Improving the performance of the progeny of gilts

Report prepared for the
Co-operative Research Centre for an Internationally Competitive Pork Industry

by

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Executive Summary

Gilts contribute a large proportion of progeny to the growing herd. This occurs as a result of the high replacement rates in breeding herds in the Australian pig industry,. The progeny born to gilts (first parity sows) are lighter at birth (Hendrix et al., 1978; Tantasuparuk et al., 2001) and weaning (Burkey et al., 2008; Holyoake, 2006) than progeny from older parity sows. Post-weaning, gilt progeny are more susceptible to disease than sow progeny, with higher rates of medication and mortality (Holyoake, 2006). The aim of this project was to identify risk factors for the poor post-weaning performance of gilt progeny, with particular emphasis on identifying the roles of weaning weight, pathogen carriage and immunity.

The first component of this study focused on attempts to improve the post-weaning growth performance of gilts by increasing their weaning weights with supplemental full-cream milk. The weaning weights of supplemented gilt litters was similar to non-supplemented sow litters, but the post-weaning medication and mortality rates of gilt progeny continued to exceed that of sow progeny. This result suggests there are weight-independent factors responsible for this relatively poor performance.

There were no apparent differences in pathogen carriage rates (using *Lawsonia intracellularis* as the model pathogen) between gilt and sow progeny. The age at which pigs produced antibodies to *L. intracellularis* (reflecting their age of infection) was similar between gilt and sow progeny. The use of medication to suppress *L. intracellularis* infections in pigs less than 10 weeks of age on the study farm may have impacted on our results. Additional experimental work comparing carriage rates between gilts and sows with other endemic farm pathogens is warranted.

Gilts produced a higher concentration of circulating antibodies in response to a novel antigen (tetanus toxoid) than sows. However, sows were able to transfer more tetanus toxoid-specific antibodies from their blood to colostrum. This result suggests that maternal transfer of antibodies is more efficient in sows than gilts. The immune responsiveness of the progeny themselves was no different between piglets born/reared on gilts or sows. There was a trend of increased rate of antibody decay in piglets reared on gilts, suggesting that this may contribute to this population's increased susceptibility to disease. More research in this area is warranted.

In summary, our research suggests that the three main risk factors identified as contributing to the poor growth and survival of gilt progeny are piglet birthweight, piglet milk intake and the immunity transferred from the gilt compared to that from older parity sows. Gilt progeny weighed on average 200g less at birth than sow progeny. Birthweight independently influenced piglet pre- and post-weaning growth and survival through to market. Heavier piglets drank more milk than their lighter counterparts – most likely due to increase suckling stimulus causing increased mammary gland growth. Sows produced more milk than gilts, independent of average piglet birthweight in the litter. Sow progeny are likely to transfer more antibodies specific to endemic pathogen to their progeny than gilt progeny due to their repeated exposure to these, and the production of memory immune cells.

The main strategy currently being used to manage gilt progeny is segregated rearing of gilt and sow progeny. This has led to reduced requirements for medication and vaccination in the sow progeny while the performance of gilt progeny remains unchanged (Moore, 2004, 2001). However, as the degree of segregation required (by farm, shed or pen) is unknown, segregated rearing cannot be easily implemented on smaller farms. Improvement in the exposure of gilts to farm-endemic pathogens may alleviate the current disease

susceptibility differences between gilt and sow progeny simply by improving the specificity of the maternal protection provided.

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1. Introduction

The progeny born to gilts (first parity sows) are lighter at birth (Hendrix et al., 1978; Tantasuparuk et al., 2001) and weaning (Burkey et al., 2008; Holyoake, 2006) than progeny from older parity sows. Post-weaning, gilt progeny are more susceptible to disease than sow progeny with higher rates of medication and mortality (Holyoake, 2006).

With the current high replacement rates in breeding herds in the Australian pig industry, gilt progeny encompass a large proportion of the growing herd. The increased disease susceptibility of this subpopulation of gilt progeny in the herd potentially leads to a greater variation in pig weights as well as fewer pigs at market. Additionally, this disease-susceptible subpopulation is likely to facilitate the proliferation of farm-endemic pathogens, thereby exposing the rest of the herd to disease.

The aim of this project was to identify risk factors for the poor post-weaning performance of gilt progeny with particular emphasis on identifying the roles of weaning weight, pathogen carriage and immunity.

2. Methodology

Experiment 1

To investigate the role of weaning weight on post-weaning growth and survival, experiment 1 investigated the use of supplemental milk pre-weaning as an intervention to increase piglet weaning weights. Gilts (80 parity 0) and sows (80 parity 2-5) were randomly allocated to either a supplemented or a non-supplemented treatment group in both a winter and a summer replicate. Litters were standardised to 10 piglets per litter. Re-constituted full cream milk powder was provided 2-3 times daily to the supplemented litters from Day 3 post-farrowing until weaning (~Day 28). Piglet teat position was recorded for all piglets on 3 occasions during the first 2 weeks of lactation. This teat position was converted to udder section (US) 1 (anterior two teat pairs), US 2 (middle two teat pairs), and US 3 (remaining posterior teat pairs). Post-weaning, piglets were penned according to their treatment groups and feed consumed per pen was measured to calculate pen feed conversion efficiency. Five pens per gender per treatment group were randomly formed, each containing 20 pigs /pen. Pre- and post-weaning weights and medications/mortalities were recorded for individual piglets through to 10 weeks of age. Sow weights and P2 backfat at entry to the farrowing shed (~8 days before farrowing) and at weaning, as well as daily feed intake over the 4 week lactation, were recorded for individual sows. These sows were also followed through to the subsequent farrowing to measure litter weight and size as well as the interval to re-mating. Ambient temperature in each farrowing shed was recorded.

