibiotics could be replaced with vaccines then there is clearly enormous potential for growth in vaccine markets. It would be valuable to review within an economic analysis of the acceptable price of vaccines to producers, the likelihood that amounts currently spent on antibiotics would be available to spend on new efficacious vaccines. More expensive vaccines, if effective, may then be acceptable.

Table 18. World animal health and nutrition market share by product sector.

Product sector	World market $USB	Use in pig * $USM
Antibacterial/antibiotics	2.8	700
Anticoccidials	0.5	N/A
Biologicals/vaccines	2.7	700
Medicinal feed additives	2.5	600
Nutritional feed additives	4.4	1,100
Others	4.8	N/A
Total	17.8B	

*Assumes 25% of animal health and nutrition market assigned to pig products is spread evenly amongst the listed sectors. N/A, not applicable to this discussion. Wesley (1998 a, b).

Conclusions

- Vaccines currently play a large role in reducing disease
- This role can be expanded by improving existing vaccines (thus increasing adoption) and developing new vaccines for both existing and emerging diseases
- Increasing the role of vaccines in disease reduction will need investment in new vaccine technologies
- Vaccination is most likely to be useful against defined pathogens and is unlikely to be a substitute for antibiotics used as growth promoters
- If use of in-feed antibiotics is withdrawn, vaccination is most likely to be of benefit to control pathogens that are currently suppressed by such prophylaxis. Enteric pathogens are most likely to fit this category (e.g., swine dysentery and Porcine Proliferative Enteritis) although this may also apply to some respiratory diseases (e.g., *S. suis* meningitis and Glasser's disease).

Symposium continued on next page

SYMPOSIUM CONCLUSIONS

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During this symposium it was reinforced that there are negatives associated with antibiotic use in the pig industry. Consumer concern is mounting. Antibiotic use selects for populations of resistant bacteria and can result in antibiotic residues in tissues of treated pigs. However, what is the significance of these problems balanced against the importance of antibiotic therapy and the economic benefits of growth promotion? A question asked by Barton (pp. 194-199). Antibiotic resistance is readily detectable in bacterial isolates from pigs, and was found as long ago as 1957. Antibiotic residues still occur in pig tissues. To control resistance and residue problems a detection and monitoring system is required. Such a system is in place for residues but not for antibiotic resistance in any livestock in Australia. There is worldwide concern about spread of antibiotic resistance from animal isolates of bacteria to human pathogens. The recommendations in 1999 of the Joint Expert Technical Advisory Committee on Antibiotic Resistance (JETACAR) are directed towards better controls on the use of antibiotics, encouragement of prudent use of antibiotics and a review of use for growth promotion and long term prophylaxis.

Reduction in antibiotic use can be achieved through reduction in the microbial load presented to a pig from its environment. Madec and Leon (pp. 200-209) argued that disease prevention is better than cure and it does not require antibiotics. Disease prevention should be directed towards provision of zootechnical profiles that reduce risk, especially when many factors contributing to disease, both in the animal and the environment, must be taken into account and tackled step by step. The use of antibiotics to reduce microbial pressure in a prevention scheme is questionable. Under adequate environmental conditions and perhaps combined with the use of vaccinations, the impact of enzootic diseases can be low. Attention to optimal hygiene, building design, strategic animal movement, biosecurity, air quality and pig welfare all contribute significantly to reducing the risk of disease.

Whether diet can be used to reduce the susceptibility of pigs to enteric pathogens was discussed by Hampson *et al.* (pp. 210-219). It has been shown that diets with very low levels of soluble non-starch polysaccharides and resistant starch offer complete protection against experimentally induced swine dysentery. While the only diet giving complete protection, being cooked white rice with animal protein, is not a practical alternative, further work is progressing including the application of exogenous enzymes to standard diets. Porcine intestinal spirochaetosis and colonisation by enterotoxigenic *E. coli* were slowed with the rice-based diet but this did not prevent development of disease. The stomachs of weaner pigs fed a finely ground wheat-based diet were shown to have severe ulceration and were colonised by *Helicobacter* species, while feeding extruded wheat showed no ulceration and no bacteria. It would appear that diet influences the presence of bacteria involved in stomach ulceration. The results presented by Hampson (1999) indicate that a number of enteric bacterial infections can be modified by diet, and research should continue to elucidate practical diets.

Conway (pp. 220-224) presented a case study which showed that, by meticulous strain selection, probiotics can be developed for use in the pig industry. Probiotics are preparations of live microbes, added to pig feeds to improve health by enhancing the function of microbes already present in the pig. Emergence of resistance to this beneficial activity is unlikely due to the mode of action of probiotics. Combination of various probiotic strains can increase the spectrum of activity. Efficacy can be further enhanced with the addition of prebiotics for selective utilisation by beneficial bacteria. This combination, referred to as synbiotics, gives a selection advantage to the probiotic strain in a piglet's digestive tract.

Antibiotics act against bacteria, so in order to reduce antibiotic use, vaccines that will reduce bacterial disease are required. Certain viral infections can predispose to bacterial infections in pigs so vaccines against viruses are also important in reducing

antibiotic use. Hodgson (pp. 225-229) suggested that if fully efficacious vaccines existed for every pig disease agent, antibiotics would not be required. This is clearly not the case. Sophisticated research and high costs could prohibit development of the perfect vaccine therefore, in many cases, vaccines are a necessary compromise between efficacy and the cost of production. Antibiotics also have a wider spectrum of activity than vaccines, with both therapeutic use and prophylactic use. Vaccines may substitute for therapeutic use but it is unlikely they could be used as growth promoters. Vaccine development must be considered for bacterial diseases that are likely to become more prevalent should antibiotics be withdrawn from particular uses in pigs.

New and better ways must continually be sought to reduce the risk of diseases in pigs. Some of these methods have been outlined in this symposium and they provide useful directions for further research and consideration. Mechanisms to combat disease in pigs must be retained. These may be modified as applicable to address consumer concerns whilst maintaining optimum pig welfare.

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PASSIVE PROTECTION OF PIGLETS AGAINST DIARRHOEA WITH SPECIALIZED EGG IMMUNOGLOBULINS (PROTIMAX®)

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Specific spray-dried immunoglobulin G (Protimax®) is an alternative to in-feed antibiotics for piglets. This product is derived from hens' eggs, which have been vaccinated against bacterial infections (enterotoxigenic *E.coli*, serotypes K88, K99 and 987P) and porcine rotavirus (Mroz *et al.*, 1999). The objective of this study was to evaluate the effect of supplementing the diet with Protimax® (0.0 vs 1.1 g/kg), during four different periods of rearing (22-28 vs 22-42 vs 22-56 vs 28-56 days of age), on the growth rate, feed efficiency, apparent digestibility of nutrients, incidence of diarrhoea, and mortality of piglets up to 4 weeks after weaning.

A total of 339 piglets from 32 Large White x Polish Landrace sows were selected from a herd known for the presence of the intestinal receptor for the K88 antigen. They were weaned at 28 days of age, allotted randomly to eight treatments (four litters/treatment), and fed *ad libitum* with prestarter (21-35 days of age) and starter diets (36-56 days of age). These diets contained no antibiotics or increased amounts of Cu and Zn. The prestarter and starter diets contained 192/182 g/kg crude protein, 13.2/10.5 g/kg lysine and 13.8/13.8 MJ ME/kg, respectively. Data were subjected to analysis of variance according to a completely randomised design using the ANOVA procedure of Genstat 5, and t-tests to compare the means at $P<0.05$ and $P<0.01$.

Table 1. Main effects of PROTIMAX® on the feed intake (FI), body weight (BW), average daily gain (ADG), feed conversion ratio (FCR) and losses of piglets (%) from 28 to 56 days of age.

Main effects:	PROTIMAX® (g/kg)		Period of rearing with PROTIMAX® (d)				
	0.0	1.1	22-28	22-42	22-56	28-56	SED
Feed intake (g/piglet/d)	478.1	482.8	478.1	480.2	479.8	493.2	20.2
BW (kg): 28 days of age	7.3	7.4	7.3	7.4	7.5	7.4	0.13
56 days of age	16.0	16.5**	16.3	16.3	16.6	16.6	0.14
Average daily gain (g/d)	311.1	323.2**	319.6[a]	317.1[a]	326.1[ab]	330.0[b]	5.43
Feed conversion ratio	1.54	1.49*	1.50	1.51	1.47	1.49	0.02
Losses of piglets: a) total	7.6	2.5*	2.3	2.4	2.3	2.5	0.2
b) due to *E.coli* (%)	4.8	0.0*	0.0	0.0	0.0	0.0	0.0

*$P\leq0.05$; **$P\leq0.01$. [a,b]Values in the same row with different superscripts are significantly different ($P\leq0.05$).

For pigs receiving the control diet, which did not contain PROTIMAX®, there was a greater frequency of severe diarrhoea caused by enterotoxigenic K88 *E.coli*. This was reflected by the significantly greater losses of control pigs ($P<0.05$) throughout the entire experimental period (Table 1). The mortality rate of control piglets during the particular periods of rearing (22-28, 22-42, 22-56 and 28-56 days of age) was also greater (by 4.8, 6.9, 7.5 and 2.1%-units, respectively). Addition of PROTIMAX® to the diet reduced the occurrence and duration of diarrhoea by 4.9%-units and 1.1 days respectively ($P<0.05$). In addition, this product stimulated the growth performance of piglets and improved the apparent digestibility of crude protein in the prestarter diet by up to 2%-units ($P<0.05$) when included in the diet at 22 days of age. Piglets fed PROTIMAX® over a longer period (days 28-56) tended to have greater daily gains and utilized less feed per kg gain than those given this product over days 22-28 and 22-42, but the differences were not statistically significant. In summary, antibodies in PROTIMAX® (titres from 320 to 640 against K88 *E.coli*) appeared to be effective in improving piglets' resistance to these diarrhoea causing organisms.

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ACTIVE IMMUNIZATION AGAINST ADRENOCORTICOTROPIN (ACTH) ALTERS THE ENDOCRINE RESPONSE TO STRESS BUT HAS NO EFFECT ON GROWTH PERFORMANCE IN PIGS

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A previous study comparing singly- and group-housed pigs in a clean and dirty environment indicated that adrenal cortisol secretion, under the control of ACTH, was stimulated by both group housing and poor environment (Lee *et al.*, 1997). Active immunization against ACTH results in the generation of antibodies that inhibit ACTH bioactivity resulting in a reduction in plasma cortisol concentrations (Wynn *et al.*, 1994). This study examined the effect of this procedure on growth, circulating cortisol and β-endorphin in pigs maintained in single and group pens.

Male (n=64), 10 week old, Large White x Landrace pigs with live weight (LW) of 57 ± 0.6 kg (mean ± SE) were allocated to a 2 x 2 factorial experiment involving two pen sizes (single and group) and two immunization treatments. Each of two rooms (maintained at 22°C) contained eight single and four group pens (1 m^2/pig; 6 pigs/pen). Half of the pigs in each room were immunized against ACTH (1-24)-ovalbumin conjugate (0.5 mg) in DEAE dextran (2 ml), and animals in the remaining pens received adjuvant alone in weeks 0, 4 and 8. The pigs were offered a commercial, pelleted diet *ad libitum*. At week 9, each pig was subjected to acute restraint with a nose snare for 1 minute. Blood was sampled by venipuncture at times 0 and 10 minutes after release. Plasma cortisol and β-endorphin concentrations were measured by radioimmunoassay. At week 5, titres for ACTH-specific antibodies ranged from 1:500-2500.

Table 1. Mean (±SEM) growth rate (week 5-9), week 9 plasma cortisol and β-endorphin concentrations of grower pigs (57-87 kg LW) housed in either single (n=16) or group pens (n=8, 6 pigs/pen) and immunized against either ACTH or adjuvant (control).

Group size (G)	Single		Group		Main effect[a]	
Vaccination (V)	Control	ACTH	Control	ACTH	G	V
Daily live weight gain (g)	1166 (28.6)	1218 (33.6)	1025 (34.5)	1091 (40.8)	**	NS
Cortisol (ng/ml) 0 min[b]	8 (1.4)	11 (2.6)	11 (1.3)	10 (0.9)	NS	NS
Cortisol (ng/ml) 10 min[b]	20 (3.2)	12 (1.5)	21 (3.9)	12 (0.9)	NS	**
β-endorphin (pg/ml)	20 (2.3)	57 (17.8)	25 (3.9)	113 (27.5)	NS	**

[a]NS, Not significant, *P≤0.05, **P≤0.01 using a log transformation for cortisol and β-endorphin. There were no significant interactions between G x V. [b]Restraint for 1 minute and bled at 0 and 10 minutes.

Pigs grew 10% slower when housed in groups while basal concentrations of cortisol were similar in both ACTH-immune and control pigs. In contrast, the acute cortisol response to restraint was attenuated by ACTH immunization while basal β-endorphin concentrations were elevated in ACTH-immune pigs. However, growth was not significantly affected by ACTH immunization, suggesting that circulating glucocorticoid concentrations were not elevated sufficiently in the non-immunised animals to compromise growth in either single or group pens. Thus elevated cortisol secretion may not be responsible for slower growth in group-housed pigs.

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A SUBUNIT MEMBRANE ANTIGEN FOR THE SEROLOGICAL DETECTION OF ANTIBODIES AGAINST *ACTINOBACILLUS PLEUROPNEUMONIAE* IN PIGS

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Pleuropneumonia is an economically important respiratory disease of pigs caused by the Gram negative coccobacillus *Actinobacillus pleuropneumoniae* (App). Serovars 1, 7 and 12 are most frequently associated with disease in Australia but disease due to other serovars can occur as well. Current serological tests for App are based upon serovar specific capsular and lipopolysaccharide antigens. A serological test based on a common antigen to all App serovars (Sv) would be valuable to monitor pleuropneumonia in herds without the need to identify the causative serovar. Data will be shown to demonstrate that a major outer membrane protein (molecular size of 39 kDa) is antigenically conserved amongst all 12 type strains of App. This antigen was purified and assessed as an ELISA reagent in pigs experimentally infected with each of the three major App serovars.

Bacterial challenge strains HS54 (Sv1), WF83 (Sv7) and HS143 (Sv12) were kindly provided by Dr. P. Blackall (DPI, Qld). In Trial 1, 16 pigs were challenged intra-tracheally (IT) with $5x10^5$ cfu Sv1 in 2 ml Brain Heart Infusion broth (BHI)). These seeder pigs were allowed to recover for 2-9 d before mingling with uninfected cohorts (n=35). In Trial 2, two groups of six pigs, each located in separate rooms, were challenged IT with either $3x10^8$ cfu of Sv7 or $3x10^8$ cfu of Sv12. However, these seeder pigs were mixed with their respective contact cohorts (n=8) within a few hours post-challenge.

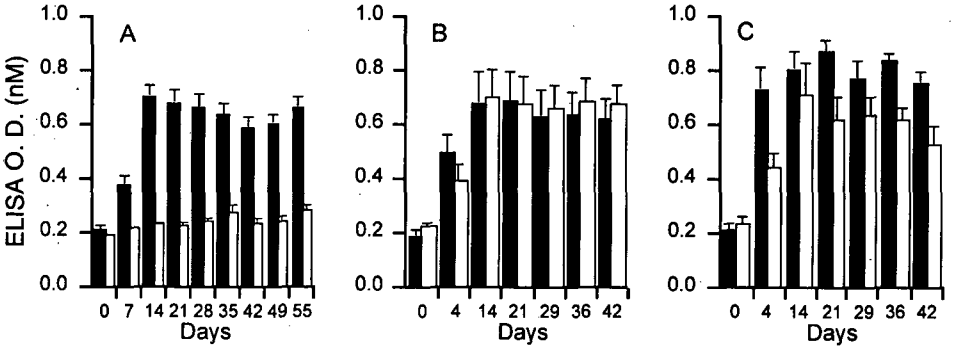

Figure 1. *Temporal serum antibody response (mean ± SEM) in seeder pigs after experimental challenge with App Sv1 (A), Sv7 (B) or Sv12 [solid bars] and their contact cohorts [open bars]. In A, all seeder pigs were clinical while contacts were disease free. In B and C, both seeder pigs and contact pigs developed clinical pleuropneumonia.*

The serum antibody responses of challenged seeder and contact pigs in both trials are summarized in Figures 1 A, B and C respectively. Evidence of clinical disease for each pig in the challenged or contact groups was based on the presence of lethargy, dyspnoea, anorexia and coughing and recorded on a quantitative scale. In Trial 1, all direct challenged but no contact pigs developed clinical disease. In contrast, clinical disease was seen in Trial 2 amongst all Ap7 or Ap12 direct challenged and contact pigs. In both trials, clinically-affected pigs rapidly developed antibodies against the 39 kDa antigen within 2 weeks of exposure (Figs. 1A, B and C). However, pigs without clinical signs (Trial 1 contacts), had low ELISA titres throughout the trial period.

