Sow development, reproductive performance and longevity

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Introduction

Selection of a gilt for breeding is the first step in the chain of events that ultimately determines her lifetime of productivity. Unfortunately, selection for improved production attributes can be accompanied by increased sow culling rates (Tholen *et al*., 1996) and elevated mortality levels in piglets (Knol, 2001), which compromises productivity as well as welfare and profitability. On the sow side, this phenomenon is generally thought to be a consequence of selection altering the balance between nutrient partitioning for maintenance versus productivity, combined with suboptimal nutrition (Ball *et al*., 2008) to meet requirements of genetically superior sows. However, it is not well known how attributes at selection (individual phenotype) and genetic potential (EBV) are associated with subsequent body development of the sow, particularly since maternal development occurs concurrently (in competition) with reproduction. This has shifted some research emphasis to investigating traits like lactation intake, whereas sow development prior to lactation is ongoing throughout reproductive cycles (Figure 1).

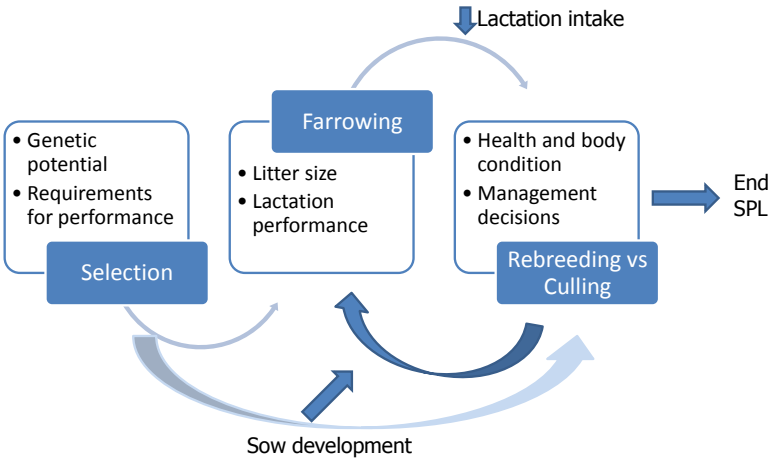

Figure 1. Connections exist between sow body development, reproductive performance and sow productive life (SPL)

Several phenotypic studies have identified low lactation feed intake, leg conformation or lameness (Anil *et al*., 2006; Deen *et al*., 2007), excessive weight and/or fat loss during lactation (Whittemore, 1996), and pre- and post-partum health issues (Hoy, 2006) as contributing factors to premature culling of sows, often manifested via their contributions to rebreeding failure. Current literature suggests production traits are relatively minor explanatory variables for sow longevity and lifetime performance. However, key areas in which knowledge of genetic associations are sparse include sow body development post-selection, feed intake attributes of sows and their association with reproductive outcomes and survival between parities. The aim of this workshop paper is to briefly

report on results from a CRC project developed to investigate these associations in detail from the gilt as a finisher pig through to her second parity.

Sow development from finisher to farrowing

1. Data characteristics

Data are described in more detail by Bunter *et al*. (2010). Briefly, performance traits included lifetime and on-test (21-26 weeks) average daily gain (LADG and TADG: g/day), back fat (BF: mm) and eye muscle depth (EMD: mm) at selection, along with average daily feed intake (ADI: kg/day) and feed conversion ratio (FCR: kg/kg) during test. Attributes recorded post-selection were sow weight (kg) and fatness (mm) at 29 weeks (WT29, FT29), at farrowing (SWPF, FT110) and at weaning (WTW, FTW), along with derived maternal weight and fat changes during the gestation (WTΔG, FTΔG) and lactation periods (WTΔL, FTΔL). Reproductive data included historical records for total born and live born piglets (TB, NBA: pigs/litter), along with more limited data for average piglet birth weight (APBW: kg/pig). Project sows were additionally recorded for total litter gain between 1 to 10 days of age (LITG: kg). Lactation intake was averaged up to day 35 of lactation (LADI: kg/day). Targeted lactation length was 30 days, and the occurrences of a shortened lactation (SHORT) in the first parity and survival to farrow in later parities (PAR2 to PAR5) was known (0/1).