Experiment 2

During the summer replicate of Experiment 1, a study was undertaken in non-supplemented litters as an indirect measure of sow and gilt milk yields. This was conducted according to methodology by King et al. (1993) and adjustments made

by Theil et al. (2002). This was conducted over three 2-day measurement periods (7-9, 14-16, and 21-23 days lactation) and included 11 gilt and 11 sow litters.

Experiment 3

Pathogen carriage was investigated using *Lawsonia intracellularis* as a model pathogen. Equal numbers of sows and gilts testing seropositive and seronegative for serum IgG antibodies against *L. intracellularis* were used (30 for each of the 4 treatment groups). Pens were thoroughly cleaned and disinfected prior to pig entry. Strict biosecurity was enforced in the farrowing and weaner sheds preventing unnecessary entry of staff into sheds/pens. Where pen entry was essential, single-use, single-pen boot-covers were provided. Presence of *L. intracellularis* was monitored in sow faeces using PCR (polymerase chain reaction) at days 7, 14 and 21 of lactation. Presence of *L.intracellularis* IgG and IgA antibodies were monitored using IFAT (immunofluorescent antibody test) in sow colostrum/milk at days 0 and 14 of lactation. Also, IgG antibodies were measured using IFAT in sow serum 5 weeks prior to farrowing and in piglet serum at 4, 8, 12, 16, and 20 weeks of age. Piglet weight measurements were collected at 4, 10, 16, and 22 weeks of age along with carcass weight/back fat and gross intestinal lesion scoring.

Experiment 4

Responsiveness of the innate and adaptive immune systems of gilts and sows and their progeny were investigated in the final experiment. A novel antigen (tetanus toxoid), was chosen to investigate immune system differences to eliminate the effect of the improved immune responses expected in older parity sows over gilts, as a result of repeated pathogen exposure. Sows (64) and gilts (64) were vaccinated 7 and 4 weeks prior to farrowing with tetanus toxoid (Equivac T vaccine; Pfizer). Blood samples were collected from the dams prior to vaccination and then 2 weeks after the second vaccination. On-trial dams were also blood sampled at 21 days of lactation. Colostrum/milk samples were collected on Days 1 (day after farrowing), 8, and 22 of lactation.

Progeny immune system responses were investigated in relation to their birth or rearing dam parity by cross-fostering prior to their first suckling. This led to the formation of 16 gilt and 16 sow litters, each consisting of 5 gilt-born and 5 sow-born piglets, with no piglets remaining on their birth dam. Weights were collected for all piglets at 0, 4, 10, 16, and 22 weeks of age. Blood samples were collected from the same randomly-selected 3 gilt-born and 3 sow-born piglets per litter at 2, 4, and 7 weeks of age. At weaning (4 weeks of age), half of each piglet blood sample group was randomly designated to vaccination with either saline (negative control) or tetanus toxoid.

All laboratory tests used in the final experiment were developed using samples generated during a pilot study with 1 non-vaccinated sow and 3 vaccinated sows. All serum, colostrum and milk samples were tested for the presence of tetanus toxoid-specific IgG, IgA and IgM antibodies. Whole blood samples from the piglets (only at 2 and 7 weeks of age) and the sows/gilts were incubated with tetanus toxoid to determine specific production of IFN ¤ and therefore the presence of circulating cell-mediated immunity against tetanus toxoid. Piglet blood samples

(only at 2 and 7 weeks of age), along with 21 day lactation dam blood samples, were mixed with a fixed concentration of yeast particles to determine the phagocytic index (percentage of phagocytic cells with ingested particles of yeast and the number of particles per cell). A differential cell count performed on whole blood to identify numbers of each type of leucocyte.

3. Outcomes

Experiment 1

The provision of supplemental milk was successful at significantly increasing piglet weaning weight and bridging the growth gap between gilt and sow progeny in winter (Table 1) although it did not influence piglet pre- or post-weaning mortality or medication rates ($P > 0.05$). The provision of supplemental milk did not influence the within-litter weight variation in supplemented compared to non-supplemented litters. Dam parity independently influenced piglet growth during winter, but not summer, both pre- and post-weaning (Table 1) with gilt progeny growing slower than sow progeny.

Table 1 Predicted means of piglet weights (kg) by dam parity / supplemental milk treatment group, separated by replicate and adjusted for birth weight (1.6kg)

Replicate	Dam parity		21 days	Weaning	10 wks
Winter	Gilt	No milk	6.0 ^{a}	7.0 ^{a}	21.7 ^{a}
Winter	Gilt	Milk	6.3 ^{ab}	7.3 ^{ab}	22.5 ^{a}
Winter	Sow	No milk	6.6 ^{bd}	7.6 ^{bd}	24.6 ^{b}
Winter	Sow	Milk	6.8 ^{d}	8.0 ^{d}	25.7 ^{b}
Summer	Gilt	No milk	5.6 ^{a}	6.8 ^{a}	24.1 ^{ab}
Summer	Gilt	Milk	6.2 ^{b}	7.7 ^{b}	25.4 ^{a}
Summer	Sow	No milk	6.0 ^{b}	6.8 ^{a}	23.2 ^{b}
Summer	Sow	Milk	6.7 ^{c}	7.5 ^{b}	24.6 ^{ab}