These results demonstrate the utility of the 39 kDa antigen as an ELISA reagent for monitoring the serological response in pigs exposed to the 3 major App serovars in Australia. In addition, the serological response is rapid and maintained over several weeks, and was correlated with the clinical status of pigs with pleuropneumonia.

PIG RESEARCH AND DEVELOPMENT CORPORATION

THE DEVELOPMENT OF IMMUNITY TO *LAWSONIA INTRACELLULARIS* IN WEANED PIGS

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Love *et al* (1977) suggested that pigs exposed to the aetiologic agent of proliferative haemorrhagic enteropathy (PHE) develop immunity to further infection. They observed two sequential episodes of PHE in a minimal disease piggery. The first episode affected highly susceptible breeding sows and boars of all ages, but was quickly controlled with antibiotics. The second episode occurred 2 to 3 weeks later affecting only gilts and young boars, introduced into the breeding unit during the first *Lawsonia intracellularis* infection period. Pigs previously exposed appeared immune to infection during the second episode. The aim of this project was to determine if weaned pigs inoculated with *L. intracellularis* (the aetiologic agent of PHE and porcine intestinal adenomatosis, PIA), could recover from infection and remain immune to re-infection.

Eleven weaned, 3-week-old, Large White x Landrace pigs were housed in individual pens in isolation and offered feed without antibiotics. Four pigs were orally inoculated with *L. intracellularis* ($6x10^9$) extracted from homogenised PHE affected mucosa, and seven pigs were not inoculated. Pigs were monitored for clinical signs of PIA, including diarrhoea and reduced weight gains. Serum was collected weekly for an indirect immunofluorescent test (IFAT) against *L. intracellularis*. Faeces were collected every 2-3 days for polymerase chain reaction (PCR) amplification of *L. intracellularis* DNA.

Clinical signs of PIA were evident in all four pigs inoculated with *L. intracellularis*. Diarrhoea was observed between 13 and 19 days post inoculation (pi), and the average weekly weight gain of inoculated pigs (3.7 ± 0.3 kg) was significantly lower than control pigs (4.6 ± 0.2 kg) at 21 days pi ($P<0.05$). *Lawsonia intracellularis* DNA was not amplified from the faeces of any pigs at day 0 pi, but was detected in all pigs inoculated with *L. intracellularis* between 10 and 24 days pi. Over the same period, *L. intracellularis* DNA was not detected in the faeces of uninoculated pigs. Serum IgG antibodies against *L. intracellularis* were detected in all of the inoculated pigs between 21 and 35 days pi, but not in any control pigs over the same time. Histopathology from the ileum and colon of one inoculated pig euthanased at 21 days pi showed recovering lesions of PIA when compared with tissue sections from an uninoculated pig.

At 10 weeks of age, the remaining nine pigs (three previously inoculated and six controls) were orally inoculated with the same source of *L. intracellularis* ($1x10^9$). Prior to inoculation, PCR amplification of faecal samples failed to detect *L. intracellularis* DNA in any pig. From 14 days pi onwards, *L. intracellularis* DNA was detected in five of six pigs inoculated for the first time, but was not detected in the faeces of any pigs previously infected with *L. intracellularis*. All pigs inoculated for the first time at 10 weeks of age developed an IgG response to *L. intracellularis* from 21-56 days pi. Serum IgG antibodies against *L. intracellularis* were not detected in pigs previously infected with *L. intracellularis* from 0-56 days pi. Clinical signs were not evident in any pigs.

Faecal PCR and serum IFAT results confirmed that the *L. intracellularis* inoculum extracted from homogenised PHE mucosa was able to infect pigs that had not been previously infected with *L. intracellularis*. However, no evidence of infection was detected in pigs with a previous *L. intracellularis* infection. Using a PCR assay of similar sensitivity, Jones *et al* (1993) demonstrated a strong correlation between the presence of *L. intracellularis* in faeces and the presence of microscopic PIA lesions. It would appear that pigs infected with *L. intracellularis* can recover naturally, and remain immune to further exposure by *L. intracellularis*.

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EFFECTS OF NATURAL PINE AND FIR TREE EXTRACTS GIVEN TO WEANER PIGS FROM 21 TO 84 DAYS OF AGE ON GROWTH PERFORMANCE AND SUBSEQUENT IMMUNE STATUS

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The world wide trend towards banning antibiotics in pork production will force the industry to follow more natural methods of production. Natural products derived from pine tree extracts are rich in natural antibiotics, fatty acids and vitamins. The present experiment was designed to investigate the effect of supplementing diets for weaner pigs with pine extracts on subsequent growth performance and immune response. Thirty-six Large White X Landrace boars from 12 litters were allocated at weaning at 21 days of age on a within litter basis to one of three dietary treatments. The treatments were a weaner diet containing 15.0 MJ DE/kg, 238 g CP/kg and 14.5 g lysine/kg, an adequate mineral/vitamin premix but no antibiotics and the same diet to which was added either 0.5 g/kg Bioeffective A or 0.2 g/kg Bioeffective V. Pigs were offered the diets *ad libitum* between 21 and 84 days of age. At the end of the growth study, haematological parameters and the increase in ear thickness in response to the mitogen, leucoagglutinin, were used as indicators of *in vivo* cellular immunity.

Table 1. The effect of Bioeffectives A and V on the growth performance of weaner pigs between 21 and 84 days of age, and immune response at 84 days of age.

	Treatment			
	Control	Bioeffective A	Bioeffective V	LSD
Growth rate (g/d)	635	650	640	60
Food intake (kg/d)	1.10	1.12	1.07	0.11
FCE	0.58	0.58	0.60	0.03
Increase in ear thickness (mm)	2.37	2.30	2.27	0.57
Neutrophils (x 10^6 cells/ml)	6.1	5.9	4.9	1.4
Eosinophils (x 10^6 cells/ml)	0.34	0.26	0.26	0.17
Lymphocytes (x 10^6 cells/ml)	10.1	12.2	11.0	2.8
Neutrophils/Lymphocytes	0.65	0.57	0.48	0.23

Supplementation of the weaner diets with Bioeffectives did not alter growth performance or the mitogen response (Table 1). However, during the experiment, pigs were moved at 56 days of age to larger individual pens in a different shed where hygiene and environmental conditions were less controlled. During the first 7 days after transition, the pigs fed Bioeffectives ate more (1074 vs 968 g/d, P=0.05) and tended to grow more quickly (685 vs 611 g/d, P=0.13). The major blood parameters were not significantly affected by dietary treatment although supplementation with Bioeffectives tended to modify the composition of white blood cells (Table 1). The neutrophil to lymphocyte (N/L) ratio is an indicator of stress in pigs (Yen and Pond, 1987) and the ratio tended to be lower (P=0.07) for the pooled Bioeffective treatments indicating that these animals may have been less stressed. Pooled data also showed that Bioeffectives reduced eosinophils (0.34 vs 0.26, P<0.05) which is an observation found in pigs treated with other immune enhancing dietary supplements (Dunshea and Ostrowska, 1999). The alterations in haematological parameters and reduced growth check during transition indicate that pigs receiving Bioeffectives may have been less stressed. The effects of these Bioeffectives should be examined further in other models in which animals are under greater stress challenges.

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PREVALENCE OF GASTRIC LESIONS IN WESTERN AUSTRALIAN PIG HERDS: AN ABATTOIR SURVEY

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Oesophagogastric ulceration (OGU) occurs commonly with prevalences at slaughter of between 1% and 50% (Palomo *et al.*, 1996), and is one of the most important causes of death in grower pigs (Love, 1981; Driesen *et al.*, 1987; Fogarty *et al.*, 1992). Abattoir surveys in Australia have found between 4.7% and 25% of pigs to have ulcers (Driesen *et al.*, 1987). The objective of this study was to determine the prevalence of OGU in pigs from Western Australia (WA) at a commercial abattoir. The hypothesis tested was that the prevalence of lesions would be similar to those reported in other Australian states.

Stomachs from 2232 pigs from 18 herds were examined with at least 40 stomachs included from each herd. These were cut along the greater curvature, washed, inverted, and examined within 30 minutes of exsanguination. There were no changes to the routine abattoir operation during this survey and pigs were held in lairage for 2 to 24 hours. Stomachs were scored as: 0 - normal; 1 - hyperkeratotic; 2 - eroded; or 3 - ulcerated (Christensen and Cullinane, 1990). Pigs were classified as porkers (head-off dressed weight <54.5 kg) or baconers (>54.5 kg). Comparisons between groups were analysed using the χ^2 test for independence.

The prevalence of OGU (Score 3) was 23.1% (range 0 - 75% in individual herds) (Table 1). When advanced lesions (score 2) were included the overall prevalence increased to 32.8% (range of herd prevalences of 0% to 82.7%). The prevalence of normal stomachs was 30.1%. No specific gender effects were found amongst a sample of 127 stomachs from 4 farms, both overall and for individual herds.

Advanced lesions (score 2 and 3) were significantly more common in porkers (47.5%) than in baconers (21%) (P<0.0001). In the single herd where both classes of pigs were examined the prevalence in porkers was also significantly higher than in baconers (71.4% and 29.2% respectively).

Table 1. Overall prevalence (%) and range of herd prevalences of oesophagogastric lesions in porkers and baconers at slaughter.

Score	Porkers (n=854)	Baconers (n=1378)	Total (n=2232)
0	22.8 (2.5-88.2)	32.2 (0.8-98.5)	30.1 (0.8-98.5)
1	29.4 (8.8-77.5)	46.8 (0-73.4)	42.2 (0-77.5)
2	11.4 (1.5-21.6)	7.7 (0-26.9)	9.7 (0-26.9)
3	36.1 (1.5-75.0)	13.3 (0-48)	23.1 (0-75)

As in other Australian states, gastric lesions were common in pigs in WA at slaughter. The prevalence of OGU in individual herds varied widely, so caution needs to be exercised when comparing this to other studies. An epidemiological survey is being carried out to identify risk and protective factors that may play a role in the development of OGU, including time off-food before slaughter. The impact of this condition on the pig productivity is also being evaluated.

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THE NUMBER OF VILLUS AND CRYPT CD4+ T CELLS IN THE JEJUNUM OF PIGLETS INCREASES AFTER WEANING

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Weaning of piglets is accompanied by marked changes in intestinal morphology, such as villous atrophy and crypt hyperplasia. Epithelial compromise may allow antigens to pass into the lamina propria where a localised inflammatory response can occur (McCracken *et al.*, 1999), resulting in decreased production. Dietary bovine immunoglobulin G (IgG) increases the IgG content in the piglet gut (Morel *et al.*, 1995), and may ameliorate the weaning check by enhancing gut immunity and function. This experiment tested the hypothesis that an IgG-fortified bovine colostrum powder (Immulac) fed before and for 24 h after weaning would reduce inflammatory responses associated with weaning.

At weaning (28 d), one randomly selected piglet was euthanased from each of 12 litters that were offered *ad libitum* either Immulac (780 g/kg CP, 75 g/kg IgG) in liquid form (200 g/kg DM) (n=6) or a starter diet (14.8 MJ DE/kg, 225 g/kg CP; n=6) for 21 days during the suckling period. Remaining piglets were weaned into conventional flat decks and fed the same diet that they had been offered as a supplement during the suckling period. After 24 h, another four piglets from each of the two dietary treatments were euthanased. Samples of jejunum were collected and processed, and populations of CD4+ and CD8+ T cells (indicators of the inflammatory response) were analysed and enumerated according to methods described by McCracken *et al.* (1999). Data were analysed by two-way ANOVA with time of sampling and diet as independent variables.

Table 1. Numbers of CD4+ T cells in the jejunum of pigs (cells per villus or per crypt).

Diet type	Solid		Immulac			Statistics[1]		
Weaning	Before	After	Before	After	SEM	Time (T)	Diet (D)	T x D
Villi	1.7	7.6	4.0	9.8	0.88	***	***	NS
Crypt	10.6[a]	19.8[c]	11.6[a]	15.9[b]	1.64	***	*	***

[1]NS, not significant, *P≤0.05, ***P≤0.001. [a,b,c]Values within rows with different superscripts are significantly different (P≤0.05).

Numbers of CD4+ T cells in villi increased after weaning (8.7 vs. 2.8, P<0.001) and were higher in pigs given Immulac (6.9 vs. 4.3, P<0.001). In the crypts an interaction (P<0.001) for CD4+ numbers occurred with more T cells found after weaning. However, feeding Immulac to piglets reduced T-cell proliferation by 25% compared to piglets fed the starter diet. There were no treatment differences in CD8+ T cell counts (data not shown). Increased numbers of CD4+ T cells in villi after weaning concurs with the work of McCracken *et al.* (1999), and may be attributed to low food intake after weaning (186 vs. 36 g DM/group in piglets receiving Immulac and starter diet, respectively). However these data did not support fully the hypothesis tested in this experiment, since villi and crypts responded differently to Immulac. The reason(s) for this are unknown, but may be related to the difference in supplement intake that occurred during lactation (294 vs 134 g DM/litter/day in piglets fed Immulac and starter diet, respectively). Alternatively, breakdown of the extracellular matrix that occurs after weaning and is associated with intestinal inflammation (McCracken *et al.*, 1999) may have a dietary component.

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A REVIEW - NUTRITIONAL CONSTRAINTS TO PIG PERFORMANCE AND PIG VARIABILITY

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Abstract

Although the average growth performance of pigs in commercial pork production has improved considerably over the past 20 years, there is still an appreciable gap between the potential growth performance of pigs and average production figures in commercial herds. Constraints to growth potential include genotype, voluntary feed intake, dietary protein intake and imposed feed restriction. Lifting these constraints will allow pigs to grow faster and more efficiently and reach slaughter weights at a younger age. Alternatively, most producers who overcome many of these constraints choose to slaughter their pigs at heavier body weights because it is usually a more profitable system of pork production. However, the range and natural variation in slaughter weight and backfat thickness are likely to become greater and more variable as producers go to heavier weights. The processor, on the other hand, requires a consistent carcass, preferably of similar weight and fat content. There must be a compromise between the wishes of the processor to have an even line of carcasses in terms of weight and fat content and what is possible by the producer taking into account the natural variation, management and sustained long term profitability of their production systems.

Introduction

Growth targets of 600g/day from birth to slaughter are now commonplace in commercial pork production. Producers regularly testing the growth performance of selected gilts observe growth rates well in excess of 600g/day. In addition, high health status herds also record growth rates much greater than 600g/d, particularly during cooler months.

Pork producers are not only aiming for these higher growth targets, but becoming increasingly aware of the need to reduce the variability in growth performance and carcass composition. Within a herd, genetic variation between individuals, season, sex and dietary and other management changes will all result in increased variability in growth performance and more importantly, carcass composition. This review will examine the potential performance of the growing pig and some of the constraints to both achieving this potential and reducing the variability of growth performance and carcass composition.

Potential growth performance of pigs

Growth rates and feed conversion efficiencies in commercial piggeries have improved considerably over recent years, particularly during the 1980s (Figure 1), and this has been associated with significant drops in the backfat of slaughter pigs.

However, there is still an appreciable gap between recent performance figures and the pig's potential for growth. The potential growth performances of pigs at various stages of the production cycle are compared to commercially feasible figures and the average figures from Meo and Cleary (1999) in Table 1. Data on the growth potential of pigs were collected from studies where nutritional intake was not limiting and male pigs available in Australia had been reared in individual pens under optimum environmental conditions. Data used to determine commercially feasible growth performance were from studies where pigs were reared in group pens under commercial conditions.

The biggest gaps between the potential growth performance of the pig and itsaverage performance or its commercially feasible performance occur in the early growth stages, particularly prior to weaning and in the immediate period after weaning. However, the average performance of pigs during the rest of the growing period is also often well below what is commercially feasible.