To place parameters in context, project gilts weighed 142 kg at 29 weeks and had 15.2 mm fat depth, averaged across the P2 and ham (P4) sites. Maternal weight and fat gains during gestation were 51.7 kg and 2.02 mm in parity one and 47.1 kg and 0.56 mm in parity two. Pre-farrowing weight and fat measures averaged 224 kg and 19.3 mm in parity 1 and 259 kg and 18.4 mm in parity 2, demonstrating that on average sows failed to fully regain subcutaneous fat depths after weaning, during their second gestation. Sow weight was lowly variable (CV~10%). However, sow fatness was more variable (CV~20%), and fat gain during gestation was extremely variable (CV 176-602%) compared to weight gain in gestation (CV 28-40%) or weight and fat loss during lactation (CV 107-207%). Thus, mean population values do not convey the presence of high variability amongst individual sows within the population. Maternal weight and fat losses during lactation averaged 11.3 (16.8) kg and 1.99 (1.46) mm in the first (and second) parity. Piglet birth weight in each parity averaged 1.42 and 1.57 kg/pig (CV<20%) for TB of 11.6 and 12.5 pigs/litter (CV<10%). The percentages of first parity sows that farrowed in later parities were 76, 63, 51 and 42%, up to parity five.

2. Genetic parameters

Estimates of heritability and genetic correlations between performance traits and subsequent sow body composition or reproductive attributes were estimated using ASREML (Gilmour *et al*., 2006) (Table 1). Parameters for binary traits were estimated on the underlying scale using a logit link under a sire model for all trait combinations. Heritability estimates (Table 1) demonstrate that weight (h^2: 0.18-0.33) and fatness (h^2: 0.22-0.53) remain moderately heritable throughout a sow's life. Additive genetic variation for weight increased with parity, but declined with parity for fat. The heritabilities of maternal weight changes during gestation and lactation were of similar magnitude to estimates for absolute weights but variances were lower: heritability estimates for WTΔG and WTΔL were 0.15 (0.16) and 0.23 (0.20) for parity 1 (or p2).

Table 1. Estimates of additive genetic (ra) and phenotypic (rp) correlations between performance traits and sow attributes or survival to later parities

Trait			LADG		BF		EMD		TADG		ADI		FCR	
	h^2×100	σ^2p	ra	rp	ra	rp	ra	rp	ra	rp	ra	rp	ra	rp
29WT	29	150	**87**	**66**	**18**	**5**	**-16**	**-7**	**61**	**46**	**50**	**43**	-6	**-11**
29FT	53	8.32	6	**-8**	**90**	**64**	4	0	6	3	**45**	**28**	29	**18**
SWPF	24	273	**74**	**42**	-5	-2	-4	**-5**	**54**	**27**	**29**	**15**	-21	**-15**
	18	452	**62**	**32**	-12	-1	12	-5	**47**	**16**	**27**	**7**	-15	**-10**
WTW	33	285	**61**	**39**	4	-3	-2	**-8**	**60**	**26**	**47**	**19**	-15	**-11**
	27	395	**55**	**37**	-3	0	14	-5	**64**	**24**	**42**	**16**	-19	**-12**
WTΔG	15	179	7	**-5**	-13	-2	3	0	-2	**-8**	**-42**	**-18**	**-37**	**-8**
	16	295	6	-2	-14	0	11	3	-12	-5	-24	**-10**	-11	-2
WTΔL	23	194	-16	0	13	1	6	-1	22	3	24	5	-2	1
	20	249	-5	4	13	0	6	1	**37**	**8**	**35**	**12**	-1	0
FT110	33	11.9	**28**	**12**	**75**	**38**	5	-2	11	5	**46**	**17**	**31**	**9**
	22	12.0	**28**	5	**83**	**32**	11	-2	22	**6**	**54**	**11**	**36**	4
FTW	35	11.3	**17**	**11**	**73**	**41**	3	**-5**	19	**9**	**53**	**21**	**27**	**9**
	26	10.5	7	**7**	**70**	**33**	16	-2	23	**6**	**55**	**14**	**28**	**6**
FTΔG	22	9.76	**-36**	**-15**	**-37**	**-14**	16	**4**	-8	**-16**	**-33**	**-23**	-22	**-5**
	2	10.3	5	-9	-66	**-13**	39	**5**	19	-3	-83	**-11**	-63	**-6**
FTΔL	10	8.53	-18	0	-5	-4	9	-1	2	2	6	2	-3	0
	1	8.72	B	2	-17	-2	44	1	-57	0	-3	3	4	3
TB	12	10.7	-1	**7**	-4	**-3**	-4	-1	-3	**6**	1	3	7	-2
	9	8.98	**-15**	**4**	-7	-2	2	1	-6	2	-7	1	-3	-2
NBA	9	8.98	-9	**3**	**9**	0	5	0	1	**4**	1	1	5	-3
	6	7.58	**-21**	1	-1	0	11	2	-9	0	-15	-1	-7	-1
APBW	36	0.048	**47**	**7**	**-36**	**-10**	**-14**	-1	-11	5	-6	4	5	-2
	31	0.047	**55**	**5**	-30	**-6**	**-11**	-2	4	2	-1	-2	-2	-3
LITG10	8	36.1	**40**	1	-7	-2	-5	-3	-29	2	-34	0	-2	-3
	5	41.4	**39**	-3	10	**6**	21	-2	17	1	-35	-1	-55	-2
LADI	15	0.62	**42**	**9**	-11	**-6**	3	-3	14	**6**	26	**7**	10	1
	24	0.70	**50**	**14**	-18	-4	-8	-2	**34**	**10**	**39**	**10**	7	-2
SHORT	15	3.42	2	**-4**	10	3	-18	-1	38	2	26	2	-5	0
	32	3.57	35	0	12	3	-7	**-4**	**73**	2	**63**	**6**	-1	2
PAR2	6	3.34	24	0	45	**8**	-29	**3**	2	0	-42	**3**	-42	3
PAR3	8	3.36	-11	-2	37	**7**	-14	**4**	-3	-2	-39	0	-31	2
PAR4	6	3.34	-29	**-4**	**60**	**10**	-27	**4**	-14	-2	-38	0	-15	1
PAR5	14	3.41	-28	**-5**	**37**	**10**	1	**5**	-19	-3	-21	0	-2	3