Different superscripts within column within replicate indicates significance ($P<0.05$)

Dam parity-related differences in pre-weaning piglet growth are likely to have resulted from milk yield differences. During the summer replicate, the average ambient temperature in the farrowing sheds (26°C) was higher than the preferred temperature range of sows (12-22 °C), unlike during the winter replicate (21°C). It is suspected that this higher ambient temperature negatively affected older parity sows more than gilts. The increase in dam body temperature would have led to a re-direction of blood flow away from the mammary gland to the skin for heat exchange and a reduction in potential milk yield (Black et al., 1993). While there was a drip cooling system provided for dams, this may have been insufficient to provide an adequate wet surface area for heat exchange in sows compared to the lighter bodyweight gilts. During the winter replicate, when the average

temperature was in the preferred temperature range for sows, they produced heavier piglets at weaning than gilts, regardless of the average piglet birthweight in the litter.

The slower post-weaning growth of gilt progeny compared to sow progeny during the winter replicate occurred despite adjustment for differences in weaning weight and appeared to be related to the incidence of disease. Gilt progeny had a significantly higher rate of medications and mortality than sow progeny. While both dam parity groups had a significantly higher incidence of post-weaning medications during winter than summer, gilt progeny had a significantly higher rate of being taken "off-trial"(for "ill-thrift" requiring targeted intervention) in winter only. There was no significant difference in feed conversion efficiency between the two dam parity groups, but gilt progeny had a lower feed intake than sow progeny during the winter replicate. It may be that this lower feed intake, due to presence of disease, was directly responsible for the relatively poor post-weaning growth of gilt progeny compared to sow progeny during winter.

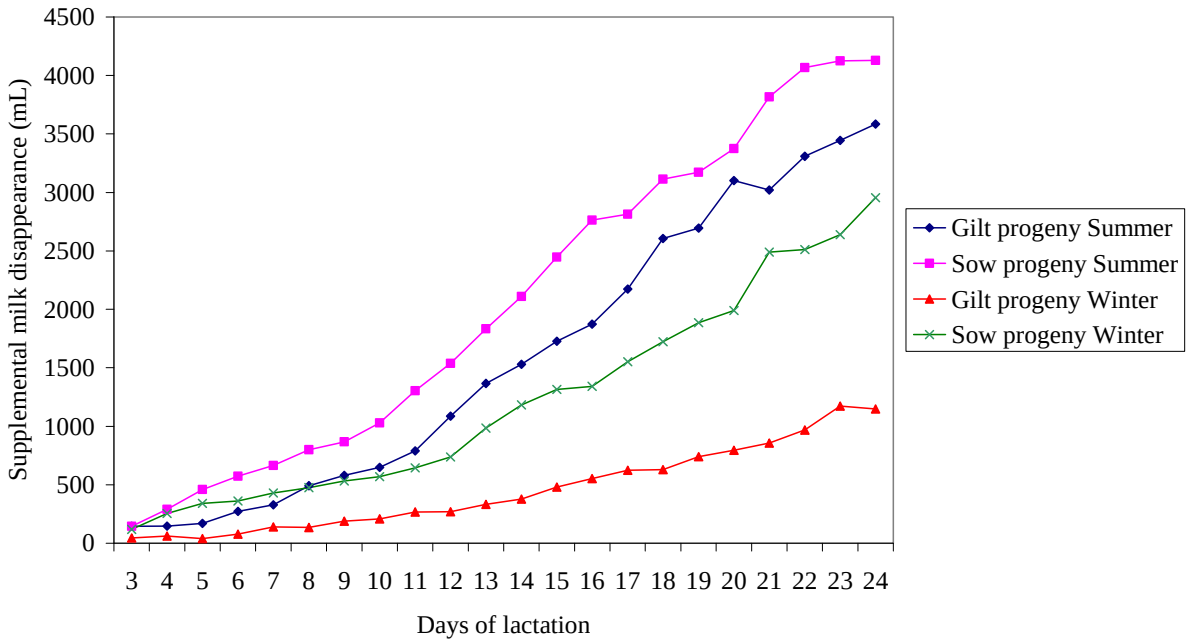

Figure 1. Average daily supplemental milk disappearance (apparent intake) per litter (mL/litter/day) among gilt and sow litters in winter and summer experimental replicates.

There appeared to be dam parity effects on the disappearance rate, or apparent intake, of supplemental milk in both summer and winter. There was a lower disappearance rate in gilt litters compared to sow litters, even after adjusting for average piglet birthweight in each litter (Figure 1). Reasons for this dam parity effect on litter supplemental milk disappearance is currently unknown. This does not appear to be a result of milk yield differences between the parities. During winter, the difference between the average gilt and sow litter supplemental milk disappearance was larger (Figure 1) and there was a concurrent significant difference in weaning weights of supplemented groups even after adjustment for birthweight (Table 1). Further investigation is required to determine why some

litters "consumed" significantly more supplemental milk than others. Importantly, under the conditions of our study, supplemental bovine milk did not reduce post-weaning disease susceptibility of gilt litters.