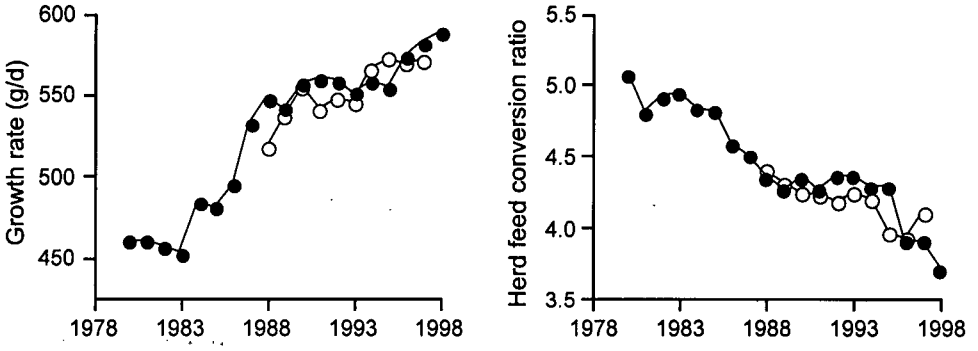

Figure 1. *The changes in growth rate and herd feed conversion ratio over the last 20 years as reported in the Victorian Pig Management Recording Service (●) (D.Treacy, personal communication) and PigStats (O) (Meo and Cleary, 1999).*

Table 1. Average commercial, feasible and potential growth performance of pigs at different stages of production

Age (d)	Average commercial growth[1]	Commercially feasible growth			Potential growth		
	Live weight (LW, kg)	LW (kg)	GR (g/d)	Reference	LW (kg)	GR (g/d)	Reference
0	1.5	1.5			1.5		
			285	King *et al.* (1998)		576	Hodge (1974)
25	6.8	8.6			15.9		
			250	Dunshea *et al.* (1997a)		576	Hodge (1974)
35	7.8	11.1			21.7		
			629	Campbell *et al.* (1995)		832	Hodge (1974)
55	17.8	23.7			38.3		
			770	Brewster *et al.* (1995)		1000	Mullan *et al.* (1997)
85	37.3	46.8			68.3		
			750	Brewster *et al.* (1995)		966	Brewster *et al.* (1995)
115	60.2	69.3			97.4		
			1066	Campbell (1999)		1376	King *et al.* (1997)
145	84.2	101.3			138.7		
				Overall Growth Rate			
	570 g/d		695 g/d			946g/d	

[1]Meo and Cleary (1999)

Contraints to growth potential

The objective in commercial pork production for pigs beyond about 20 kg live weight is to continue to achieve high voluntary feed intake so that the pigs can express their true potential for growth and lean deposition. The gap between potential growth rate and that expressed in commercial pork production declines with age and weight because often, voluntary feed intake approaches that required for reaching the potential protein deposition rate of the pig.

Improved genotypes

Previously, the growing pig's potential for protein deposition often lay within the limits of appetite (Standing Committee on Agriculture, 1987) and high levels of feeding caused excessive and variable fat deposition (Campbell *et al.*, 1985). For these older genotypes, reduction in both the amount and variation in fat content of carcasses to the level required by the processor could only be made in the short term by reduction of slaughter weight, restriction of feed intake or use of porcine somatotropin (Campbell *et al.*, 1991). However, associated with the development of new genotypes in Australia, King *et al.* (1997) demonstrated that protein deposition continues to respond linearly up to the limit of appetite in pigs grown to 120 kg live weight with the result that both male and females had acceptable carcasses. Thus, there are now pigs in Australia that are well suited to production systems that promote high voluntary feed intake. Efficiencies in pork production will be gained through promotion of heavier slaughter weights. But the processor must still be prepared to accept an increase in the depth and variation of backfat in the carcasses at these heavier slaughter weights. However, the variation in carcass backfat depth with the use of heavier genotypes at heavier slaughter weights will still be less than that for the older genotypes.

Voluntary feed intake

During the grower/finisher stages, voluntary feed intake is usually not constrained by dietary factors provided dietary energy content is in excess of about 14.5 MJ DE/kg (Henman *et al.*, 1999). Other dietary factors such as protein and amino acid balance, anti-nutritional factors, physical characteristics such as particle size, mash/pellet, availability of water, and feeder design and delivery may influence voluntary feed intake.

However, under commercial conditions where pigs are housed in groups, factors such as environment, social interactions and health status have much more influence over voluntary feed intake and constrain the growth potential during the later stages of growth. These non-nutritional constraints to feed will not be discussed here.

Dietary protein intake

Body fat content and backfat thickness of pigs decrease with increases in dietary protein up to that amount required for maximum body protein deposition. Even beyond the requirement for maximum protein deposition, fat may further decrease, accompanied by small increases in lean content (Campbell *et al.*, 1988). The protein and amino acid requirements have been well established for pigs of the various live weights, sexes and genotypes available in Australia and are incorporated into growth simulation models such as Auspig. Thus, information and tools are available to the commercial pork producer to ensure that they supply protein adequate diets to pigs and thus optimise growth performance and carcass quality.

The pig's requirement for amino acids changes very rapidly early in its growth period (Figure 2) and the use by commercial pork producers of only a small number of diets during the grower/finisher stage may limit the growth potential of the pig, affect fat deposition and affect overall profitability. For example, Brewster *et al.* (1995) reported that the dietary lysine content needed for near maximum growth performance between 63-84 days of age actually depressed the rate of gain in the 85-112 day period. It is likely

that the depression in growth rate resulted from dietary energy being diverted to synthesise and excrete the excess urea resulting from excess dietary protein and amino acids (Campbell *et al.*, 1988). Thus one common diet for the period between 63-112 days is no longer appropriate. These data support the concept of phase feeding as a further means of improving the efficiency and profitability of pig production. Phase feeding and the use of a greater number of diets will ensure that the dietary supply of protein and amino acids matches more closely the pig's requirement. This should ensure more consistent and predictable growth performance and carcass quality.

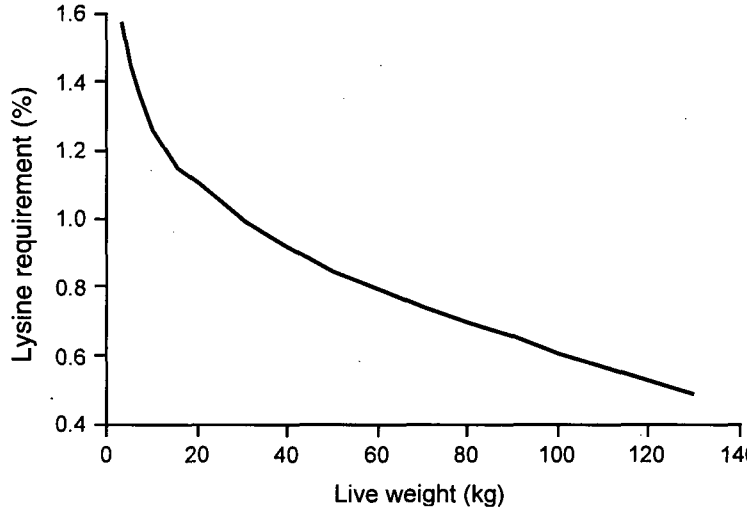

Figure 2. *The change in dietary lysine requirement with live weight of the pig (National Research Council, 1998)*

Other nutritional manipulation

Provided that voluntary feed intake is not constrained by nutritional factors and pigs have been offered protein-adequate diets throughout their growing period, the growth potential of the pig will be maximised. However, depending upon the genotype, the sex of the pig and live weight at slaughter, the resultant carcass fat thickness may be high and unacceptable to the processor. Porcine somatotropin has been used very successfully by pig producers to markedly reduce fat depth and increase carcass lean (Campbell *et al.*, 1991). There are other new technologies based upon nutritional manipulation that are also available to commercial pork producers to limit fat deposition in finisher pigs. Products such as chromium picolinate (Mooney and Cromwell, 1997; Lindemann *et al.*, 1993), betaine (Cadogan *et al.*, 1993), and conjugated linoleic acid (Dunshea *et al.*, 1998) have been effective to varying degrees in reducing carcass fat thickness to the satisfaction of the processor. These technologies that reduce the amount of body fat should also reduce the absolute range and thus the variation in backfat thickness of carcasses.

Feed restriction is also an effective means of reducing fat thickness but pig throughput and feed efficiency will suffer. Usually the reduction in feed efficiency and throughput will outweigh the advantages of a higher price for the leaner carcass and overall profitability will decline if feed restriction is used to meet the market specifications.

Consequences of increased slaughter weight

Because of the continued development of improved genotypes that are capable of meeting carcass specification at heavier slaughter weights, there has been a gradual increase in the slaughter weight of pigs in Australia to lower unit costs during both production and processing stages. Average slaughter weight has increased from 57.5 kg to over 70 kg since 1983 (Meo and Cleary, 1999).

Associated with this increase in slaughter weight there is the likely to be an increase in the variation in carcass and meat quality. Protein deposition increases with energy intake until a genetically determined upper limit to body protein deposition is reached (Campbell and Taverner, 1988). Previous studies have indicated that in pigs with high genetic capabilities for lean growth, the pigs upper limit to protein deposition cannot be reached below about 80 to 90 kg body weight (Campbell and Taverner, 1988; Rao and McCraken, 1991; Bikker, 1994). Thus increasing slaughter weight to above 100 kg may, for many pigs and particularly gilts and castrates, be beyond the pig's upper limit to protein deposition. In these cases where there is a linear/plateau relationship between protein deposition and energy intake, excess fat deposition will occur at high feed intakes. Not only will this increase the variation in fat deposition *per se* but, because of individual variation in feed intake under *ad libitum* feeding systems in commercial piggeries, the variation in fat deposition will increase even further. This may be best illustrated by examining the data of Campbell and Taverner (1988) who reported a linear relationship between protein deposition and energy intake for the better genotype, but a linear plateau relationship existed for castrates of a poorer genotype (Figure 3). At intakes between 33 to 47 MJ DE/day, protein deposition is relatively constant at about 85 g/day in the castrates of the poorer genotype, whereas the percentage of fat in the carcass varies by over 7 percentage units. However, if individual feed intake varies over a similar range of 33 to 47 MJ DE/day in pigs of the better genotype, there will be very little difference in the body fat content of the carcass.

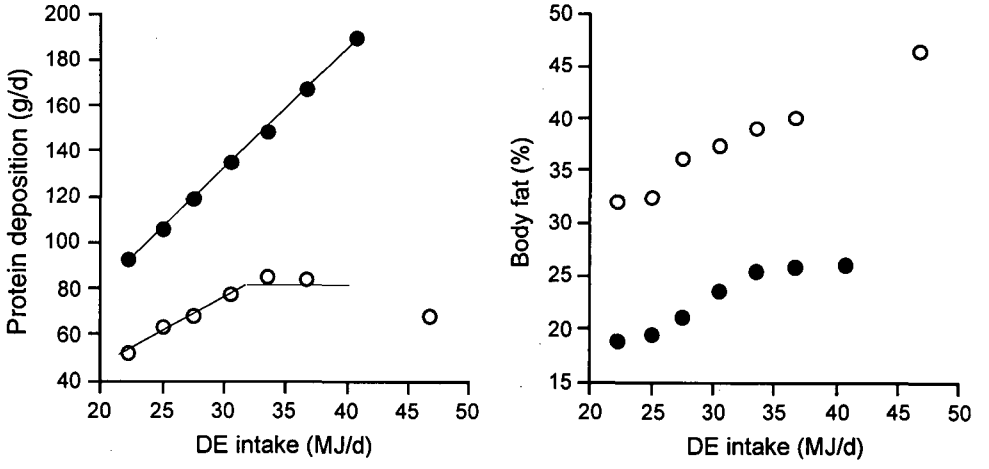

Figure 3. *The effects of DE intake on protein deposition and body fat of two genotypes, better genotype* (•) *and poorer genotype* (o) *(Campbell and Taverner, 1988).*

In situations where the producer experiences a linear/plateau relationship such as at heavier slaughter weights, there will be increased variation in the backfat depth and proportion of lean in the carcass because of individual differences in voluntary feed intake. Variation in the protein accretion curve for individual pigs will create even more variation in protein deposition rates and more importantly variation in backfat depths at heavier slaughter weights.

Table 2. The effect of age on the mean and variation in live weight (LW) of growing pigs kept in group pens.

Age (weeks)	14	15	16	17	18	19	20	21	22	23
LW (kg)										
Mean	49.0	53.1	58.2	65.7	71.1	77.8	84.4	91.7	98.8	105
s.d.	5.5	6.2	6.8	7.1	7.3	7.6	8.0	8.9	9.1	9.9
CV%	11.2	11.7	11.7	10.8	10.3	9.8	9.5	9.7	9.2	9.4

Protein accretion curves for individual pigs within a genotype are likely to diverge more as pigs get heavier in a similar manner to the protein accretion curves of different genotypes and sexes (Schinckel and de Lange, 1996). There are very few quantitative data in the literature on the change in variation in weight and backfat thickness of pigs at increasing slaughter weight. Data compiled in a study at Bunge Meat Industries, Corowa (F.R. Dunshea unpublished results), reveal that the variation in body weight increases quite markedly as body weight increases. Individual pigs in 12 pens, each of which contained 15 pigs, were weighed on a weekly basis between 14 and 23 weeks of age. The mean growth rate was 890 g/day and, although the coefficient of variation of live weight of groups of pigs within pens tended to decrease as the pigs got heavier, the absolute variation (sd) increased (Table 2). The range, or 2 standard deviations around the mean, of live weight for pigs at 20 weeks was only 16 kg whereas three weeks later the range increased to 20 kg. The range and natural variation in backfat thickness is likely to become even more as producers go to heavier slaughter weights. With all-in/all-out production systems, the processor must be prepared to accept this increased variation at heavier slaughter weights. Alternatively, the producer must develop a system of drafting heavier pigs on a weekly basis thereby disrupting growth rate, throughput and efficient use of grower space and, ultimately, increasing the cost of production.

Conclusion

In any pork production enterprise, there will be variation in pig growth performance and carcass quality; the better managed the operation, the less the variation between pigs. The processor requires a consistent carcass, preferably of similar weight and fat content. The production costs involved in the manipulation of the diet and intake and management of individual pigs to produce pigs of similar and desired weight and fatness would be quite high and unlikely to be economic to the producer. There must be a compromise between the wishes of the processor to have an even line of carcasses in terms of weight, fat level and meat quality and what is possible by the producer taking into account the natural variation, management and sustained long term profitability.

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AN IMPROVED METHOD FOR INDUCTION OF PORCINE ENZOOTIC PNEUMONIA VIA AEROSOL ADMINISTRATION OF IN VITRO CULTURED *MYCOPLASMA HYOPNEUMONIAE*

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In the past, experimental reproduction of porcine enzootic pneumonia (PEP) has relied on intranasal and intratracheal inoculations of *in vitro* cultured bacteria, contact with infected pigs or single/multiple intratracheal administration of a lung homogenate taken from infected pigs. These methods are somewhat cumbersome and have been used with inconsistent results. This paper presents the results of a series of studies in which a nebuliser system was used to reproduce PEP in previously unexposed piglets, via a single dose of *in vitro* cultured bacteria.

In two separate trials, 10 Large White x Landrace piglets, from a *Mycoplasma hyopneumoniae* (MHP)-free herd, were weaned 11-14 days post partum. They were randomly divided into experimental groups of five at one day post weaning, and placed, in twos or threes, into a purpose built chamber previously used for aerosol challenge of chickens with various respiratory pathogens (Whithear *et al.*, 1996). Compressed air was passed through a nebuliser containing the challenge material (either media control or 4th passage *in vitro* culture of the virulent Beaufort strain of MHP) at the rate of 15 l/min, creating an aerosol of the treatment, which passed through the chamber containing the animals before exiting via a HEPA filter. Rates of challenge were: Experiment 1, 244 colour changing units (ccu)/l air over 13 min; Experiment 2, 219 ccu/l air over 10 min. Feed and water were available *ad libitum* during the course of the experiments. Necropsies were performed 25 days after challenge. Serum samples collected immediately prior to inoculation and necropsy were analysed by Western Blotting against MHP LKR strain whole cell lysate.

Table 1. Weight gain per pig (mean ± SE) and total water consumption per group from challenge to necropsy, and lung pathology at necropsy.

	Experiment 1		Experiment 2	
	Control	Challenge	Control	Challenge
Lung Goodwin scores*	No lesions	[1]5, 0, 32, 18, 26	No lesions	15, 2, 7, 8, 24
Serology	All negative	All positive	All negative	All positive
Weight gain (kg/25 d)	7.2 ± 0.9	6.3 ± 0.4	6.7 ± 0.6	5.0 ± 0.6
Water consumption per group (l/25 d)	157.8	95.6	100.5	87.8

*Goodwin *et al.* (1969). [1]Individual scores for each pig in the group.

Pigs infected with MHP showed significantly lower weight gains (P=0.003, by analysis of variance; two-factor with replication) as compared to uninfected controls. Total water consumption was also depressed (Table 1), a possible indicator of decreased dry feed intake, which could not be recorded due to the isolator design. Lesions were histologically typical of MHP infection. These results demonstrate that significant disease and decreased growth performance due to MHP infection alone were inducible by the described method.

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EFFECT OF MILD PLEUROPNEUMONIA ON FEED INTAKE, GROWTH AND PLASMA CORTISOL IN GROWING PIGS

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An experimental endotracheal challenge model was established with the aim of inducing mild clinical and subclinical pleuropneumonia due to *Actinobacillus pleuropneumoniae* (App). This model was used to investigate the effects of these forms of the disease on individual feed intake, growth and hormonal changes in grower pigs.