See text for trait abbreviations; correlations sig. different to zero in bold; first line: parity 1 data; second line: parity 2 data; * Covariate for starting point included in the model for parity 2 data; underlined rp have opposing residual and genetic correlations.

In stark contrast, the heritability estimates were moderate (0.22) for FTΔG in parity 1, but low or negligible for FTΔG in parity 2 and for FTΔL in both parities. Therefore, ***the primary source of variation between sows in changes to fatness during gestation and lactation are not genetic in origin after parity 1.*** Grandinson *et al*. (2005), with data from considerably lighter, fatter sows, reported similar heritability estimates for weight and fat loss during lactation. Regression coefficients for FTΔG or FTΔL on fatness at the start of each period were negative indicating that fat gain in gestation was less and fat loss in lactation was more substantial for fatter sows. These coefficients were almost identical in parity 2 (-0.435±0.022 and -0.432±0.020) supporting the theory that *lactating mammals have a tendency to return to their pre-parturition body composition for fatness* (Butte and Hopkinson, 1998).

Genetic correlations for early growth (LADG) generally supported the concept that selection for growth will result in heavier sows with higher lactation intake capacity and heavier piglets at birth.

However, negative residual (not presented) and phenotypic correlations indicate that high growth sows (LADG and TADG) have restricted maternal weight and fat gain in gestation, especially in parity 1, and piglet weight is also partially compromised. ***This pattern of correlations for growth traits suggests environmental limitations to performance of sows with high genetic potential for growth.*** Further, neutral correlations between LADG or TADG with PAR2 were followed by increasingly unfavourable associations between early growth and later parity longevity, as larger sow size and higher maintenance requirements become more of a limitation with increasing parity.

Gilts that were genetically fatter at selection remained phenotypically fatter throughout repeated parities despite gaining less fat during their gestations. After fitting the initial phenotype as a covariate (results not presented), there is evidence that genetically fatter sows do retained a positive potential for fat deposition at higher initial phenotypic levels of fatness. The genetic correlation of BF with APBW was negative as expected (Hermesch *et al*., 2001), but residual correlations between BF and APBW were favourable: environmental causes of sow fatness favour a positive outcome for APBW, and also for litter gain in the second parity. The net association between BF and APBW remained negative at the phenotypic level. Genetic and phenotypic correlations indicate that ***fatter sows had consistently better survival to later parities***, extending knowledge from previous analyses where fatness prior to the first farrowing was identified as important for survival to farrow in parity 2 (Bunter *et al*., 2008).