Supplemental milk did not impact dam weight or P2 backfat changes over lactation. Similarly, there were no effects on reproductive performance (interval to re-mating or subsequent litter weight and size) in agreement with findings by others (Azain et al., 1996; Dunshea et al., 1999; Dunshea et al., 1997). The average piglet birthweight in litters from gilts in the subsequent litter (now parity 1 sows) were not significantly different from that of older parity sows (now parity 3-6) (Table 2). This indicates that the lower birthweight of gilt progeny is confined to the first litter and improves in the subsequent litter as a result of either the increase in dam age/maturity or parity.

Table 2. Average piglet birth weight within gilt and sow litters born during the current experiment (Trial litter) and the subsequent litter.

		Trial litter		Subsequent litter	
Replicate	Dam parity	Born alive	Average birth weight (kg) *	Born alive	Average birth weight (kg) *
Winter	Gilt	10	1.42 [a]	11	1.72 [a]
Winter	Sow	12	1.62 [b]	12	1.63 [a]
Summer	Gilt	11	1.44 [a]	11	1.62 [a]
Summer	Sow	12	1.66 [b]	12	1.58 [a]

Different superscripts within columns indicates significance (P<0.05)

The minimal fostering employed in this experiment resulted in only 6.2 % of piglets being fostered, but fostering - along with birth weight, significantly predicted piglet teat position. Heavier birthweight piglets were significantly more likely to be found on an anterior teat than a posterior teat after adjusting for the effect of fostering (P<0.05; Table 3). Fostered piglets were significantly more likely to be found on posterior teats than on anterior teats, after adjusting for birth weight (P<0.05).

Table 3 The probability* that a piglet of a particular birth weight (kg) will be found on a particular teat pair; anterior (1) to posterior (6+), adjusted for the effect of fostering.

Birth weight (kg)	Teat 1	Teat 2	Teat 3	Teat 4	Teat 5	Teat 6+
0.6	0.11	0.14	0.18	0.18	0.18	0.21
1.6	0.16	0.18	0.19	0.17	0.15	0.15
2.2	0.20	0.20	0.19	0.15	0.13	0.12
3	0.26	0.23	0.19	0.13	0.10	0.09

* The probabilities across all teat pairs for a particular birth weight combine to equal 1.00 or 100 %

Piglets on anterior US grew faster than those on posterior US for both parity groups but this effect was not significant post-weaning (Table 4). There was an

interaction of birth weight and US on subsequent piglet weight (P<0.001). Piglets grew faster on US 2 than US 3, regardless of their birth weight. Udder section 1 growth benefits, however, were more obvious for lighter than heavier birth weight piglets. Lighter piglets grew faster on US 1 than any other US but heavier piglets appeared to grow equally well, regardless of which US they occupied.

Table 4 Model-based means for piglet weights (kg) for each udder section (US) adjusted to average birth weight (1.6 kg).

	Piglet age (weeks)		
	3	Weaning (4)	10
US 1 (anterior)	6.55 [a]	7.70 [a]	24.31 [a]
US 2 (middle)	6.38 [b]	7.54 [b]	24.14 [a]
US 3 (posterior)	5.99 [c]	7.13 [c]	24.22 [a]

[abc]Different superscripts within column indicate significance (P<0.01).

Experiment 2

The deuterium oxide dilution experiment was conducted during the summer replicate of the supplemental milk experiment. The results confirmed that dam parity did not affect milk yield, after adjusting for piglet birthweight. Additionally, while litterweight had an important effect on milk yield, this effect was more pronounced in early lactation (Figure 2). This may result from a greater potential for mammary tissue growth early compared to later in lactation (Hurley, 2000).

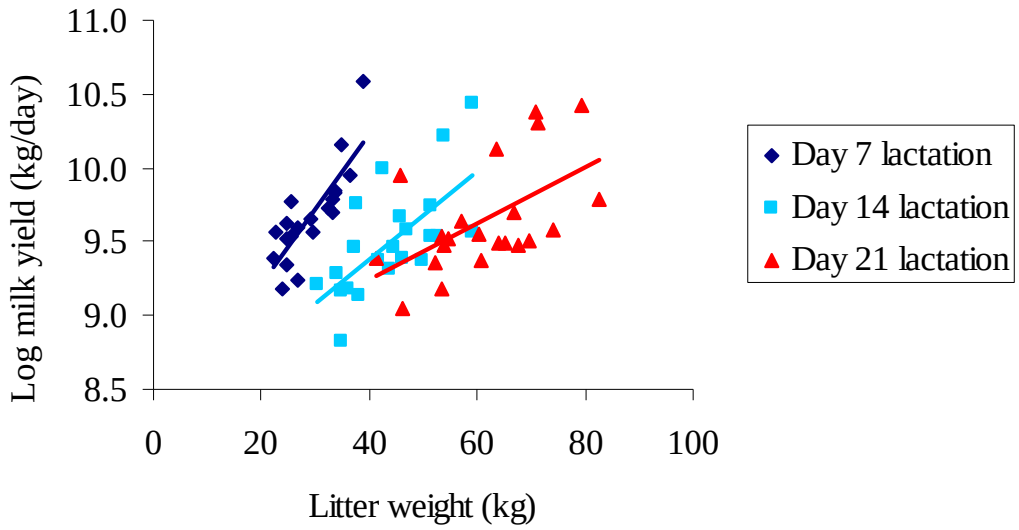

Figure 2 The relationship between litter weight and (log) dam milk yield at each stage of lactation.