Twenty pigs (mean live weight (LW) ± SEM, 41 ± 5.4 kg) were obtained from a commercial herd with no prior clinical or pathological evidence of pleuropneumonia or mycoplasmosis. Pigs were individually penned in two rooms maintained at 22°C, and offered a commercial, pelleted diet free of antibiotics. Indwelling ear vein catheters were inserted, and 2 d later all pigs were anaesthetised intravenously with xylazine/ketamine. Pigs (n=10) in one room received App serovar 1, strain HS54 (1×10^5 cfu) in 2 ml chilled culture broth endotracheally (App group); those in the second room received anaesthesia alone (sham group). Clinical response of each pig was monitored for 7 d post-challenge. Blood samples were collected without restraint at 0, 1, 2, 5, 6 and 7 d post-challenge, and plasma cortisol was measured by radioimmunoassay. Feed intake was monitored daily for 1 week before and 1 week after challenge. Feed intake and plasma cortisol concentrations were compared using repeated measures analyses. Pig LW were recorded 5 d before and 7 d after challenge. Four pigs from each group were slaughtered 7 d after challenge, and the remainder killed at 20 weeks of age. Pleuropneumonia was quantified by lesion weight as a proportion of total lung weight. One pig, with a rectal prolapse, was removed from the App group at 18 weeks.

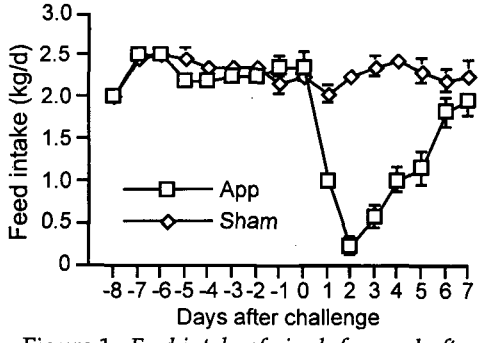

Figure 1. *Feed intake of pigs before and after either A. pleuropneumoniae or nil challenge.*

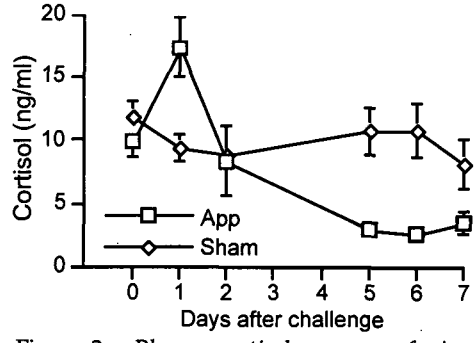

Figure 2. *Plasma cortisol response of pigs after A. pleuropneumoniae or nil challenge.*

All challenged pigs showed clinical signs of App within 1 day; half these animals recovered to clinical normality by 2 days after challenge, and all by 4 days. Lesions of necrotic pneumonia (ranging from 1-15% affected lung) were present in 7/9 App pigs examined while all 10 sham pigs were free of necrotic lesions. Challenge with App resulted in a feed intake depression of 50% over the first 7 days ($P<0.05$), reaching a maximum at day 2 of 90%. The App pigs were 5.8 kg lighter (49.5 ± 7.7 vs 55.3 ± 5.9 kg) at 7 days post-challenge, a mean weight gain difference of 6.8 kg over the 12 days of study. Plasma cortisol concentrations were initially elevated by 75% at day 1 (post-challenge) in pigs that received App ($P<0.05$), which corresponded to the period of maximum clinical disease. At days 5-7 post-challenge, challenged animals showed a 70% reduction in plasma cortisol compared to the sham group ($P<0.05$). Thus even mild forms of pleuropneumonia significantly reduce food intake over several days and induce a rise in plasma cortisol concentrations during clinical disease but a subsequent fall during the subclinical phase.

UTILIZATION OF THE ENERGY IN SOW'S MILK BY THE PIGLET

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Sow's milk is the major source of nutrients for sucking piglets. While little is known about its efficiency of utilization by the piglet (Noblet and Etienne, 1987), this knowledge is necessary to assess the growth response of the piglet to a changing energy supply. Therefore, this study was designed to evaluate the energy utilization of sows' milk by the piglet.

Twenty-four unsuckled Large White x Landrace x Piétrain piglets with an initial body weight (BW) of 1,544 ± 51 g (mean ± SEM) were used. Within a litter, four piglets were allocated to one of the four treatments: killed at birth, or bottle-fed sows' colostrum for 30 h and sows' milk thereafter for 6 d at the rate of 300 (H), 200 (I) or 100 (L) g of colostrum or milk/kg BW/d. Piglets were placed in individual cages at an ambient temperature of 32°C. Energy retained (ER) was determined on day 6 and day 7 by indirect calorimetry as the difference between metabolizable energy intake (ME) and heat production. Energy retained was also determined over the whole period using the comparative slaughter technique.

Table 1. Effect of the amount of sows' milk intake on energy balance and the partition of energy retained (ER) between protein and fat.

	Amount of milk intake				
	H	I	L	SEM	Significance[2]
Metabolizable energy intake[1]	1628^{A}	1077^{B}	521^{C}	18	L***
Heat production[1]	621^{A}	491^{B}	335^{C}	14	L***
ER as protein[1]	488^{A}	328^{B}	126^{C}	8	L***
ER as fat[1]	519^{A}	259^{B}	61^{C}	16	L***
Respiratory quotient	0.80^{a}	0.77^{b}	0.73^{c}	0.008	L***

[1]Data are expressed in kJ/kg $BW^{0.75}$/d. [2]L, linear effect of the amount of milk intake, ***P≤0.001. Within a row, means with a different superscript are significantly different, ^{A,B,C}P≤0.001, ^{a,b,c}P≤0.05.

Milk dry matter:weight gain ratio (g:g) was 0.67 ± 0.01 in piglets on treatment H. Energy digestibility, ME:gross energy intake ratio and N digestibility determined over day 4 to day 8 were independent of the amount of milk intake and averaged 0.985 ± 0.004, 0.969 ± 0.006 and 0.985 ± 0.004, respectively. The efficiency of ME for ER calculated by indirect calorimetry on day 6 and day 7 and by the comparative slaughter technique over the whole period provided values of 0.74 (± 0.01) and 0.72 (± 0.03), respectively. The mean value of 0.73 is similar to those of 0.69 and 0.71 reported for cows' and ewes' milk, respectively (Holmes and Davey, 1976; Degen and Young, 1982). Maintenance energy requirement amounted to 340 kJ ME/kg $BW^{0.75}$/d, which is lower than that of 544 kJ ME/kg $BW^{0.75}$/d calculated from the data of De Goey and Ewan (1975). The efficiency of ME for protein (ERp) and fat (ERf) deposition was calculated from the following model: $ME = C + 1/k_p\ ERp + 1/k_F\ ERf$ (Equation 1). However, the efficiency (k_F) of ME for fat deposition was not different from 1. Substituting k_F by 1.0, Equation 1 becomes: ME - ERf = 1.77 (± 0.06) ERp + 239, which corresponds to an efficiency of ME for protein synthesis of 0.56. Results of this study suggest that sows' milk is utilized with a high efficiency similar to that of cows' or ewes' milk.

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BOVINE COLOSTRUM SUPPLEMENTATION INCREASES VILLOUS HEIGHT IN SUCKING PIGLETS

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Oral administration of bovine colostrum products to piglets improve the health and absorptive capacity of the gut, this may be due to the high immunoglobulin (Ig) and growth factor content of colostrum. In the present experiment, the hypothesis that provision of supplementary colostrum (reconstituted spray-dried bovine colostrum "Immulac") in the diet of sucking piglets improves small intestine (SI) gut morphology was tested.

Thirty-six litters of Large white X Landrace piglets were allocated to three treatment (T) groups on the basis of parity and litter size. From days 7 to 28 of lactation, piglets were offered one of three diets in liquid form: Bovine whey protein concentrate (WPC), Immulac7.5 (IM1), and Immulac15 (IM2) (New Zealand Dairy Ingredients, Te Puke, NZ). Diets contained 0, 19 and 38 g/l IgG, and 0, 62 and 125 µg/l IGF-I, 5.5, 5.4 and 5.1 MJ/l gross energy, 1.1, 1.5 and 1.5 % ash, 0.025, 0.586, 0.025 % fat, 20.02, 12.64 and 18.45 % crude protein, respectively. On day 28, six piglets randomly selected from each treatment group were sedated and euthanased. Gut histological measurements were made according to the method of Pluske *et al.* (1996). Small intestine sites (S) referred to as S1, S2 and S3 correspond to the proximal jejunum, mid jejunum and distal ileum.

Table 1. Least square means of histological SI measurements in piglets fed supplementary liquid diets containing whey protein concentrate (WPC) or colostrum powder (Immulac).

	Treatment			Site				P-value		
	WPC	IM1	IM2	S1	S2	S3	SE	T	S	T x S
VH[1] (µm)	503[a]	601[b]	545[c]	649[a]	596[b]	405[c]	8	0.03	0.0001	0.08
CD[2] (µm)	196	195	190	201[a]	210[a]	171[b]	2.8	0.92	0.0001	0.88
ECH[3] (µm)	22.6[a]	24.9[b]	24.4[b]	23.4[a]	24.3[b]	24.3[b]	0.2	0.09	0.0032	0.56

[a,b,c]Values within treatment or site with different superscripts are significantly different ($P \leq 0.01$). [1]VH, villous height. [2]CD, crypt depth. [3]ECH, epithelial cell height.

Compared to the control diet (WPC), feeding Immulac resulted in significant increases in SI villous height and epithelial cell height (Table 1). There were significant differences among sites in the SI for VH, ECH and CD. The greatest response was observed with the IM1 treatment, which contained half the concentration of IgG and IGF-I found in IM2. This response may have resulted from the significantly greater intake of the IM1 diet, compared to WPC and IM2, recorded during the experiment ($P<0.05$). Least-square means for daily solid feed intake were 134, 294 and 163 (SE 42) g/litter/day for treatments WPC, IM1 and IM2, respectively. A T x S interaction was observed, with both Immulac treatments showing the greatest increase in VH when compared to the control at the S2 site (data not shown), however the interaction was not significant ($P=0.08$). Crypt depth was not different among treatment groups, which suggests the increase in VH may not result from increased mitotic activity in the crypts, but rather to a decrease in cell shedding from the villi. These results show a significant effect of colostrum supplementation on piglet gut morphology, which may translate to improved piglet growth and/or health (Pluske *et al.*, 1999).

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A BOVINE COLOSTRUM PRODUCT IN A WEANER DIET INCREASES GROWTH AND REDUCES DAYS TO SLAUGHTER

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Poor performance after weaning extends the number of days producers must feed and house their pigs until market weight. Much of this problem is associated with low voluntary food intake. The use of spray-dried plasma products, such as porcine and bovine plasma, in diets for weaner pigs generally increases food intake and growth rate after weaning (Hansen *et al.*, 1993). Some conjecture exists as to their mode(s) of action, although the IgG fraction of the plasma has been implicated (Weaver *et al.*, 1995). The aim of this study was to examine whether a bovine colostrum powder rich in IgG and added to a starter diet would increase post-weaning performance and reduce days to slaughter.

A total of 393 Large White x Landrace mixed-sex pigs (7.5 ± 0.02 kg live weight (LW)) weaned at 28 days of age on a 280-sow commercial piggery was used. Pigs received one of three experimental wheat-based diets: Diet Im0 (Control), and Diets Im50 and Im100 that contained 50 or 100 g/kg Immulac™, respectively. Immulac™ contained 150 g/kg IgG and 500 ng/g IGF-1. All diets were formulated to contain 14.8 MJ DE/kg and 12.6 g/kg available lysine. Pigs were housed in pens containing 20-24 pigs in environmentally-controlled weaner rooms, and were offered the diets *ad libitum* for 10 days after weaning. After this time, pigs on all treatments were offered the same series of diets through to slaughter at 83 kg LW. Data were analysed using the GLM procedures of SAS.

Table 1. Least-squares means for average daily gain (ADG, g), daily food intake (ADFI, g), feed conversion ratio (FCR) and number of days to slaughter of pigs fed three different diets for a period of 10 days immediately after weaning.

	Diet				
	Im0	Im50	Im100	Pooled SD	Statistics[1]
ADG 1-7	114[a]	161[b]	204[c]	19.3	*
ADG 8-14	212[a]	286[b]	292[b]	17.8	*
ADG 0-14	163[a]	223[b]	248[c]	10.9	**
DFI, d 1-7	184	206	230	51.0	NS
FCR, d 1-7	1.66	1.40	1.14	0.445	NS
Days to slaughter	121.6[a]	118.7[b]	117.4[b]	0.79	***

[1]NS, not significant, *P < 0.05, **P < 0.01, ***P < 0.001. [a,b,c]Values in the same rows with different superscripts are significantly different (P<0.05).

Pigs fed diets Im50 and Im100 grew faster (P<0.01) than pigs fed a similar diet in the first 14 days after weaning, but which was devoid of the colostrum product. Fastest growth was achieved in piglets consuming diet Im100. Pigs eating diets Im50 and Im100 consumed 12% and 25% more food, respectively, than pigs eating Diet Im0, however no statistical difference (P=0.472) in food intake was observed. Similarly, no significant difference (P=0.304) in FCR was seen among dietary groups. Pigs fed diets Im50 and Im100 took 2.9 and 4.2 days less (P<0.001), respectively, to reach a standardised slaughter live weight of 83 kg than pigs fed the Control diet. Inclusion of a bovine colostrum product in a starter diet improved post-weaning performance and reduced days to slaughter, and may be an alternative to spray-dried plasma products.

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THE EFFECT OF DIETARY CONJUGATED LINOLEIC ACID AND FAT ON PLASMA METABOLITES IN FINISHER PIGS

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Conjugated linoleic acid (CLA) has been shown to decrease body fat deposition in growing pigs (Ostrowska *et al.*, 1999a). However, the mechanism whereby CLA reduces fat accretion is unknown. Examination of the temporal response of metabolites involved in fat metabolism has provided valuable information on the mechanism of action of other growth regulators (Dunshea *et al.* 1992a). Therefore, the aim of this study was to determine the effect of dietary fat and CLA on metabolites involved in fat metabolism.

Twenty cross bred (Large White x Landrace) gilts (initial weight 65 kg) with venous catheters were fed either a low fat diet (25 g/kg) or a high fat (100 g/kg) diet with either 0 or 10 g/kg of CLA-55 (containing 55% CLA isomers) for 8 d. Palm oil was added to the high fat diet thereby ensuring that saturated fatty acids were predominant. While the diets had different digestible energy (DE) contents the lysine to DE ratios were similar (0.65 g available lysine/MJ DE). Pigs were fed every 3 h (90% *ad libitum* DE intake) for 8 d over which time pigs were bled frequently. Data were analysed by split-plot ANOVA.

Table 1. Effect of dietary fat and CLA on feed and DE intake and plasma constituents in finisher gilts. Data are the means over 8 days of feeding.

Fat (g/kg) (F)	25	100	100	25	Significance		
CLA-55 (g/kg) (C)	0	10	0	10	F	C	F x C
Rate of gain (kg/d)	0.98	0.84	0.84	0.92	0.77	0.77	0.27
DE intake (MJ/d)	37.5	35.4	35.4	34.4	0.32	0.11	0.87
Feed intake (kg/d)	2.54	2.40	2.28	2.17	0.006	0.11	0.82
Plasma NEFA (μmol/l)	78.8	88.0	123	130	0.001	0.015	0.65
Plasma glucose (mmol/l)	5.45	5.41	5.61	5.42	0.39	0.24	0.45
Plasma urea (mmol/l)	13.2	11.7	15.8	15.6	0.001	0.13	0.25
Plasma triglycerides (mg/dl)	21.1	23.7	24.8	26.7	0.001	0.008	0.65
Plasma insulin (mU/l)	15.9	18.0	12.0	13.9	0.04	0.30	0.96

Plasma nonesterified fatty acid (NEFA) concentrations were increased by dietary CLA suggesting an increase in fat mobilisation. However, the increase in fat mobilisation is equivalent to only a 6 g/d reduction in fat accretion (Dunshea *et al.*, 1992a,b), which is a small portion of the observed reduction of 86 g/d in pigs fed this level of CLA (Ostrowska *et al.*, 1999a). The increase in plasma triglyceride concentration suggests that CLA reduced fat accretion via decreased adipose tissue uptake of preformed fatty acids. Plasma glucose, the primary substrate for *de novo* lipogenesis, and plasma insulin was unaffected by dietary CLA suggesting that *de novo* lipogenesis was largely unaffected. Also, dietary CLA had no effect upon the ability of insulin to stimulate glucose removal (Ostrowska *et al.*, 1999b). In conclusion, it is proposed that dietary CLA reduces fat accretion via reduced lipogenesis from preformed fatty acids and increased lipolysis.