Correlations between EMD and sow body development or reproductive characteristics were generally small. Genetically muscular gilts on a weight constant basis were phenotypically lighter and leaner, but gained more fat during gestation. The net effect on longevity to later parities was positive. Gilts with high genetic potential for feed intake between 21 and 26 weeks were heavier and fatter as sows, but with diminished weight and fat gains during gestation. Genetic correlations of ADI with litter size and birth weight traits were negligible. High finisher ADI was associated with increased LADI and diminished weight or fat loss during lactation. Of note, ***the genetic correlations between ADI and LADI were significantly lower than one, suggesting that appetite expression in the different physiological states (growing vs lactation) is controlled by different stimuli*.** The negative genetic correlations between ADI and LITG10, combined with an increased chance of a shortened lactation, suggest some antagonism of ADI with mothering performance despite the favourable association of ADI with lactation feed intake. The net associations of ADI with LITG10 or longevity were neutral phenotypically, although genetic correlations were consistently negative. Sows with high FCR tended to be lighter and fatter, with significantly lower weight and fat gain during gestation. FCR was uncorrelated with litter size or birth weight traits, but was negatively (favourably) correlated genetically with sow longevity, probably because sows were both smaller and more efficient.

Repeatability of sow attributes between consecutive farrowings

Correlations between the same trait recorded in adjacent parities (1 and 2) were also estimated. These genetic correlations were generally very high (~0.90) for weight and fat, with corresponding phenotypic correlations of 0.59 (SWPF) and 0.72 (WTW) for weight traits, and 0.50 (FT110) and 0.72 (FTW) for fatness traits. Compared to results from Table 1, sow weight and fatness prior to the first farrowing are substantially better predictors of these attributes in parity 2 than was LADG, as expected. However, phenotypic correlations for the transition traits were 0.22-0.28 for weight (WTΔG, WTΔL) and 0.03-0.10 for fatness (FTΔG, FTΔL), unless starting points for fatness were known, whereby correlations increased to about 0.20. This demonstrates overall that absolute measures of maternal weight and fatness have significant genetic and *permanent* environmental components, whereas traits indicative of changes to these sow attributes during gestation or lactation were mostly affected by *temporary* environmental effects specific to that gestation or lactation, along with the underlying genetic effects. Similarly, the within trait genetic correlations for litter size, APBW, LITG10 and LADI were very high, in the range of 0.68 (LITG10) to 0.91 (LADI), whereas phenotypic

correlations were ~0.2 for TB, NBA and LITG10, 0.28 for LADI and 0.42 for APBW. Probably the conclusion one could draw from these observations is that, for example, ***a low lactation intake is unlikely to be a permanent characteristic of the sow (over and above her genetic potential for the trait) but rather a reflection of the specific circumstances of that lactation.*** This implies a strong adaptive process between specific characteristics of a gestational outcome at the phenotypic level (eg litter size, piglet and sow attributes) and LADI, whereby genetic potential and phenotypic outcomes are not aligned.

Sow body composition, reproductive performance and longevity

Within parity and trait, correlations between measurements at the start and completion of lactation are high. Genetic correlations were 0.75 between WT110 and WTW and 0.90 for FT110 and FTW; corresponding phenotypic correlations of 0.56 and 0.63 (not presented). Further, genetic correlations of weight loss with fat loss were also very high; 0.76 in parity 1 and 0.97 in parity 2; whereas phenotypic correlations were much lower at 0.41 and 0.40. ***Since the genetic correlation between weight and fat loss is high, this supports a co-ordinated genetic mechanism for simultaneous catabolism of fat and protein to generate energy during lactation,*** although genetic variation in fat loss was limited in parity 2. Correspondingly, the correlations of weight with fatness were weaker at the start compared to the end of lactation.

The size of the litter gestated had consequences for sow body composition at farrowing in parity 1 (Table 2). Negative correlations indicate that sows gestating larger litters had lower maternal weight gain and sow fatness pre-farrowing, whereas sows producing individually heavier piglets had lower pre-farrowing fatness only. Higher APBW and LITG10 was associated with lower sow weight and fatness levels at weaning, resulting from increased weight and fat loss during lactation, despite increased LADI. Sows with reduced weight loss during lactation, but more significantly higher fat at weaning, were the most likely to farrow in later parities.

Of significance, phenotypic correlations between LADI and sow longevity traits were positive, in spite of strong negative genetic correlations. Sows with a high phenotypic lactation feed intake reared the litter more effectively and reduce their own weight or fat loss (Table 2), which are desirable outcomes. They are also more likely to be healthy (Bunter *et al*., 2009a). However, sows with higher genetic potential for lactation feed intake are larger and leaner with a neutral genetic capacity for rearing a litter, once birth weight is accounted for (Table 1). This might explain the apparently counter-intuitive results of both Bergsma *et al*. (2008) and (Bunter *et al*., 2009b), showing antagonistic genetic, but favourable phenotypic correlations, between *ad-libitum* lactation intake and sow longevity. In this study, higher sow weights were beneficial in early parities and indeed, for successful entry into the herd in the first place (not presented). But they were increasingly less beneficial in later parities where the nutritional demands of prolific and heavier sows are less likely to be met. The transition of sow weight with increasing parity from a beneficial to a detrimental effect (Table 2), and the inconsistency between genetic and phenotypic correlations (eg LADI with longevity) serve to mask important associations between these traits and sow longevity because of non-linear relationships.