Both piglet suckling pressure (weight and vigour) and the potential mammary gland milk yield determines piglet milk intake, which subsequently determines

piglet pre-weaning growth. Previous studies suggest that gilts produce less milk than sows (Ferreira et al., 1988; Speer and Cox, 1984). Our results are in agreement with these (Table 5), but also suggest that litter weight plays a major role in determining milk yield. The potential inherent difference between gilts and sows in milk yield (unrelated to litter weight differences), likely to be the cause of the difference in pre-weaning growth between gilt and sow litters, is likely to result from dam immaturity (leading to nutrient partitioning effects; (Pluske et al., 1998), immaturity of the mammary tissue or simply insufficient quantity of mammary tissue.

Table 5 Calculated average milk yield of gilts and sows over lactation (kg/day +/- standard deviation)

	7 days	14 days	21 days
Gilts	14.1 (2.4)	12.4 (4.0)	13.7 (3.2)
Sows	19.6 (8.1)	16.5 (7.5)	19.6 (8.8)

The higher milk yield of anterior US than posterior US may be explained by location (blood pressure/proximity to the heart) and by tissue development over successive lactations. Our results suggest that preferential fostering of heavier piglets may allow their lighter counterparts to occupy anterior udder sections. Not only could this lead to a reduction in within-litter weight variation, but it could lead to better development of the posterior udder sections and improve their potential milk yield in the subsequent lactation. The occupation of the higher yielding anterior udder sections by lighter birth weight piglets would not result in permanently lower milk yields from anterior udder sections, unlike posterior udder sections.

Importantly, increasing the suckling pressure across the whole udder, by increasing the number of teats being suckled during lactation, is important for the development of the whole udder. Suckling pressure is therefore a vital factor influencing milk yield during that lactation as well as subsequent lactations. This should be considered, along with consequences for sow longevity, when deciding on litter sizes for gilts and sows.

Experiment 3

Under the conditions of the study, dams were not a source of *Lawsonia intracellularis* to their piglets. This finding is in contrast to others (Bronsvoort et al., 2001; Jensen et al., 2005; Møller et al., 1998; Smith and McOrist, 1997). *Lawsonia intracellularis* was not detected in any dam faecal samples collected during lactation. Neither dam parity nor pre-farrowing sero-status influenced the timing of sero-conversion in the progeny, probably as a result of the timing of progeny exposure (Figure 3). Piglets were not exposed to *Lawsonia intracellularis* until 10 weeks of age due to farrowing and weaner shed biosecurity and medications in feed and water post-weaning.

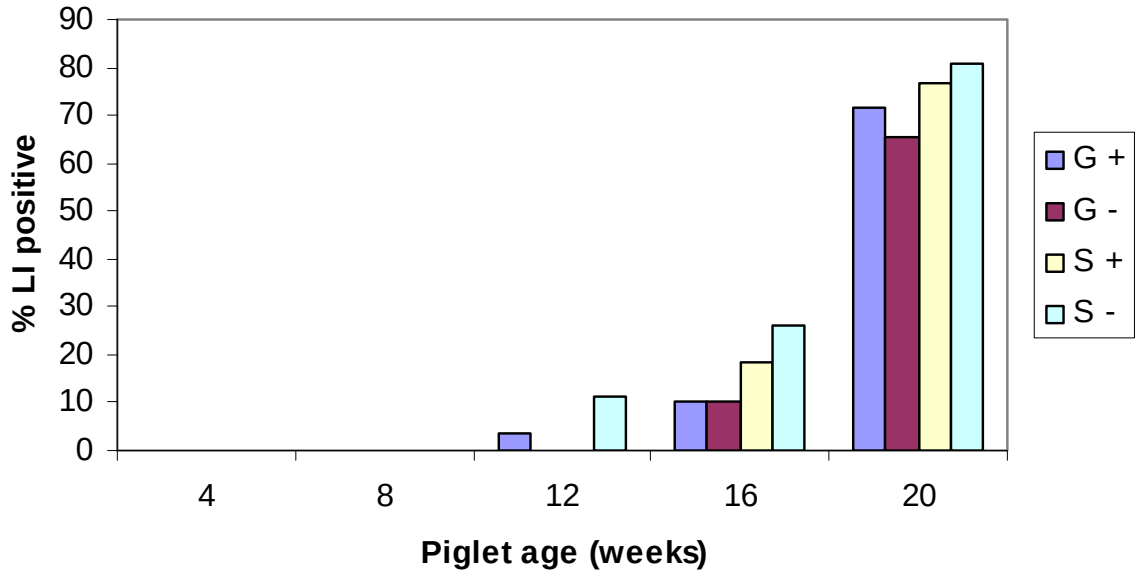

Figure 3 Percentage of progeny within each treatment group that tested positive to *L. intracellularis* (LI) over time, by dam treatment group (gilts (G), sows (S), with (+) or without (-) serum antibodies).

Gilt progeny grew slower than sow progeny post-weaning, independent of weaning weight differences (Table 6). This result occurred despite there being no differences in expression of clinical disease (rates of medication). As this effect does not appear to result from differences in timing of infection with *Lawsonia intracellularis*, further investigation is required into other farm-endemic pathogens. The dam parity-related effect on piglet performance, associated with a particular farm-endemic pathogen, might only occur when piglets are exposed pre-weaning or in the early post-weaning period.