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PERFORMANCE OF ENTIRE, SURGICALLY CASTRATED AND IMMUNOLOGICALLY CASTRATED MALE PIGS

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The castration of boars ceased to be a management strategy within the Australian pig industry more than 30 years ago because of the production efficiencies associated with leaving males intact, especially in regard to average daily gain (ADG), depth of backfat (P2) and feed conversion efficiency (FCE). However, the industry has begun to question whether this practice is still appropriate because of the increased market opportunities in Asia and concern about boar taint. The aim of this experiment was to compare the performance of male pigs of two different genotypes that had been left entire, or which had been surgically or immunologically castrated.

Sixty crossbred male pigs were allocated to a 2 x 3 factorial design experiment. Main treatments were Genotype (A – lean with 50% Duroc bloodlines, and B – a propensity for increased fat deposition with <25% Duroc bloodlines) and Sex (entire male (EM), surgically castrated (SC) and immunologically castrated male (ICM)). Pigs were stratified to treatment according to litter of origin and live weight (LW) at weaning (15 days). Pigs were housed in individual pens from approximately 65 days of age and fed *ad libitum* a diet that supplied adequate energy and amino acids at all stages of growth to meet the animals' potential. Immunocastrated pigs were vaccinated (Improvac; Hennessy *et al.*, 1997) approximately 10 and 4 weeks prior to slaughter. Pigs were slaughtered at similar LW.

Table 1. The performance of entire male (EM), surgically castrated male (SCM) and immunologically castrated male (ICM) pigs of two different genotypes.

	Genotype (G)		Sex (S)			lsd		P values	
	A	B	EM	SCM	ICM	G	S	G	S
LW start (kg)	24.4	26.4	26.4	24.1	25.8	1.66	2.04	0.023	0.066
LW end (kg)	105.7	106.7	107.4	104.9	106.3	1.45	1.79	0.162	0.024
ADG (g)									
- total	856	931	906	881	899	27.3	33.6	0.001	0.332
- last 30 d	768	824	809	761	822	57.3	70.5	0.052	0.198
VFI[1] (kg/d)									
- total	2.38	2.42	2.36	2.44	2.40	0.106	0.130	0.529	0.482
- last 30 d	2.67	2.90	2.71	2.70	2.96	0.168	0.207	0.009	0.023
FCE									
- total	2.79	2.61	2.61	2.78	2.69	0.144	0.177	0.014	0.171
- last 30 d	2.50	3.21	2.80	2.83	2.99	0.230	0.282	0.001	0.355
P2 (mm)	13.8	17.1	15.0	15.4	16.2	1.54	1.88	0.001	0.386

[1]Voluntary food intake.

There were no significant interactions between genotype and sex. Overall, pigs of genotype A had a lower ADG and P2, but a higher FCE, than pigs of genotype B (Table 1). Castration had no significant effect on overall ADG, VFI or FCE. However, pigs that received the immunocastration vaccine consumed significantly more food during the 30 days prior to slaughter than either EM or SCM. Growth rates during the last 30 days prior to slaughter were comparatively poor which was attributed to deterioration in the flooring of the pens. The effect of castration on ADG and FCE may not be as great as it was with genotypes common to the industry several decades ago.

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PIG RESEARCH AND DEVELOPMENT CORPORATION

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FAT SOURCES FOR WEANER PIGS

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There is conflicting opinion on the value of feeding fats to weaner pigs. Frobish *et al.* (1970) found that fats of animal origin had better digestibility than those of vegetable origin, whereas Cera *et al.* (1989) found the opposite. This study was undertaken to investigate the effect of tallow, canola oil (canola) and a combination of both on growth performance, feed intake and feed:gain ratio for pigs growing from 8 to 20 kg.

The study was based on a 28 d randomised complete block design using 120 Large White x Landrace weaner pigs (60 male + 60 female), with a mean (± SD) starting weight of 7.8 ± 0.4 kg. The pigs were individually identified and allocated by sex and weight (15 pigs/treatment; two replications) to one of four dietary treatments. The dietary treatments were: (i) Control diet (40 g/kg crude fat; no canola or tallow added), (ii) 25 g/kg canola + 25 g/kg tallow, (iii) 50 g/kg canola, (iv) 50 g/kg tallow. All diets were formulated to contain 15 MJ DE/kg, 0.8 g available lysine/MJ DE, and 220 g/kg crude protein. Diets (ii), (iii) and (iv) contained 70 g/kg crude fat. Wheat was the major energy source for all diets. In addition to fat supplementation the major difference between diets was the level of soya bean meal. Inclusion levels were 190, 180, 160 and 210 g/kg for diets (i), (ii), (iii) and (iv) respectively. All diets were fed *ad libitum* as a mash, and pigs had free access to water. The pigs were housed in 2.25 x 1.9 m pens in an environmentally controlled room. The pigs were individually weighed at the start of the study, and then every 7 d. Feed usage (FU) was measured daily on a pen basis. From these data ADG, FU and feed:gain were calculated. As there were no sex effects all data within a treatment was pooled. Results are presented in Table 1.

Table 1. Effect of dietary fats on growth performance (average daily gain, ADG; feed:gain F:G) for weaner pigs from 8 to 20 kg live weight.

	Control (i)	C + T (ii)	Canola (C) (iii)	Tallow (T) (iv)	SEM	Statistics[1]
Number	30	30	30	30	-	-
ADG (g)	411.2[a]	382.5[b]	423.7[a]	426.2[a]	188.2	*
Mean feed usage (kg/pig)	13.2	13.6	12.3	13.6	3.2	NS
Mean weight gain (kg/pig)	11.5[a]	10.7[b]	11.8[a]	11.9[a]	1.4	*
F:G (kg:kg)	1.15[a]	1.27[b]	1.04[a]	1.14[a]	0.66	*

[a,b]Values within the same row with different superscripts are significantly different ($P \leq 0.05$). [1]NS, not significant; *$P \leq 0.05$.

The growth performance of pigs fed diet (i), (iii) and (iv) was similar. Feed usage of diet (ii) was low over the first 12 days. This resulted in a lower growth rate for pigs fed this diet. Over the final 10 days FU and ADG of these pigs improved. However, due to the severity of the initial set back final weight gain was well below that in the other treatments. The reduction in FU of pigs fed diet (iii) without a reduction in ADG resulted in improved feed efficiency, which was in line with expected results. Reductions in FU may result in an imbalance in the protein:energy ratio of a diet because of the greater utilization of dietary energy from fat relative to dietary protein utilization. The lack of differences in growth performance among pigs fed diets (i), (iii) or (iv) suggests that the protein:energy ratio was balanced. The possibility that negative interactions between ingredients in diet (ii) may have reduced palatability and/or digestibility needs further investigation. Before adding fat to weaner diets, factors such as the amount and type of fat, possible ingredient interactions and the protein:energy balance need to be considered.

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ALL GILT AND MIXED SEX LITTERS GROW FASTER THAN ALL BOAR LITTERS

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Many producers segregate pigs into sex groups from weaning onwards. There is interest in cross-fostering to produce single-sex litters prior to weaning. When sucking pigs are provided with supplemental milk it has been shown that boars grow more slowly than gilts (King *et al.*, 1998). Therefore, it is possible that boar piglets are less hungry than gilt piglets. If lower hunger also means lower sucking intensity then litters of all boars may not maximally stimulate sow milk production. This study was conducted to determine whether all boar litters grew more slowly than all gilt or mixed litters.

The experiment used litters of 32 Large White x Landrace sows. Within 24 h of farrowing, after piglets were suckled and received colostrum, the piglets were cross fostered to produce litters of either 10 boars (n=9), 10 gilts (n=11) or 5 boars and 5 gilts (n=12). Piglets were weighed at birth and at weekly intervals until 4 weeks of age. Data were analysed by ANOVA with the main effect being sex of the litter and with the sow as the experimental unit. To test the stated hypothesis a separate ANOVA was performed comparing all boar litters with pooled data from the mixed and all gilt litters.

Table 1. Effect of sex of litter on litter live weight (kg).

	Litter composition (L)					Significance	
	Boar	Gilt	Mixed	sed[A]	sed[B]	L[A]	B[B]
Birth	14.2	14.6	15.0	0.63	0.55	0.44	0.28
7 days of age	24.8	27.7	26.9	1.70	1.49	0.25	0.10
14 days of age	39.2	44.5	43.1	2.54	2.24	0.13	0.050
21 days of age	53.9	59.3	59.5	3.25	2.85	0.18	0.063
28 days of age	66.5	69.8	71.8	4.46	3.92	0.50	0.28

[A]Boar vs Gilt vs Mixed. [B]Boar vs (Gilt + Mixed).

Over the first 2 weeks of lactation the all boar litters tended to grow more slowly than the all gilt and mixed litters (1.78 *vs* 2.07 kg/d, P=0.063). Consequently, at 14 days of age the all boar litters were 10 % lighter than the all gilt and mixed litters (39.2 vs 43.8 kg, P=0.050). While the proportional difference in litter weight was maintained at 21 days of age (53.9 vs 59.4 kg, P=0.063) it was diminished by 28 days of age (66.5 vs 70.8 kg, P=0.28). A decrease in litter growth often occurs after 3 weeks of lactation (Cranwell *et al.*, 1995), particularly in large litters (Dunshea and Walton, 1995). Therefore, it is not surprising that the growth rate of the heavier litters containing gilts decreased over the latter stages of lactation. After they are born gilts are more active and suckle sooner than boars (Bate and Hacker, 1982). If the greater level of activity or hunger of gilts is sustained, as appears to be the case for litters receiving supplemental milk (King *et al.*, 1998), it is possible that gilts may provide a greater sucking pressure than boars. These data suggest that some gilts are required in a litter to maximise sow milk yield in early lactation.

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IMPROVED FEMALE PIG (30-100 KG) PERFORMANCE AND REDUCED N-EXCRETION BY OPTIMIZATION OF AMINO ACIDS AND PROTEIN SUPPLY

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Commercial production of a slaughter pig (30-100 kg live weight) in Denmark in 1995 was consistent with a total nitrogen loss of about 3.4 kg (Fernández *et al.*, 1999). Nitrogen excretion from pigs can be a factor limiting production considering that manure application is regulated by law to a maximum of 170 kg N/ha/year (140 kg N by 2002). Protein content in commercial diets can be reduced provided that diets are supplemented with the industrial amino acids (IAA) lysine (LYS), methionine, threonine and tryptophan to meet requirements. Supplementation of diets based on cereals and soya bean meal with IAA makes leucine (LEU) and valine the next limiting amino acids. Thus, reduction of dietary protein is limited by the requirement for LEU. The aim of this experiment was to determine whether dietary protein content could be decreased below the Danish recommendations for LEU (Fernández *et al.*, 1999) without impairing pig performance.

Three Landrace x Yorkshire pigs from each of 10 litters were subjected to three dietary treatments in two phases (30-60 and 60-100 kg live weight). Three diets consisting of wheat, barley and soya bean meal were formulated, on the basis of *in vitro* faecal and ileal digestibilities of energy and amino acids (Boisen and Fernández, 1995, 1997) respectively, and according to Danish recommendations for essential amino acids. The diets were: a) HL-HIAA, positive control (corresponds to commercial diets) high LEU (+5%), and high IAA (+5%); b) NL-NIAA, normal LEU and normal IAA; and c) LL-NIAA: low LEU (-14%) and normal IAA. All diets contained 14.1 MJ ME/kg (approx).

Table 1. Effect of protein and amino acid supply (mean consumption) on the growth performance (average daily gain, ADG; feed conversion ratio, FCR), meat % and protein utilization of female pigs grown from 30 to 100 kg live weight.

Diet	Intake (g ileal digestible/MJ ME)			ADG (g/d)	FCR (kg/kg)	Meat %	Diet protein/ gain (g/kg)
	Protein	LEU	LYS				
HL-HIAA	9.8	0.66	0.55	1039	2.34[a]	61	415[a]
NL-NIAA	9.5	0.63	0.54	1013	2.42[a]	60	409[a]
LL-NIAA	8.7	0.57	0.54	1067	2.22[b]	61	349[b]

[a,b]Values in the same column with different superscripts are statistically different ($P<0.05$).

In relation to average commercial pig production in Denmark (Fernández *et al.*, 1999), the mean ADG, FCR and dietary protein expenditure of the experimental pigs were different by +43%, -12% and -18%, respectively. There were no statistical differences between high and normal levels of LEU. Reduction of dietary protein below LEU recommendations resulted in better FCR (-120 to -200 g feed/kg gain), unchanged ADG and meat % and reduced dietary protein expenditure (-60 to -66 g/kg gain). These results indicate that the recommended levels of LEU (and possibly also of valine) are excessive. However, during the first phase (30-60 kg) pigs fed diet LL-NIAA showed inferior ADG and FCR compared to pigs fed the other two diets, which suggests that amino acid supply was deficient during the first phase but adequate during the second. To avoid deficiencies, it is necessary to analyse actual ingredients for nutrient content and availability before formulation. The average nitrogen excretion of pigs in this study was 2.4 kg, 30% lower than in commercial production in 1995. Dietary treatments (in the order given in Table 1) induced a reduction in N-excretion of 18%, 25% and 40%, respectively.

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MEASUREMENT OF FEED INTAKE IN GROUP HOUSED SOWS

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Feed intake during pregnancy, particularly the early stage, has been shown to be critical in maximising fertility. In group-housed sows fed a restricted amount of feed, individual feed intake is variable. However, no simple method has been available to quantify this variability. The aim of this study was to evaluate lithium (Li) as a marker for individual feed intake in group-housed sows.

Lithium ions are rapidly and almost completely absorbed from the gastrointestinal tract. The ion is initially distributed in the extracellular fluid compartment and then gradually distributes to other compartments (Goodman and Gilman, 1975).

Five multiparous mated sows were housed in individual pens and fed a commercial pelleted sow diet (2.5 kg/d). Early in the third week post-mating, jugular vein catheters were inserted via an ear vein. The following day, sows were fed 2.5 kg of feed containing 1g/kg LiCl. Blood samples were collected prior to feeding and at 30 min intervals for 9 h. Potassium EDTA was used as an anticoagulant and plasma separated by centrifugation. Lithium plasma concentrations (µg/ml) were determined by atomic absorption spectrometry.

Plasma Li concentrations increased over the 6 h following feeding, and then declined (Figure 1). As indicated by the SE bars, the plasma concentrations became much more variable at times greater than 6 h after feeding. It was decided for subsequent experiments to use the mean of 5 and 6 h plasma concentrations for correlation with feed intake.

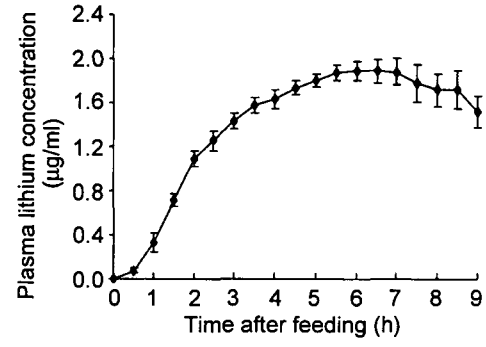

Figure 1. *Plasma Li concentration (mean ± SEM) in sows (n=5) after being fed 2.5 kg of a ration containing 1 g/kg LiCl.*

It was evident from the initial experiment that Li plasma concentration was influenced by body weight and eating duration. In a subsequent experiment, 20 mated sows, varying in body weight from 182 kg to 265 kg, were fed individually 2.5 kg of pelleted feed containing 1g/kg LiCl. These sows were sampled by jugular venipuncture 5 and 6 h postfeeding and a correction of plasma Li concentration, [Li] µg/ml, for body mass of the sow was developed by regressing body weight against the mean of the 5 and 6 h plasma Li concentrations (r^2=0.25, coeff.=-0.0054±0.002, Y=3.699-0.0054X, t=-2.71, P=0.013) such that:

Body mass adjusted [Li] = (Body weight - Mean body weight) x Regress. coeff + [Li]

In another experiment, groups of six mated sows were fed individually either 1.0, 1.75 or 3.25 kg of pelleted feed containing 1g/kg LiCl. The mean of five and six h plasma Li concentrations corrected for body weight when regressed against feed intake, gave an r^2 value of 0.948 and regression coefficient of 0.72±0.04 (Y=0.331+0.719X, t=16.59, P<0.0001). A formula for calculating feed intake was developed from this regression such that:

Feed intake = (Body mass adjusted [Li] – Y Intercept)/Regress.coeff.

Plasma Li concentrations following feeding a diet containing 1g/kg LiCl provided a relatively simple way of measuring feed consumed. In a group-fed situation, competition would reduce the variation in duration of feeding and so provide a more accurate measurement of variability in feed intake.