Table 2. Estimates of genetic (ra) and phenotypic (rp) correlations between sow body composition attributes, reproductive traits and sow survival to later parities

Trait	SWPF		FT110		LADI		WTW		FTW		WTΔL		FTΔL	
	ra	rp	ra	rp	ra	rp	ra	rp	ra	rp	ra	rp	ra	rp
TB	-7	-3	-13	**-9**	1	**7**	-8	2	0	-1	-3	**9**	**35**	**11**
	-16	2	-10	1	-21	3	-5	3	-11	-3	14	2	11	-2
NBA	1	**-4**	4	**-4**	-3	**8**	-5	1	9	1	-5	**7**	21	**7**
	-21	0	-7	1	-30	4	-17	1	-17	-2	3	2	-27	-2
APBW	**45**	**22**	-8	**-8**	13	-2	-12	**-8**	-17	**-13**	**-72**	**-37**	-19	**-11**
	9	**18**	-14	-2	35	**10**	-18	-4	**-38**	**-12**	**-36**	**-29**	-64	**-9**
LITG10	-14	2	-10	-1	6	**16**	**-38**	**-17**	-27	**-20**	**-31**	**-23**	-42	**-22**
	-58	3	-23	**7**	2	**25**	**-**	**-16**	-43	**-10**	-60	**-26**	-43	**-21**
LADI	33	**-9**	-16	**-12**	-	**-**	**56**	**38**	20	**16**	**43**	**54**	**87**	**33**
	-26	**-16**	-19	**-13**	-	**-**	21	**21**	-12	3	**65**	**47**	57	**18**
PAR2	9	0	46	**8**	13	**9**	20	**10**	24	**14**	33	**15**	-24	**7**
PAR3	2	-2	41	**8**	-14	**7**	3	**4**	24	**12**	19	**9**	-32	**4**
	18	-1	56	**6**	**-74**	**13**	8	**8**	44	**13**	4	**12**	-	**7**
PAR4	-18	-3	**69**	**9**	-50	**6**	4	1	46	**11**	39	**5**	-44	1
	17	-5	58	**7**	**-96**	**6**	0	1	62	**9**	-14	**7**	-	1
PAR5	-10	-4	54	**10**	-35	**5**	-5	-2	25	**10**	12	**4**	-48	-1
	10	-3	33	**9**	**-74**	**4**	-2	-1	41	**10**	-15	3	-	0

See text for trait abbreviations; 1st line: parity 1 data; 2nd line: parity 2 data. Values ×100

Take home messages

1. Selection to improve production traits has consequences for the ongoing body development of sows, their longevity, and the pre-natal development and pre-weaning performance of their progeny.
    - Selection changes average nutrition and management requirements in populations, and commercial producers need to keep up with these changes
2. There are some strong antagonistic genetic correlations to contend with across the complete trait complex
    - a more complete model that aligns genetic potential with prevailing environmental constraints is required to achieve desired phenotypic outcomes across the full trait complex.
    - Breeding goals need to be expanded to include sow longevity, but some potential selection criteria to improve longevity (eg sow fatness) are antagonistically associated with other components (eg production traits).
3. Alternative selection criteria, such as fat gain in gestation and fat loss in lactation, have limited genetic variation under normal management practices.
    - Non-genetic avenues for improvement of sow longevity and lifetime performance might be to develop management strategies for turning genetically lean sows into phenotypically fatter sows prior to their first farrowing.
4. Reducing reliance on lactation feed intake to manage sow body condition and litter gains is desirable since selection for increased lactation intake will likely have the undesired result of larger sows.

- Feeding to individual requirements during gestation could limit the necessity of very high lactation intakes, and would ensure adequate sow body reserves prior to the farrowing event, when other (non-genetic) factors might limit intake (eg health or heat!).
- Non genetic factors (not all of which are recorded) have large effects on lactation intake, and it is these factors that drive the positive phenotypic association between lactation intake and sow longevity.

5. Within a population, there is variation in both maternal requirements and litter size to contend with. Competition for resources exists at two levels: between the growing sow and her litter and between litter mates during gestation.
   - Feeding strategies that better meet requirements of individual sows and their litters during both gestation and lactation could potentially improve sow longevity, along with piglet survival and performance, particularly during the first lactation.

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