Table 6 Model based average live weights (kg) of gilt and sow progeny over time adjusted for average wean weight (6.8 kg).

	Gilt Progeny	Sow Progeny
10 weeks	22.0 [a]	23.9 [b]
16 weeks	53.5 [a]	57.3 [b]
22 weeks	83.8 [a]	88.7 [b]

Different superscripts within row indicate significance ($P<0.05$).

These findings have two important implications. Firstly, biosecurity in farrowing sheds is likely to prevent pre-weaning exposure of piglets to *L.intracellularis*. Secondly, if piglets are exposed to *L.intracellularis* pre-weaning (either through live oral vaccination or natural exposure), the earlier that they are exposed, the more influence maternally derived protection (and therefore dam parity) is likely to have on the subsequent immune response of progeny. Any effect of dam parity on piglet infection with *L. intracellularis* is therefore likely to be limited to

situations in which piglets are exposed in the pre-weaning or early post-weaning period which could similarly be the case for other farm-endemic pathogens.

Experiment 4

The tetanus toxoid model used in the final experiment was an appropriate model for investigating specific IgG antibody production but not for cell-mediated or mucosal (IgA) immunity. The vaccine adjuvant (aluminium hydroxide) and the route of vaccination, as expected, preferentially induced an antibody response (not cell-mediated) and an IgG (not IgA) response respectively in both vaccinated dams and piglets. Maturity of the piglet immune system from 4 to 7 weeks of age was evident with the laboratory tests used. There was an increase in the number and responsiveness of cells involved in the innate and adaptive sides of the immune system. Birth dam parity did not appear to be an important influence on piglet immune system development or their response to vaccination. Rearing dam parity, however, influenced the progeny antibody concentrations.

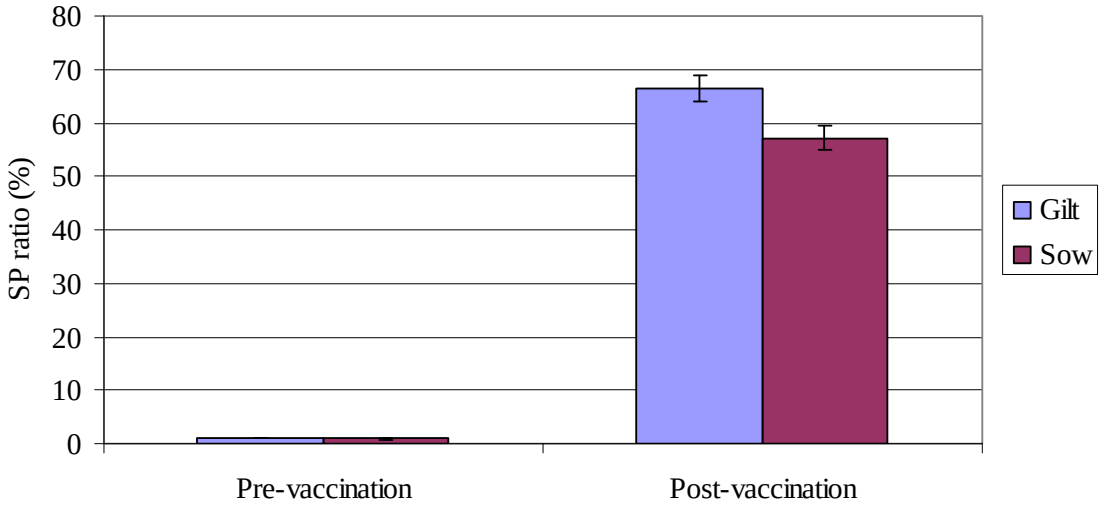

Figure 4 Tetanus toxoid specific IgG relative concentrations (SP ratio %) in gilts and sows pre- and post-vaccination. Bars indicate standard errors of the mean.

There were no detectable IgG antibodies to background tetanus in any dams prior to vaccination, as expected. Two weeks after the second vaccination, however, gilts had a higher circulating concentration of IgG compared to older parity sows (Figure 4). This was not surprising considering the younger age and higher circulating concentration of lymphocytes in gilts (although it is unknown if these were B or T lymphocytes). Despite the higher concentration of IgG in serum, there was no difference between gilts and sows in the concentration of IgG present in colostrum or milk throughout lactation (Figure 5). This was despite the fact that 100% of IgG is transported directly from serum to colostrum. There is either a reduced ability of gilts to transport IgG or a threshold in the quantity of antibodies able to be transported into colostrum.

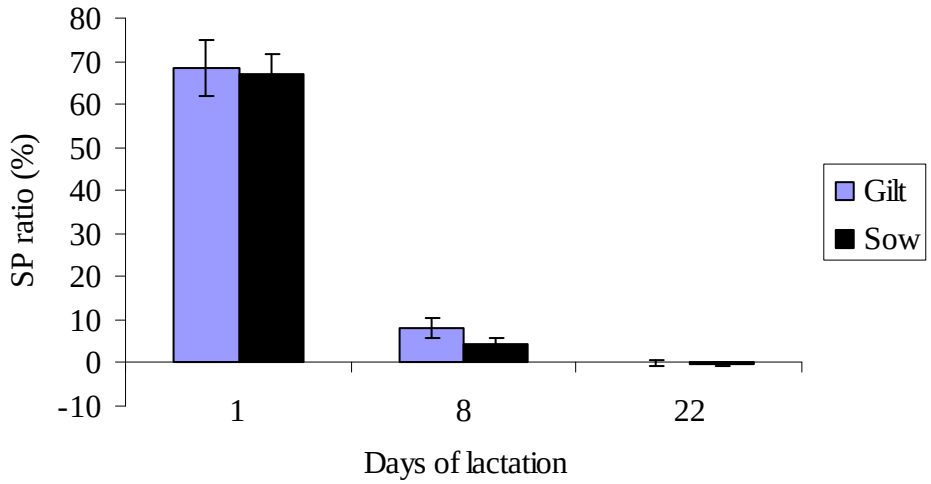

Figure 5. Average colostral tetanus toxoid-specific IgG relative concentrations (SP ratio %) in gilts and sows over lactation (days post-farrowing). Bars indicate standard errors of the mean.