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RESPONSE OF MALE AND FEMALE FINISHER PIGS TO DIETARY ENERGY DENSITY

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Optimal nutritional management of "finisher" pigs is constrained by lack of quantitative information on the response of animals between 60 and 110 kg live weight (LW) to digestible energy (DE) content. Under "ideal" conditions modern genotypes appear to adjust average daily feed intake (ADFI) to maintain a constant DE intake over a much wider range of DE concentrations than was previously thought (Mullan *et al.*, 1998). However, under commercial pen conditions, voluntary feed intake is lower, and pigs respond in terms of both growth rate and feed conversion to DE density considerably higher than those thought to maximise biological and economic responses. The present study was designed to provide information on the response of finisher pigs to DE content found in commercial housing.

Five hundred female and male commercial crossbred pigs were allocated in a 2 x 5 factorial design with sex and DE density (12.0, 12.8, 13.6, 14.4 and 15.2 MJ DE/kg) as factors. Pigs were kept in commercial pens of 10 animals per pen (size 5.25 m^2). The facility was naturally ventilated with concrete floors and three-quarters of the floor area slatted. The diets were offered *ad libitum* to animals for six weeks commencing at 16 weeks. The pigs were slaughtered at 22 weeks of age. Growth performance and carcass traits were used to assess treatment effects.

Table 1. Effects of digestible energy density on growth performance of pigs offered feed *ad libitum* for 42 days from 112 days of age housed in commercial conditions.

DE[3] (MJDE/kg)	Start LW[4] (kg)	42 d LW[4] (kg)	Daily gain (kg/d)	Feed:gain	ADFI[5] (kg/d)	Carcass wt (kg)	Carcass P2 (mm)
12.0	63.1	96.1	0.815	3.08	2.46	74.27	11.4
12.8	63.2	98.6	0.876	2.92	2.54	76.31	11.7
13.6	63.0	100.8	0.935	2.78	2.59	78.69	12.6
14.4	63.3	101.8	0.952	2.65	2.51	80.50	13.4
15.2	63.1	101.7	0.952	2.59	2.45	80.53	13.5
Significance[1]		**	**	**	NS	**	**[2]
SEM[6]		0.6	0.014	0.04	0.03	0.54	0.25

[1]NS, Not significant, **P≤0.001. [2]Not significant when carcass weight is taken as covariant. [3]DE, Digestible Energy. [4]LW, Live Weight. [5]ADFI, average daily feed intake. [6]SEM, Standard error of the mean.

There were significant sex differences for all measurements except ADFI. There were no interactions between sex and DE for any measurement. The results showed that growth rate increased with increasing dietary energy density. There was a tendency for a plateau in growth rate at 14.4 MJ DE/kg which was consistent with the carcass weight results. Carcass P2 significantly increased with increasing DE density but was more a reflection of increasing carcass weight. There was no significant difference in ADFI across any of the energy densities, although there was a significant increase in feed efficiency at the highest energy density. The decrease in ADFI at the 15.2 MJ DE/kg may not have been real as the physical quality of this diet was very poor and feed wastage was observed to be higher for this diet. These findings are in contrast to the classical theory that increasing energy density would result in a consistent decline in ADFI. Feed intake maybe restricted more by physical or social rather than physiological constraints.

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INFLUENCE OF CANOLA OIL EXTRACTION METHOD ON THE PERFORMANCE OF GROWING PIGS FED DIETS CONTAINING THE RESULTING MEALS

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Three processes are currently employed in Australia to extract oil from canola meal – cold-pressing, expeller extraction and solvent extraction. These processes extract oil with different efficiencies and impart different amounts and durations of heat on the meal, which results in large variations in total fat, digestible energy and true ileal digestible amino acid content for growing pigs (van Barneveld, 1998). This experiment assessed the growth response of pigs (25-55 kg live weight, LW) fed diets containing canola meals derived using different extraction methods.

Cold-pressed, expeller-extracted and solvent-extracted canola meals were included in diets at 405 (diet 2), 435 (diet 3) and 420 (diet 4) g/kg, respectively. Soya bean meal was used as a control (290 g/kg; diet 1). Diets were formulated using a sugar and starch base to contain 0.40 g true ileal digestible lysine/MJ digestible energy (DE) and 15.0 MJ/kg DE. All other essential amino acids were added so that they were at least 30% in excess relative to lysine. A soya bean meal diet (diet 5) was formulated to 0.52 MJ true ileal digestible lysine/MJ DE to demonstrate that lysine was limiting in diets 1-4. Diet allocations to 20 male and 20 female Large White pigs housed in individual pens (1 x 1.5 m) were based on a randomised block design (8 pigs/diet). Diets were offered at 3 x maintenance ($1.5W^{0.75}$/diet DE) and water was offered *ad libitum*.

Table 1. Performance of pigs (25-55 kg live weight, LW) fed sugar/starch based diets containing cold pressed, expeller extracted and solvent extracted canola meals.

Diet†	Intake (kg/d)	LW gain (kg/d)	††EBW gain (kg/d)	†††FCR (on EBW)
1	1.52	0.752[a]	0.663[a]	2.30[a]
2	1.50	0.574[b]	0.510[b]	2.95[b]
3	1.57	0.673[c]	0.580[c]	2.70[c]
4	1.54	0.640[c]	0.568[c]	2.72[c]
5	1.52	0.718[a]	0.624[d]	2.44[a]
SEM	0.012	0.031	0.026	0.114
Statistics††††				
Diet	NS	***	***	***

†Diet 1, soya bean meal; Diet 2, cold-pressed canola; Diet 3, expeller-extracted canola; Diet 4, solvent extracted canola; Diet 5, soya bean meal + lysine. ††EBW, empty-body-weight. †††FCR, feed conversion ratio. ††††NS, not significant; ***P≤0.001. [a,b,c]Values in the same column with different superscripts are significantly different (P≤0.05).

Feed intake was not affected by canola meal type. The performance of pigs fed any form of canola meal was significantly lower than pigs fed diets containing soya bean meal (P<0.001). Pigs fed cold-pressed canola meal had significantly lower growth rates and higher feed conversion ratios (P<0.001) than pigs fed either expeller- or solvent-extracted canola meal. There was no difference in the growth performance of pigs fed diet 1 and diet 5 suggesting that lysine was not limiting in the test diets, possibly due to an overestimation of the true ileal digestible lysine content of the soya bean meal. Alternatively, factors other than lysine availability may be limiting pig production when canola meal of any type is included in diets at 400 g/kg or more. While canola meal represents a potentially valuable source of nutrients for pigs, the optimum amount to be included in the diet needs to be defined accounting for the method of oil extraction.

Supported by the Australian Oilseeds Federation

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PREDICTING THE DIGESTIBLE ENERGY CONTENT OF CEREALS FOR PIGS USING NEAR INFRARED SPECTROSCOPY

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Variation in the digestible energy (DE) content of feed grains has a significant influence on the efficiency of pig production (van Barneveld, 1999). Unfortunately, assessment of DE in grains for pigs currently involves *in vivo* analysis, making it unsuitable for use in routine diet formulations. The objective of this research was to evaluate the accuracy of two recently developed near infrared (NIR) spectroscopy calibrations (van Barneveld *et al.* 1999) applied to predict the DE content of a range of cereals with previously measured *in vivo* DE contents.

Two NIR calibrations for the prediction of DE in cereals for pigs were developed by van Barneveld *et al.* (1999) using a scanning monochromator (Model 6500, Foss-NIRSystems Inc., Silver Spring, MD, USA) and Intrasoft International (ISI) NIRS 3 version 4.0 software (Foss-NIRSystems Inc., Silver Spring, MD, USA). The first calibration was based on combined data for samples of whole barley, wheat, sorghum and maize, while the second was based on milled samples of the same. As the samples used in the development of these calibrations were derived from laboratories in Australia, Canada, France and New Zealand, indicator variables were used to detect the presence of, and compensate for, interlaboratory variability. In an attempt to validate this calibration, 35 cereal samples (19 barley, 10 wheat, 6 sorghum) were obtained from Australia (South Australia and southern NSW) and Canada (Saskatchewan). The DE content of all cereal samples was determined *in vivo*, in their country of origin, using total faeces collection methods. Samples were scanned using NIR at 2 nm intervals across the spectral range of 400-2500 nm and the predicted values compared with the measured *in vivo* values. Calibration statistics arising from this comparison are presented in Table 1.

Table 1. Near infrared spectroscopy calibration and validation statistics derived from assessment of the digestible energy (MJ/kg) content of whole and milled cereal samples fed to growing pigs.

Calibration	SECV[1]	SEP[2]	SECV/STD[3]
Whole grain	0.30	0.87	0.50
Milled grain	0.46	0.73	0.61

[1]SECV, standard error of cross-validation. [2]SEP, standard error of prediction. [3]STD, standard deviation.

The standard error of cross validation (SECV) values suggest that DE can be predicted to an accuracy of 0.30 MJ/kg and 0.46 MJ/kg for whole and milled grain, respectively. Despite this, the standard error of prediction is higher for this sample set. This may be due to several factors including between laboratory and between experiment differences in measured DE content of the test cereals, and the spread of DE values in the cereals employed for this validation. The cereals chosen for *in vivo* assessment tended to represent extreme values over the range of 11.5-17.5 MJ/kg and further validation of the calibration may be required using additional samples with an *in vivo* DE in the range of 13.0-14.0 MJ/kg. The SECV to standard deviation ratio provides a means of standardising the SECV enabling comparisons between different applications. While a ratio of 0.30 is desirable, the values achieved as part of this preliminary validation confirm the potential of this NIR calibration for predicting the DE content of cereals. The data for the cereal samples used in this validation process will be incorporated into the existing calibrations to enhance their accuracy and robustness and further validations will be completed using additional cereal samples.

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RELATIONSHIP BETWEEN PIG DIGESTIBLE ENERGY AND BROILER APPARENT METABOLISABLE ENERGY CONTENT OF BARLEY AND SORGHUM

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Digestible energy (DE) is one of the primary factors used to assess the nutritional quality of feed ingredients for pig diets. If diet formulations are to be accurately matched to pig requirements then cheaper, more rapid means of DE assessment are required. Apparent metabolizable energy (AME) is used as a measure of energy availability in broiler production systems, and is far easier to determine than DE for pigs. This experiment explored the relationship between AME for poultry and DE for pigs when measured on barley and sorghum grains.

The DE and AME content of 11 barley and 10 sorghum samples was measured using *in vivo* total faecal collection experiments. The DE content was determined using 84 Large White male pigs (25 kg) based on an allocation of 4 pigs per diet, with test diets comprising (g/kg): test grain, 940.05; dicalcium phosphate, 30; salt, 27.5; minerals, 0.7; vitamins, 0.5; choline chloride, 1; lysine, 0 5; and Celite®, 20. Ferric oxide was used to mark the start and end of the faecal collection periods. After a 7 day diet adaptation period, faeces were collected and weighed for 7 days, and then bulked, subsampled and dried for analysis. The AME content was determined using 480 broilers at an age of 15 days as described by Hughes and Zviedrans (1999). Each diet was fed to 4 cages of birds, with 3 female and 3 male birds in each cage. The diets included (g/kg): test grains, 800; HCl-treated casein, 134; dicalcium phosphate, 26; limestone, 11; vitamin and mineral premix, 5; sodium chloride, 3.5; choline chloride, 0.4 and Celite®, 20. Digestible energy and AME values were compared using paired comparison t-test.

Table 1. Coefficients for the regression y = digestible energy (DE) for pigs and x = apparent metabolizable energy (AME) for broiler chickens in barley and sorghum grains.

Grains	Coefficient	t value	P value (2 tail)	Range (AME)
Barley				
Intercept	10.293			
Slope	0.176	0.308	0.765	10.20-11.79
Sorghum				
Intercept	7.442			
Slope	0.456	1.146	0.285	13.50-14.08
All grains				
Intercept	6.474			
Slope	0.525	5.936	≤0.001	10.20-14.08

There was no statistical difference between the mean DE and AME content in sorghum samples (13.75 vs 13.81 MJ/kg, air-dry), but DE content was higher than AME ($P \leq 0.01$) in barley samples (12.23 vs 11.01 MJ/kg, air-dry). There were no significant correlations between DE and AME for barley or sorghum samples when considered separately (Table 1). When all samples were combined, the DE content was strongly correlated with AME with a multiple R of 0.806 and standard error of estimate of 0.582. Care should be taken when interpreting these values, however, as the spread between values for barley and sorghum is unrealistically enhancing the relationship between the measurements. Further research is required to quantify the relationship between DE and AME using samples with a wider range in DE and AME content. *Supported by the Grains Research and Development Corporation*

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RANGE IN DIGESTIBLE ENERGY AND TRUE ILEAL DIGESTIBLE LYSINE CONTENT OF AUSTRALIAN BARLEY SAMPLES

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Nutritionists have a limited ability to account for variation that can exist in the nutritional quality of feed ingredients (van Barneveld, 1999). To demonstrate the range in nutritional quality that can exist within a widely used feed grain such as barley in a single year, the digestible energy (DE) and true ileal digestible lysine (TIDL) content of 11 samples were assessed.

Eleven samples of barley were collected from across Australia representing 10 cultivars, one of which (Schooner) was obtained from two sites (Junee and Forbes). Diets were formulated to contain 940 g/kg of the test grains, the remainder consisting of dicalcium phosphate, salt, minerals, vitamins, choline chloride and celite®. The DE content of the samples was determined using 44 Large White male pigs (25 kg body weight, BW) based on an allocation of four pigs per diet and using total faeces collection techniques. True ileal digestible lysine was determined in four separate experiments (2-3 test grains/experiment) using Large White male pigs (35-40 kg BW) fitted with simple T-piece ileal cannulas. Tantangara barley was used as a control in each experiment and diets were provided based on a 5x5 Latin square design. The fifth diet contained enzymically-hydrolysed casein so that endogenous amino acid losses (mean values for all pigs used in the experiment) could be determined for true ileal digestibility calculations. Diets were fed for 7 d (3 x maintenance i.e., 0.5 x $BW^{0.75}$) prior to 8 h digesta collections over 2 consecutive days. Digestibility values from each experiment were compared using an analysis of variance with treatment means separated by least significant difference.

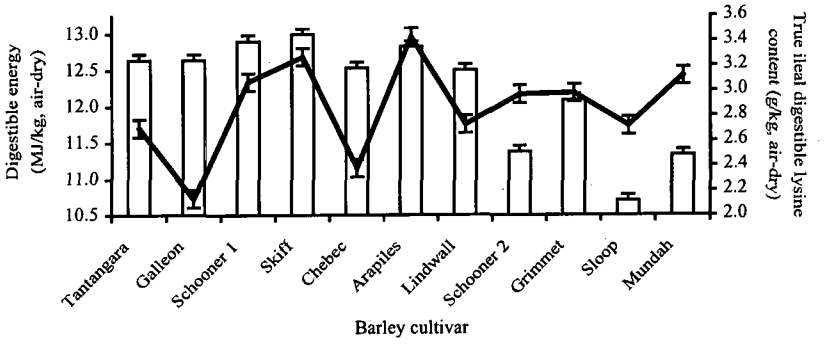

Figure 1. *Comparison of the digestible energy (DE, MJ/kg;□) and true ileal digestible (TID) lysine content (g/kg, —) of 11 samples of barley (Schooner 1, Junee; Schooner 2, Forbes).*

Barley DE values ranged from 10.70-12.99 MJ/kg, air-dry, representing a highly significant difference ($P \leq 0.001$) of 2.3 MJ/kg (Figure 1). Control barley TIDL values in one experiment were significantly different ($P > 0.05$) to values obtained in the other experiments and hence this data has been excluded. Values for TIDL varied significantly ($P \leq 0.001$) from 2.13-3.43 g/kg, air-dry, with no apparent relationship between TIDL and DE content of the grains. The level of variation observed in this experiment demonstrates that tabulated estimates of DE and TIDL content are far from adequate for diet formulations.

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NUTRITIONAL VALUE OF FROSTED WHEAT FOR WEANER PIGS

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The 1998 winter cereal crop in southern NSW was affected by a late October frost. Following harvest, significant quantities of frost-damaged grain were downgraded to feed grade. Previous studies in Australia have predicted the energy content of rain-damaged grain fed to growing pigs (Batterham *et al.*, 1980), however there is very little information on the nutritional value of frost-damaged grain for pigs. The objective of this study was to compare the growth performance of diets based on either normal or frost-damaged wheat when offered to weaner pigs.

Thirty hybrid (Large White x Landrace) pigs of both sexes were allocated at a mean (± SEM) live weight (LW) of 9.3 ± 0.40 kg to one of three mash diets. The three diets contained either normal wheat, frosted wheat or frosted wheat supplemented with blended vegetable oil. Digestible energy (DE) content was estimated by near-infrared reflectance spectrophotometry to be 14.2 and 12.6 MJ/kg for normal and frosted wheats respectively. Each diet was formulated to contain 0.8 g available lysine/MJ DE. The pigs were housed in individual weaner cages and maintained at 27°C and 60% relative humidity. The diets were offered *ad libitum* for 21 days. Feed intake and daily LW gain of individual pigs were recorded (Table 1). Water was available *ad libitum*.

