Maternal Capacity, Feed Intake and Body Development in Sows

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Introduction

Selection of a gilt for breeding is the first step in the chain of events that determines sow survival within the herd and ultimately 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 both 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 for all requirements of genetically superior sows (Ball et al. 2008), which has consequences for sow phenotype.

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, with the possible exception of the association between lean growth and the development of osteochondrosis (Stern et al. 1995). 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 paper is to report on results from a specific project developed to investigate these associations in detail from the finisher pig through to the second parity.

From gilt to sow (teenager to adult)

Mature size characteristics, reproductive performance and longevity of a genetically similar sample of gilts can be altered by varying the environment and management under which the gilts are reared both prior to herd entry and throughout subsequent reproductive cycles. What is not well quantified is 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.

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For results presented in this paper, data were recorded on maternal line purebred project gilts at selection (20 weeks), for selected gilts during additional *ad-libitum* feed intake testing (21-26 weeks: N~3500) and before mating (29 weeks), and for mated gilts at farrowing in parities one (N~2200) and two (N~1450). Following selection, there was a 9-week period of gilt development, intended to allow functional attainment of puberty and an increase in physiological maturity and development pre-mating. Diets were altered from finisher to gilt development diets in this period and gilts remained group housed and fed *ad-libitum*. Gilts were then transferred at 29 weeks to the facility for breeding. Feeding during gestation was to a fixed delivery schedule according to stage of gestation, with no systematic adaptation to variation in sow size or condition. Feed delivery during lactation was 3×daily and 'to demand', to maximise expression of lactation intake. A complete inventory ensured that culling dates and reasons for culling were known, and medication events for sows and their litters were also recorded.

To place parameters in context, current gilts in this selected population were generally characterised by higher lean growth potential and a reduced propensity for fat deposition as a finisher compared to historical counterparts, along with increased demands during gestation and lactation through both increased litter size and piglet growth potential. 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 values do not convey the high variability amongst individuals. Maternal weight and fat losses during lactation were 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.

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. Historical performance data for LADG, BF, and EMD (N~56000) were included in analyses for (REML) parameter estimation to accommodate effects of prior selection. 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). 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 and fatness remain moderately heritable throughout a sow's life: heritability estimates (×100) for LADG, 29WT, SWPF (parity 2: p2) and WTW (p2) were 21, 29, 24 (18) and 33 (27); additive genetic variation for weight increased with parity. Corresponding heritability estimates for BF, 29FT, FT110 and FTW were 38, 53, 33 (22) and 35 (26), but additive genetic variation declined with parity. The heritabilities of maternal weight changes during gestation and lactation (15 and 5 week periods respectively) were of similar magnitude to estimates for weights but variances were lower: heritability estimates for WTΔG and WTΔL were 15 (16) and 23 (20). In stark contrast, the heritability estimates were moderate (22) for FTΔG in parity 1, but low or negligible for FTΔG in parity 2 and for FTΔL in both parities. Grandinson et al. (2005), with data from considerably lighter, fatter sows than in this study, reported similar heritability estimates for weight and fat loss during lactation. In parity 2 data, genetic variation for FTΔG and FTΔL was negligible unless covariates for fatness at mating or at the start of lactation were concurrently fitted in the model, suggesting that the genetic variation in these traits was due to variable expression relative to a common phenotypic starting point. This implies that the underlying genetic potential of the sow for fat gain was largely unable to be expressed during the second gestation. 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 heavier piglets and higher lactation intake capacity. However, negative residual (not presented) and phenotypic correlations indicate that high growth sows (LADG and FADG) have reduced maternal weight and fat gain in gestation, especially in parity 1. Compared to LADG, correlations between FADG and APBW were negligible while the associations with WTΔG or FTΔG were stronger. While the overall phenotypic association of growth with birth weight was positive, the residual correlations also indicate that piglet weight was negatively affected.

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

Trait	$h^2\times100$	σ^2p	LADG 21 4789		BF 38 3.47		EMD 19 20.9		FADG 16 21848		ADI 25 0.14		FCR 25 0.24	
			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**
*	8	8.13	42	-3	**61**	**11**	26	2	33	1	29	-1	15	-2
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
*	7	6.40	-22	5	**68**	**19**	26	-1	1	3	**55**	**10**	37	**6**
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.

This pattern of correlations for growth traits suggests environmental limitations to performance of sows with high genetic potential for growth. 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 gestation. After fitting the initial phenotype as a covariate, there is evidence that genetically fatter sows 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.

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

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 at 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.

Relationships between sow body composition at farrowing, 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 litter 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 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

Conclusions

Selection for finisher traits to improve production has consequences for the ongoing body development of sows, their longevity, and the pre-natal development and pre-weaning

performance of their progeny. There are some strong antagonistic genetic correlations to contend with across this trait complex. Therefore, a more complete model that aligns genetic potential with management and the prevailing environmental constraints to achieve desired phenotypic outcomes is urgently required.

If individual sow nutritional requirements are not met, pre-farrowing maternal weight gain and fat deposition will be constrained, which may lead to reduced piglet birth weight, particularly in the first parity. These outcomes influence sow longevity, along with piglet survival and performance, particularly during first lactation. Some potential selection criteria, such as fat gain in gestation and fat loss in lactation, have limited genetic variation under current management. Non-genetic avenues for improvement of sow longevity and lifetime performance might therefore be to develop management strategies for turning genetically lean sows into phenotypically fatter sows prior to their first farrowing, and towards feeding strategies that better meet requirements of individual sows and their litters during both gestation and lactation. This reduces reliance on lactation feed intake to manage sow body condition and litter gains, which is desirable since selection for increased LADI (a strategy which has been suggested) will likely have the undesired result of larger sows.

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