There was no obvious influence of birth dam parity on the immune system responsiveness. Rearing dam parity, however, influenced antibody concentrations. There was a higher concentration of serum IgG antibodies present in gilt-reared piglets at 2 and 4 weeks of age (Figure 6) despite exposure to colostrum containing the same concentration of IgG (Figure 5). This is most likely to be due to antibody dilution (Kitching and Salt, 1995) in sow-reared piglets occurring after "gut closure" due to the significantly faster pre-weaning growth of sow-reared compared to gilt-reared piglets (Table 7).

Table 7 Model-based average weights (kg) per piglet treatment group.

	Gilt reared		Sow reared	
	Gilt-born	**Sow-born**	**Gilt-born**	**Sow-born**
Birth	1.4 [a]	1.6 [b]	1.4 [a]	1.6 [b]
4 wks	6.6 [a]	7.0 [b]	7.5 [c]	7.7 [d]
10 wks	25.3 [a]	25.9 [a]	26.3 [a]	27.3 [a]
17wks	61.0 [a]	61.8 [a]	60.7 [a]	61.6 [a]
22wks	93.2 [a]	92.3 [a]	89.9 [a]	93.2 [a]

Different superscripts within row indicate significance $P<0.05$.

The presence of maternal antibodies appeared to interfere with piglets generating their own active immune response to tetanus toxoid vaccination at weaning. This is evident by the lack of significant difference between the antibody concentrations between vaccinated piglet groups 3 weeks post-weaning (Figure 6). Also, post-weaning, non-vaccinated gilt-reared piglets appeared to have a faster decline in antibody concentrations than non-vaccinated sow-reared piglets (Figure

6). This finding still needs to be confirmed with more time points post-weaning particularly in relation to maternal antibodies effective against farm-endemic pathogens.

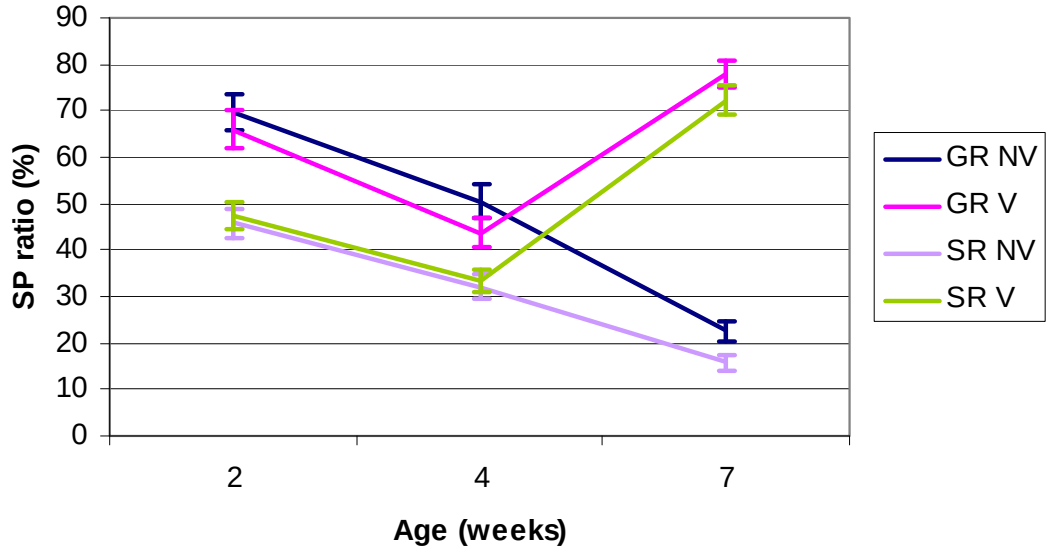

Figure 6. The tetanus toxoid specific IgG concentration (SP ratio %) in serum of piglets reared on gilts (GR) and sows (SR) and their response to tetanus toxoid vaccination (V= vaccinated, NV= not vaccinated) at 4 weeks of age. Bars indicate standard errors of the means.

4. Application of Research

There are a number of management strategies that could be directly applied on-farm as a result of this project (7. Recommendations). We have also identified a number of areas where additional research is warranted. The outcomes of this project have been reported to the Pork CRC, scientific community, veterinarians and producers through a number of publications and in seminars (see below). We recommend the Pork CRC consider opportunities for incorporating the findings of our research through amalgamation with other face-to-face producer workshops.

Publications arising from this research

Miller, Y.J. Collins, A.M. Smits, R.J. and Holyoake, P.K. 2005 Improving the performance of gilt progeny. Proceedings of the Australian Association of Pig Veterinarians. Gold Coast. p118.