Table 1. Diet composition and cost, and mean live performance of 30 weaner pigs fed diets based on either normal wheat, frosted wheat or frosted wheat plus feed oil.

	Wheat	Frosted	Frosted + oil	SEM	Probability[1]
Diet composition and cost					
Digestible energy (MJ/kg)	14.4	13.5	14.4		
Available lysine (g/MJDE)	0.8	0.8	0.8		
Fat (%)	3.7	5.4	8.8		
Least cost ($/t)[2]	270.17	231.24	273.18		
Live performance					
Daily feed intake (g)	710	629	632	35.1	0.16
Daily live weight gain (g)	521	467	476	24.9	0.18
Feed:gain	1.36	1.36	1.32	0.015	0.54
Feed cost of LW gain (c/kg)	36.7	36.0	31.4		

[1]Main effect of diet. No effect of sex or sex x diet. Start weight used as covariate.
[2]Ingredient costs current at time of experiment.

There were no significant differences in daily gain, feed intake or feed:gain among the experimental diets. Compared to the frosted wheat diet, there was a trend for pigs fed the normal wheat diet to eat more (710 vs 629 g/d P=0.16) and grow faster (521 vs 467 g/d P=0.18). Addition of vegetable oil to frosted wheat did not improve daily gain significantly (476 v 467 g; P=0.16).

The results indicate that frosted wheat has the potential for inclusion in pig diets. However, it may be expected that under commercial conditions, frosted wheat could have a greater impact on pig growth performance than under the present "ideal" conditions.

The economics of using frosted wheat rely heavily on ingredient prices. Given the ingredient prices (normal wheat $110/t, frosted wheat $90/t, feed oil $800/t) and growth performance in the present experiment, frosted wheat supported pig growth at a lower cost (c/kg LW gain) than the normal wheat or frosted wheat plus oil diets. Under these conditions frosted wheat should be considered in least cost diet formulations.

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EFFECT OF GENOTYPE ON APPARENT FAECAL ENERGY DIGESTIBILITY IN THE GROWING PIG

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The influence of genotype on the digestibility of nutrients and energy in the growing pig is unclear. Higher dietary energy digestibilities have been reported for both obese pigs (Sundstøl *et al.*, 1979) and lean genotypes (Wenk and Morel, 1985). Quin *et al.* (1995) attributed differences in digestion among pigs of different genetic backgrounds mainly to differences in fibre digestion.

The objective of this study was to determine energy digestibility in two diets varying in crude fibre content, with pigs of an improved genotype (Large White x Landrace, LWxLR) and an unimproved wild genotype (Kune-Kune, KK). The Kune-Kune pig is believed to have originated from Asia or China (Clarke and Dzieciolowski, 1991). Kune-Kune pigs are small in stature, rotund and have a characteristic pair of neck tassels. Six entire male pigs of each breed, of a similar degree of maturity were used. The LW x LR pigs (54.0 ± 1.2 kg body weight, BW, mean ± SE) were four months old and the Kune-Kune pigs (19.0 ± 1.6 kg BW) were three months old. All pigs were housed in metabolism crates and following a seven-day adaptation period, pigs were fed each of the experimental diets (D) for ten days, in a crossover design. All faeces were collected from day 6 to day 10 (total collection method). The chemical composition of the wheat and wheat by-product based diets (on an as fed basis) were: 15.75 and 16.38 MJ gross energy/kg, 9.6 and 21.9 % hemicellulose, 1.8 and 6.6 % cellulose and 1.0 and 2.8 % lignin, respectively.

Table 1. Least-squares means for apparent faecal energy (DEc) and organic matter (DOMc) digestibilities for pigs of two genotypes (GEN) fed two diets (D).

	Wheat		Wheat by-product			Significance[1]		
%	LWxLR	KK	LWxLR	KK	SE	GEN	D	GENxD
DEc	87.9[a]	87.1[a]	69.4[c]	71.3[b]	0.34	NS	***	***
DOMc	90.9[a]	90.3[a]	73.5[c]	75.3[b]	0.24	NS	***	***

[1]NS, non significant, ***P<0.001. [a,b,c]Values within rows with different superscripts are significantly different (P<0.05).

The effect of diet (D) and the interaction between diet and genotype (GENxD) were statistically significant (P<0.001) for both DEc and DOMc, while the overall effect of genotype (GEN) was not significant (Table 1). The wheat diet was better digested by both genotypes. A closer examination of the interaction GENxD showed that for both DEc and DOMc there were no differences between breeds for the low fibre diet (wheat). However, the Kune-Kune pigs digested the high fibre diet (wheat by-product) better. It is concluded that differences in dietary energy digestibility exist between extreme genotypes of pig when fed a high fibre diet.

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COMPARISON OF THREE METHODS FOR DETERMINING ENDOGENOUS ILEAL PROTEIN FLOW IN THE GROWING PIG

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It is important to be able to accurately determine the amount of protein of endogenous origin in ileal digesta when calculating true ileal amino acid digestibility and several methods have been developed for this purpose. The enzyme hydrolysed protein (EHP, Moughan *et al.*, 1990), isotope dilution (ID, Souffrant *et al.*, 1981) and guanidination (GU, Rutherfurd and Moughan, 1990) methods allow the determination of endogenous ileal protein flow under the physiologically normal condition whereby dietary peptides are present in the gut.

The EHP method allows accurate determination of endogenous ileal nitrogen (EN) and lysine (Elys) flows for the special case of feeding a hydrolysed-casein based diet to animals, with the proviso that there may be some underestimation due to loss of endogenous amino acids in the ultrafiltrate. The EN and Elys flows determined using the EHP method were accepted as a suitable baseline and comparison was made with the ID and GU methods, respectively. Comparison among methods was made within the same pigs fed the same diet.

Casein, which was previously labelled with ^{15}N, was treated with O-methylisourea to transform lysine into homoarginine, and then enzymatically hydrolysed (MW < 5,000 Da). A diet was prepared (test diet) where the sole N source was the enzymatically hydrolysed, guanidinated, ^{15}N-labelled casein at an inclusion level of 10%. A diet was also prepared (preliminary diet) which contained 10% enzyme hydrolysed casein (MW < 5,000 Da) as the sole source of N. The diets were given to Landrace x Large White entire male pigs (n=6; bodyweight 19.2 $\pm$ 0.5 kg, mean $\pm$ SEM) at 10% metabolic body weight ($W^{0.75}$) per day. The preliminary diet was given to the pigs in hourly meals over 8 hours per day for 7 d. On the following day, the pigs were given the test diet in hourly meals for 8 hours. Nine hours after the first meal of the day, the pigs were anaesthetised and digesta were sampled at the terminal ileum. Flows of EN were determined using the EHP and ID methods. Flows of Elys were also determined directly using the EHP and GU methods. Data were analysed using paired t-test comparisons and the EN and Elys flows are shown in Table 1.

Table 1. Mean (± SEM) endogenous ileal N and lysine flows (μg/g DMI) determined using the enzyme hydrolysed protein, guanidination and isotope dilution methods in the growing pig.

Method	Lysine	Nitrogen
Enzyme hydrolysed protein	287 ± 70	1543 ± 262
Guanidination	214 ± 73	-
Isotope dilution	-	1170 ± 201
Significance	P < 0.01	NS

Although the difference for the Elys flow between the EHP and GU methods was proportionally similar to that for the EN flow between the EHP and ID methods, only the EN difference attained significance (P<0.01). Given that the EHP method is expected to slightly underestimate EN flow, the ID method may also underestimate EN flow. The GU method appears to underestimate Elys flow to a greater extent than the EHP method.

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ISOLATED LUPIN NON-STARCH POLYSACCHARIDES IN SORGHUM-BASED DIETS DO NOT INFLUENCE ENDOGENOUS NITROGEN LOSSES FROM GROWING PIGS

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The addition of isolated lupin non-starch polysaccharides (NSP) to pig diets at concentrations above 100 g/kg has been shown to significantly increase digesta viscosity and decrease the apparent ileal digestibility of amino acids (van Barneveld *et al.* 1995). The NSP digestion was minimal, with no significant difference found among diets when graded amounts of NSP were added. The observed responses may be due to changes in endogenous nitrogen (N) secretions within the pig gut. The aim of this experiment was to examine the influence of lupin NSP isolate on endogenous N losses in growing pigs.

Isolated NSP from dehulled *Lupinus angustifolius* cv Gungurru was prepared using proprietary methods (CSIRO Food Science, North Ryde, NSW) to a 95% purity. It was included in a sorghum/casein based diet at concentrations of 0, 60 or 120 g/kg at the expense of starch. Celite® and n-hexatriacontane were added as indigestible markers. True ileal amino acid digestibility of the diets was determined using three Large White male pigs (35-40kg) fitted with simple T-piece ileal cannulas. Also fitted were two cephalic vein catheters, externalised in the centre of the back. Pigs were allocated to each diet using a 3 x 3 Latin square design with diets being fed for 5 d prior to 8 h digesta collections over two consecutive days for each collection period. A continuous intravenous infusion of ^{15}N-labelled amino acids (Gannon and Reeds, 1995) was administered at a rate of 1 ml/h for the entire duration of the experiment. A blood sample was taken daily at 0800 hours and during digesta collections, for determination of plasma free amino acid ^{15}N enrichment as an estimate of the enrichment of the precursor pool for endogenous protein synthesis. At the end of the experiment, following euthanasia, samples of urine, liver, pancreas, spleen, heart, and intestine were taken to determine relative sites of endogenous protein synthesis. Plasma, digesta and mucosa ^{15}N enrichment was determined using a Europa gas-isotope-ratio mass spectrometer.

Table 1. Effect of isolated lupin non-starch polysaccharide (NSP) inclusion on endogenous protein loss measured using an ^{15}N isotope dilution technique.

^{15}N Ratio	Dietary NSP content (g/kg)			Statistics		
	0	60	120	Diet	SED	LSD
Digesta/Plasma	35.3	25.2	27.6	0.38	6.86	17.64
Digesta/Mucosa	22.5	38.7	42.0	0.27	10.10	32.13

SED, standard error of the difference; LSD, least significant difference.

The mean ^{15}N ratio for Digesta/Plasma was 29.4 and for Digesta/Mucosa was 34.4 (Table 1), suggesting that approximately one third of all the N in the digesta was of endogenous origin. Inclusion of isolated lupin NSP had no effect on endogenous protein loss in growing pigs at concentrations up to 120 g/kg, regardless of whether the ^{15}N enrichment in plasma or mucosa was used as the precursor pool to determine the digesta enrichment:precursor pool enrichment value. The results suggest that factors other than endogenous N losses are responsible for reductions in apparent ileal amino acid digestibility in diets with high lupin NSP content.

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EFFECT OF DRY MATTER INTAKE ON SOME COMPONENTS OF ENDOGENOUS PROTEIN IN ILEAL DIGESTA

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The effects of dry matter intake (DMI) on endogenous protein secretion are equivocal. For instance, Butts *et al.* (1993) reported that the endogenous protein content of ileal digesta increased with dry matter intake. In contrast, Furuya and Kaji (1991) found a significant decrease in endogenous protein amount in ileal digesta with DMI. However, both these research groups used purified diets and only estimated total endogenous protein content. The aim of this experiment was to determine whether DMI affected the amount of some major individual endogenous protein components in the ileal digesta of pigs fed a commercial diet.

Four Large WhitexLandrace boars (35 kg) were surgically fitted with a simple T-piece cannulae 20 cm anterior to the ileo-caecal valve 10 days before the study began. The pigs were fed a wheat and soya bean meal based diet formulated to contain 230 g protein/kg, 14.3 MJ DE/kg and 2 g/kg chromic oxide as an indigestible marker. The experimental design was a 4 × 4 Latin Square with treatments being feed intakes of 40, 60, 80 and 100% of the previously determined mean *ad libitum* intake (1604 g/d. Each daily feed allowance was divided into eight meals that were offered every 3 hours. Pigs were fed each level for 5 days and on the fifth day an eight-hour ileal digesta collection was conducted. Mucin, mucosal cell, bacteria and trypsin protein were measured in samples of ileal digesta. The effect of DMI on the level of each endogenous protein component was analysed for linear and quadratic responses.

Table 1. Effect of dry matter intake (DMI) on individual endogenous protein (EP) components and sum of the measured endogenous protein components.

	DMI (% of *ad libitum*)					Significance		
EP component	40	60	80	100	SED	DMI	Linear	Quadratic
Mucin protein[1]	1.6	1.0	1.2	1.1	0.22	0.131	0.152	0.122
Mucosal cell protein[1]	11	8	8	10	1.9	0.357	0.732	0.110
Bacterial protein[1]	0.91	0.73	0.66	0.75	0.082	0.123	0.098	0.069
Trypsin protein[1]	5.5	4.9	5.5	5.6	1.62	0.974	0.860	0.792
Sum of measured EP components[1]	19	15	15	18	3.1	0.528	0.775	0.180
Sum of measured EP components (g/d)	12	15	19	29	3.7	0.023	0.005	0.233

[1]g/kg DMI.

The ileal flow of each endogenous protein component increased with DMI. However, as a proportion of DMI, each endogenous protein component and the total endogenous protein were less responsive to changes in intake. However, there was a clear indication that the flows were higher at the lowest amount of DMI when expressed on a per unit intake basis, at least for some of the endogenous protein components. The data suggests that restricting DMI will lead to a higher proportion of some endogenous protein components in ileal digesta. The findings are particularly pertinent to breeding animals, which are often restricted to low levels of DMI.

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PIG RESEARCH AND DEVELOPMENT CORPORATION

FEEDING OF LIQUID MILK SUPPLEMENTS TO PIGS PRE- AND POST-WEANING IMPROVES LIVE WEIGHT GAIN

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Under current feeding strategies, pigs do not reach their potential growth rates before weaning (Harrell *et al.*, 1993). After the first week of lactation sow milk yield and composition can limit piglet growth. This affects the lifetime pig performance and profitability by increasing the number of days until slaughter (Mahan and Lepine, 1991). A study was conducted to determine the effects on performance of supplementing the diet of piglets pre- and post-weaning with a number of different milk-based liquid products.

A total of 359 Large White x Landrace piglets from forty litters was used in the experiment. The sow/litter units were allocated to one of four treatments: Control (C) where no milk supplement was fed; whole milk (WM) (250 g/kg crude protein (CP), 230 g/kg fat); Veanavite (piglet) powdered milk (PM) (250 g/kg CP, 150 g/kg fat) as per manufacturer's recommendations; and reject ice cream (IC) (130 g/kg CP, 320 g/kg fat) (all nutrient analyses on DM basis). The pigs were fed the supplements *ad libitum* (provided twice daily) for one week prior to weaning at 19 days of age. At weaning each litter was stratified on the basis of live weight and allocated to two (mixed) groups of the same average live weight. One group continued to receive the same supplement as before weaning (e.g., WM/WM) while milk supplementation for the other group was withdrawn (e.g., WM/C). Supplement intake was measured daily (pre- and post-weaning) and the piglets were weighed weekly. Data were analysed using analysis of variance, based on a 3 x 2 factorial design with added control.

Table 1. Effect of supplementing the diet of piglets with various milk products pre- and post-weaning on average live weight (kg).

Treatment	Live weight (kg)				Supplement intake (g DM/pig/d ± SE)	
	Pre-weaning (12 days)	Weaning (19 days)	1 wk post weaning	2 wk post weaning	Pre-weaning	1 wk post weaning
Control	4.75	6.83	7.14[a]	8.86[a]		
WM/WM	4.74	6.83	7.59[d]	9.30[bc]	332	909
WM/Control	4.74	6.83	7.45[c]	9.30[bc]		
PM/PM	4.70	6.86	7.94[e]	9.69[c]	263	1004
PM/Control	4.70	6.86	7.39[bcd]	9.26[bc]		
IC/IC	4.74	6.58	6.91[a]	8.68[a]	313	970
IC/Control	4.74	6.58	7.21[abc]	9.11[ab]		
SED	0.328	0.149	0.163	0.28	23	39

[1]Values in the same column with different superscripts are significantly different (P≤0.05).

There was no significant difference in supplement intake either pre- or post-weaning. Piglets fed either WM or PM both pre- and post-weaning were significantly heavier at one or two weeks post-weaning compared to those who received no milk supplementation (Table 1). Ice cream was not a suitable supplement (probably due to its low CP content) as the live weight of piglets was not significantly different to that of the controls. Feeding milk supplements to piglets pre- and post-weaning (rather than just pre-weaning) can have a beneficial effect on piglet live weight, and this could be expected to have longer term benefits.