Miller, Y.J. Collins, A.M. Smits, R.J. and Holyoake, P.K. 2006. Improving the performance of gilt progeny. Proceedings of the Australian Pig Veterinarians Conference. Melbourne. p64.

Miller, Y.J. Collins, A.M. Smits, R.J. and Holyoake, P.K. 2006. Improving the performance of gilt progeny. Proceedings of the 19th International Pig Veterinary Society Congress. Copenhagen. p103.

Miller, Y.J. Collins, A.M. Smits, R.J. and Holyoake, P.K. 2007. What determines teat position within a litter? Australasian Pig Science Association Conference Proceedings, Brisbane. p62.

Miller, Y.J. Holyoake, P.K. Smits, R.J. and Collins, A.M. 2007. Effect of dam parity on transmission of proliferative enteropathy. Australasian Pig Science Association Conference Proceedings, Brisbane. p57.

Miller, Y.J. Collins, A.M. Smits, R.J. and Holyoake, P.K. 2007. Teat order may affect within litter weight variation but supplemental milk does not. Australasian Pig Science Association Conference Proceedings, Brisbane. p38.

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5. Conclusion

Three main risk factors were identified as contributing to the poor growth and survival of gilt progeny; piglet birthweight, piglet milk intake and the immunity transferred from gilts compared to that from older parity sows.

Gilt progeny are lighter at birth than sow progeny. Birthweight independently influences piglet pre- and post-weaning growth and survival through to market. Piglet milk intake is also affected by their birthweight as the growth, and therefore ultimate milk yield, of the mammary gland is determined by piglet suckling pressure (weight and vigour). Milk yield is also influenced by dam parity and is lower in gilts than older parity sows, independent of differences in average piglet birthweight in the litter.

Nutrient partitioning is one of the main factors contributing to lower piglet birthweight and lower potential milk yield of gilts. Since gilts have not reached their mature bodyweight at the time of first gestation/lactation they need to partition nutrients to requirements for their own growth as well as the requirements of their foetuses/mammary glands. Additionally, mammary tissue that is accumulated during lactation is remodeled for use in the subsequent lactation such that parity independently plays an important role in milk yield.

Despite gilts generating a greater IgG antibody response to novel vaccination compared to sows, they appear to be less capable of transferring these antibodies from serum into colostrum. While further research is required using farm-endemic pathogens, insufficient exposure to these relevant pathogens, combined with inadequate transfer of the antibodies into colostrum, is likely to result in inadequate passive protection of progeny against farm-endemic pathogens prior to development of their own active immune response.

The reasons for these differences in growth and survival between the gilt and sow progeny are therefore most likely to relate to the immaturity of gilts and the

reduced exposure of gilts to farm-endemic pathogens compared to that of older parity sows. Improvement in the exposure of gilts to farm-endemic pathogens is likely to significantly improve progeny protection against farm-endemic pathogens and therefore their growth and survival. A flow-on effect will be an improvement in overall herd health status and medication use through reduced susceptibility of gilt progeny to disease.

6. Limitations/Risks

To the application of the research findings

- As differences in growth performance between gilt and sow progeny stem largely from differences in birthweight, there is a requirement to investigate techniques to improve this
- Producers should consider providing supplemental milk to gilt litters in summer to increase weaning weights. Each farm will need to consider the costs and practicality of undertaking this practice
- Increased exposure of gilts to endemic farm pathogens has potential to improve their potential for passive immune transfer to their progeny. Exposure strategies need to be devised in accordance with current state swill feeding restrictions
- Segregated management of gilt progeny offers producers a tool for improving the health of sow progeny and reducing their medication/vaccination costs. The benefits of undertaking segregated rearing on small farms needs to be determined

7. Recommendations

As a result of the outcomes in this study the following recommendations have been made to improve the performance of gilt progeny:

1. Under current conditions, gilt and sow progeny should be considered two separate populations. This is especially important when piglets are being blood sampled for optimal vaccination timing based on circulating concentrations of maternal antibodies.
2. Maximize the birthweight of gilt progeny through optimal nutrition during gestation.
3. Maximize gilt milk production by optimizing the environmental conditions for milk production and maximising suckling pressure during lactation
4. Increase frequency and quality of exposure of gilts to farm-endemic pathogens to maximise pathogen-specific immunity transferred to their progeny.
5. Consider fostering only large piglets to reduce within-litter variation.
6. Consider increasing suckling pressure (larger litters) on gilts and early in lactation for both gilts and sows to prime the mammary glands to maximize subsequent milk production.

Further research is required into:

1. Increasing the birthweight of gilt-born progeny beyond that achieved through nutrition.

2. Non-invasive measurements of piglet milk intake – not involving deuterium oxide dilution or weigh-suckle-weigh techniques.

3. The post-weaning decline in antibody concentrations of gilt progeny compared to sow progeny particularly in relation to farm-endemic pathogens.

4. Methods for improving the transfer of antibodies from serum to colostrum in gilts.

5. Cooling systems for older parity sows, compared to the current drip-cooling system, to maximise milk yield during summer.

6. Pathogen carriage between gilts and sows and their progeny in relation to other farm-endemic pathogens especially those relating to pre-weaning diarrhoea.

7. Fostering strategies to reduce within-litter variation in piglet weights and to maximise udder development of gilts during their first lactation without compromising piglet growth and survival through to market age.

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