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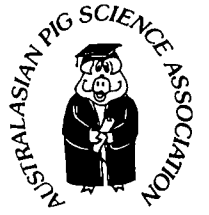

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THE EFFECT OF PIGLET WEIGHT ON PASSIVELY DERIVED ANTIBODIES TO *ACTINOBACILLUS PLEUROPNEUMONIAE* SEROVAR 1

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"Tail-enders", i.e., pigs of lower than average birth or weaning weight, often cause dilemmas for producers contemplating segregated early weaning (SEW) to control respiratory disease in herds. With SEW, piglets are weaned, usually less than 16 days of age, while they have high colostral immunity to protect them from cross infection from the sow. Many producers either destroy underweight piglets or sell them to specialist growing units as they fear these piglets have not received sufficient colostrum to be protected, and therefore pose an increased risk of transferring the disease to grower sites. The removal of "tail-enders" may be unnecessary because the interaction between weight and antibody concentrations for pigs in a SEW system has not been assessed. This experiment examined the relationship between piglet antibody concentrations and bodyweight.

A group of seven crossbred sows were vaccinated with an *Actinobacillus pleuropneumoniae* (App) vaccine containing App serovar 1 antigens and exotoxins at seven and three weeks prior to farrowing. Their progeny were blood sampled and weighed at one week of age. A group of these piglets were weaned at 14 to 16 days of age and allocated into two treatment groups. The "heavy" (H) group, (n=18) all had initial trial weights greater than 2.2 kg, whilst the "light" (L) group (n=19) all had an initial trial weights of less than 2.2 kg. The two groups were housed in the same isolation room. Piglets were weighed and blood sampled at 5, 7, 9 and 12 weeks of age and had a final blood sample taken at 14 weeks of age. Blood samples were analysed using an ELISA for antibodies to ApxI haemolysin, a major virulence factor of App (Prideaux *et al.*, 1998). Results of the serological analyses and pig weights were compared by analysis of variance.

Mean ApxI optical densities (OD's) (Table 1) between the two groups did not differ significantly at any time. The range of antibody concentrations between groups was also very similar. The H group of pigs were significantly heavier than the L group at all recorded instances ($P<0.05$) with the exception of weights recorded at 9 weeks of age (Table 1).

Table 1. Mean ApxI OD's and weights of pig weight groups from 1 - 14 weeks of age.

	Mean ApxI OD's[1]			Mean weight (kg)[1]		
Age (weeks)	H n=19	L n=17	$LSD_{(5\%)}$	H n=19	L n=17	$LSD_{(5\%)}$
1	1.687	1.731	0.246	2.73[a]	1.89[b]	0.202
2	n/a[2]	n/a	n/a	4.59[a]	4.05[b]	0.454
5	1.524	1.541	0.404	9.940[a]	8.54[b]	0.834
7	0.397	0.492	0.159	19[a]	17.32[b]	1.593
9	0.222	0.210	0.096	29.91	27.82	2.467
12	0.078	0.141	0.081	53.39[a]	49.40[b]	3.273
14	0.055	0.077	0.033	n/a[2]	n/a	n/a

[1]Values in same row with different superscripts are significantly different ($P\leq0.05$). [2]n/a-results not available.

Piglet weight is not an important influence on the initial peak levels and subsequent subsidence rates of maternally derived immunity. This finding should allow a more rational approach in dealing with smaller pigs in SEW systems.

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INFECTION WITH *ACTINOBACILLUS PLEUROPNEUMONIAE* AND EXPOSURE TO HEAT STRESS IN PIGS INDUCES LEUKOCYTE APOPTOSIS

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Exposure of mammals to pathogens and/or environmental stressors, such as heat, can disturb homeostasis of the immune system. Activation switches genes on to synthesize immunological hormones (cytokines) that can initiate inflammatory processes. Heat stress can alter endocrine hormones and such changes have the capacity to activate immune cells as well. Activation invariably causes cells to undergo programmed cell death or apoptosis, a phenomenon that allows the host to regulate cellular proliferation and to generate new cell populations to cope with the stressor. The purpose of this study was to investigate whether leukocytes undergo apoptosis *in vivo* when pigs are challenged with *Actinobacillus pleuropneumoniae* (App) and heat stressed.

Apoptotic leukocytes flip membrane phosphatidyl serine molecules to the external cell surface and this can be detected by staining with fluorescein-labelled annexin V (FITC-AnxV). Under these conditions, non-fluorescent leukocytes are scored as live cells. Pigs were infected with App by intra-tracheal challenge with $5x10^5$ CFU HS54 (serovar 1). These seeder pigs were mingled with cohort contact pigs and all were monitored clinically and serologically for 8 weeks. At the end of this period, eight of the seeder pigs (all with subclinical pleuropneumonia and ELISA positive against HS54), and eight contact pigs (disease free and serologically negative against HS54) were heat stressed at 39°C for 24 h. Heparinised blood samples were collected 24 h before heat stress, and then successively at 12, 24 and 48 h after heat stress. Buffy coats were removed and erythrocytes lysed before leukocytes were stained with FITC-AnxV and analyzed by flow cytometry.

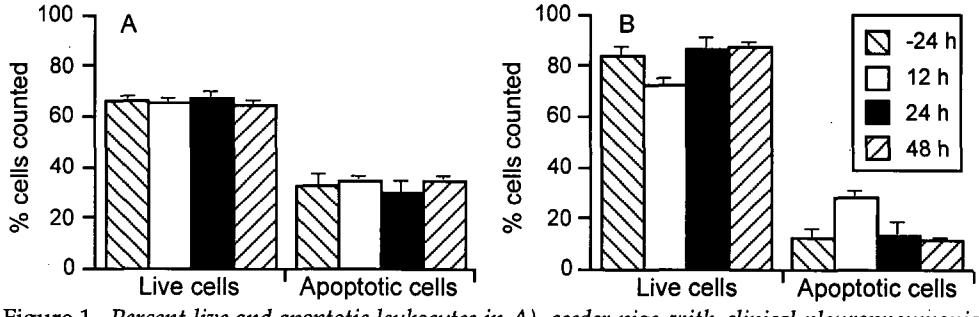

Figure 1. *Percent live and apoptotic leukocytes in A). seeder pigs with clinical pleuropneumonia, and B). contact pigs with clinical disease, after different periods of heat stress at 39°C.*

At 8 weeks after challenge, all eight pigs with subclinical App had an elevated proportion of apoptotic cells (32%) compared to the disease-free contacts (11%). During and following heat stress of both groups there was no additional effect on pigs with subclinical pneumonia as the proportion of apoptotic leukocytes remained high (range 30-34%) throughout, whereas short-term apoptotic changes occurred in disease-free pigs. After 12 h of heat induction, the frequency of apoptotic cells in contact pigs had more than doubled from 11% to 28% but returned to normal levels after 24 h of heat stress, and remained at those levels at 24 h post-heat stress.

These results show for the first time, that apoptosis can be used to assess the *in vivo* response of pigs to long-term disease stress induced by direct challenge with the respiratory pathogen App. Short-term heat stress also induced apoptosis but this was detectable only in relatively unstressed animals with prevailing low levels of apoptosis. Pigs appeared to recover more rapidly from short-term heat stress than long-term stress associated with subclinical pleuropneumonia.

PIG RESEARCH AND DEVELOPMENT CORPORATION

EFFECT OF AGE ON CLINICAL DISEASE ASSOCIATED WITH *LAWSONIA INTRACELLULARIS* INFECTION

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The presence of *Lawsonia intracellularis* within proliferating enterocytes of the ileum and colon is characteristic of both the acute and chronic forms of the porcine proliferative enteropathies (PPE). The acute form, proliferative haemorrhagic enteropathy (PHE), predominantly occurs in older and heavier pigs than the chronic disease porcine intestinal adenomatosis (PIA). Bane *et al.* (1997) suggested that variation in clinical response to *L. intracellularis* infection could be due to differences in pathogenicity of *L. intracellularis* in herds, or to the action of other synergistic bacteria in the gut. The aim of this project was to determine if the variation in clinical response to *L. intracellularis* infection could be due to the age when susceptible pigs are infected.

Three age groups of pigs were chosen from the same source (Large White x Landrace). Ten 18-week-old pigs were housed together and offered feed medicated with 250 g/tonne of oxytetracycline. Five 8-week-old pigs were housed individually and offered feed medicated with 400 g/tonne of chlortetracycline. All pigs were maintained on medicated feed from weaning to 3 days pre-inoculation to ensure that they remained susceptible to infection. A third group of eight 3-week-old pigs were housed individually and maintained on non-medicated feed. All pigs were orally inoculated with 4 to 7 x 10^9 *L. intracellularis* extracted from the same source of homogenized PHE affected mucosa. Pigs were monitored for clinical signs of infection, the presence of *L. intracellularis* DNA in their faeces and serum IgG antibodies against *L. intracellularis*, as described by Collins *et al.* (1999). Prior to inoculation, *L. intracellularis* DNA was not amplified from the faeces of any pig.

Severe clinical signs were observed in adult pigs at 17 days post inoculation (pi). One pig had persistent watery diarrhoea for 11 days, and another pig had melaena typical of PHE. All 10 adult pigs followed the same temporal pattern of infection, shedding detectable quantities of *L. intracellularis* in their faeces at 15 days pi and developing IgG antibodies to *L. intracellularis* at 24 days pi. At slaughter, 28 days pi, two pigs showed gross pathology characteristic of PHE. The mucosa of the affected ilea were thickened and ridged, and the intestinal lumen contained fresh blood.

Clinical symptoms such as diarrhoea and reduced weight gains were not evident in the inoculated 8-week-old pigs. However all pigs became infected with *L. intracellularis* as shown by the presence of *L. intracellularis* DNA in their faeces and serum IgG antibodies against *L. intracellularis*. A similar temporal pattern of faecal shedding of *L. intracellularis* and serum IgG antibodies against *L. intracellularis* were observed in all three age groups of pigs. No pigs were sacrificed for histopathology, but PCR and IFAT results indicated that all pigs recovered from infection without treatment.

Intermittent diarrhoea was observed in four of the 3-week-old pigs 13 to 19 days pi. However, diarrhoea in this age group may not be specific for *L. intracellularis* infections. Once again all of these pigs developed serum IgG antibodies to *L. intracellularis*, and *L. intracellularis* DNA was detected in all of their faeces. One of these pigs was sacrificed at 21 days pi and showed no gross pathology but histological evidence of resolving PIA lesions. All of the other pigs recovered from infection without treatment.

It was observed that the same dose and source of *L. intracellularis* inoculum in susceptible pigs of different ages could cause different clinical forms of PPE. Thus, the age when pigs are first infected with *L. intracellularis* is important in determining the nature of the clinical response to infection.

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PIGGERY SLUDGE AS A NITROGEN SOURCE FOR CEREAL CROP PRODUCTION

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Piggery sludge from effluent collection ponds spread onto cropping land might provide a form of nitrogen (N) that is available for crop production. To determine the availability of N in piggery sludge for crop production, two field trials were established on the Darling Downs, Queensland, on contrasting soils, a cracking clay (Vertosol) and a sandy loam (Sodosol). Both sites received applications of sludge in December 1997. A randomised block design of four replications also included a control and rates of 50, 100 and 150 kg N/ha as urea.

Tallon barley was planted in May 1998 and harvested in November 1998 with all treatments receiving a basal application of zinc coated MAP at planting of 50 kg/ha. Grain and plant samples were analysed for N concentration using a semi-micro kjeldahl acid digest.

Table 1. Effects of different piggery sludge treatments and urea fertiliser application on grain protein content and total N uptake (LSD 5% level).

Treatment	Application rate (t/ha)	Total N applied (kg/ha)	Vertosol		Sodosol	
			Grain protein (%)	Total N uptake (kg/ha)	Grain protein (%)	Total N uptake (kg/ha)
Control	0	0	11.63	127.3	11.32	58.6
Wet Sludge	20	80	13.16	128.5	13.48	71.4
Wet Sludge	65	260	13.55	154.5	14.53	83.5
Dried Sludge	6	40	12.27	124.9	11.42	63.1
Dried Sludge	18	120	12.18	144.0	12.08	70.9
Urea	0.109	50	13.93	142.8	12.64	79.0
Urea	0.217	100	14.73	158.8	13.27	92.0
Urea	0.326	150	14.64	156.9	14.71	98.2
LSD			0.981***	21.42***	1.160***	11.79***

***P≤0.001.

A high incidence of net blotch (foliar disease), promoted by high rainfall during crop growth, resulted in non-significant yield responses to the various sludge treatments. The harvest index (grain weight/total weight) ranged from 0.24 to 0.46 demonstrating a high level of within-plot variation resulting in apparent yield trends not being statistically significant.

In spite of the overwhelming influence of the net blotch on grain yield the application of wet piggery sludge significantly increased grain protein content at both sites (P≤0.001), and the application of 18t/ha of dried sludge had a significant effect at the Sodosol site. Only the 65t/ha application of wet sludge significantly increased total N uptake at the Vertosol site, whilst the application of wet sludge and the higher dried sludge rate increased total N uptake at the Sodosol site (P≤0.001). Schoenau *et al.* (1998) also observed higher crop N uptake and protein contents in spring wheat plots receiving piggery manure.

The results suggest there is good reason to dispose of piggery sludge onto cropping land as a N source for cereal crop production.

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PIG RESEARCH AND DEVELOPMENT CORPORATION

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THE IMPACT OF EFFLUENT APPLICATION ON SOIL P: TWO CASE STUDIES

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Land application of piggery effluent is under close scrutiny worldwide as a potential source of phosphorus (P) leading to water resource contamination. The strong retention of P by many soils is well known, and is likely to be an effective barrier against contamination of groundwater by leached P. The aim of this study was to determine if soil has been an effective and safe store of effluent P in two long-term case studies.

The soil of piggery 1 (PF1) was a Grey Sodosol with a profile depth of greater than 1.5 m. A piggery has operated at this site since 1979, the herd expanding from 8,000 to 13,500 standard pig units (SPU) in that time (one SPU produces waste volatile solids equivalent to a 40 kg pig). During this period a total of 4,000 kg of effluent P/ha in excess of crop requirements has been applied. Throughout its life effluent management at this piggery has followed the development of industry best practice, though past practices have been less conservative in their treatment of P.

The Red Dermosol (1.2 to 1.5 m deep) of the effluent application area at piggery 2 (PF2) has received a total of 35,000 kg of effluent P/ha over 30 years, with minimal nutrient removal. This piggery commenced operations in 1968, the herd expanding from 490 to 4,300 SPU in the period of operation. Effluent irrigation has been carried out purely as a disposal method.

Total and Colwell extractable P forms (CEP; Rayment and Higginson, 1992) were determined throughout the soil profiles for paired (irrigated and unirrigated) sites at PF1 and PF2 (replicated analyses; t-Tests and analysis of repeated measures applied). Phosphorus sorption and desorption studies were also undertaken (Rayment and Higginson, 1992).

Surface soil CEP has been significantly elevated (0 to 0.05m depth, $P<0.01$) at both sites (P1 increased from 23 ± 1 to 290 ± 6, P2 increased from 72 ± 48 to 3,950 ± 1,960, mean ± SD mg P kg^{-1}). From the laboratory determined P sorption curves and actual effluent concentrations, total P concentrations in the top 0.05 m of the soils are greater than 10 times the theoretical sorbed-P concentrations.

No evidence of P leaching below the profile was observed at PF1. At the PF2 irrigation area, leaching losses of P from the soil profile to the boundary with underlying weathered rock have already occurred (based on profile Total P contents; Figure 1; determined by XRF, as described in Rayment and Higginson, 1992; $P<0.05$).

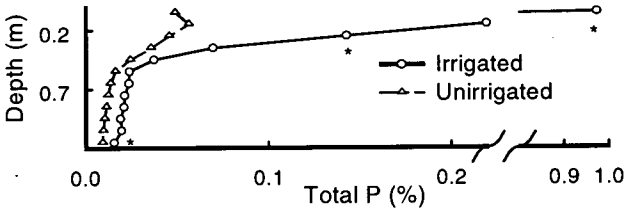

Figure 1. *Profile total P for PF2, intervals where irrigated total P significantly exceeds the unirrigated concentrations are denoted with a "*".*

The surface soil P elevation at both sites considerably increases potential for run-off transport of P. Though leaching from the profile at one of the sites was in evidence, both irrigated profiles demonstrated a capacity to retain large quantities of P against leaching. Phosphorus accumulated in the surface soils beyond the conventional P sorption curve theoretical levels.

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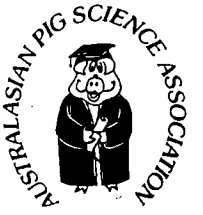

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