

VARIATION IN SOW HEALTH AFFECTS THE INFORMATION PROVIDED BY LACTATION FEED INTAKE DATA

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SUMMARY

Using data from two maternal lines of pigs (N~2200), medication events were used as proxy indicators of sow health, to examine changes in associations between lactation feed intake and other traits that occur with changes to sow health status. Estimates of heritability for total born, average piglet birth weight, litter gain until day 10 (LG10), average sow feed intake during lactation (LFI), total sow feed intake during the first three days of lactation, lactation length and the underlying liability for a shortened lactation (SL) or surviving to farrow in parity 2 (FP2) were 0.14 ± 0.01 , 0.33 ± 0.03 , 0.09 ± 0.04 , 0.18 ± 0.04 , 0.06 ± 0.04 , 0.06 ± 0.03 , 0.15 ± 0.09 and 0.06 ± 0.07 . Genetic (r_a) and phenotypic (r_p) correlations indicate that high lactation feed intake was favourably associated with SL and FP2 (r_a : -0.78 ± 0.19 and 0.42 ± 0.41 ; r_p : -0.49 ± 0.01 and 0.31 ± 0.01). Compared to estimates obtained using only subsets of data from sows that met lactation length targets or unmedicated sows, heritabilities for LFI were higher in the medicated data set and phenotypic correlations with LG10, SL or FP2 were of increasingly larger magnitude across these data subsets. Sow health status affects the information content of lactation feed intake data, but larger studies will be required to confirm if significant changes also occur in genetic parameters because of health status. Knowledge of health status could be important for other studies which examine associations between feed intake, recorded in any physiological state, and other production traits.

INTRODUCTION

Studies have shown that sows with higher lactation feed intake rear heavier piglets, have better body condition at weaning, and are more likely to farrow in the subsequent parity (Hermesch and Jones 2007; Bergmsa *et al.* 2008), but knowledge of the genetic associations between lactation feed intake and sow performance is relatively limited. In addition, where lactation feed intake data are available the health status is typically unknown. It is therefore unclear how much phenotypic or genetic associations with feed intake are influenced by prevailing acute health issues, which affect feed intake, compared to variation in feed intake in normal healthy sows. In this study we use medication events as proxy indicators of sow health to create data subsets for lactation records, to examine changes in parameters that occur with changes to sow health status.

MATERIALS AND METHODS

Approximately 2200 sows from two maternal lines (Primegro™ Genetics, Large White and Landrace based) were recorded for their first gestation and farrowing outcomes between January 2007 and June 2008. Data in this study included total number of piglets born (TB), average piglet birth weight (ABW) of live born piglets, along with litter gain from day 1 (after cross fostering) until day 10 (LG10), the average daily lactation feed intake of the sow (LFI) up to 35 days, total feed intake in the first three days of lactation (FID3), and the lactation length of the sow (LACL). Events such as a shortened primiparous lactation (SL) and farrowing in parity 2 (FP2) were treated as binary traits (0/1, where event=1) for uncensored sows. Continuous data were subsequently

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edited based on trait distributions. Proc UNIVARIATE (SAS, 2003, SAS Institute Inc. Cary, N.C.) was used to identify outliers, whereby trait records that deviated by more than $3\times$ the interquartile range from the mean value were deleted. After editing, there were data from 2164 animals representing 197 sires and 1221 dams. The pedigree was extended back to include all animals born since 2003 (N=44950).

Subsets of the complete data (ALL) for feed intake records (LFI, LACL, FID3) were then developed based on LACL and specific health information; data for other traits remained constant. The targeted lactation length and weaning age in this data was 28-32 days, with a maximum weaning age of 35 days. Subset 1 contained sows with $LACL > 21$ (N=1778); subset 2 contained sows with no evidence of medication events during lactation (N=1030); and subset 3 contained sows that received any non-feed medication within 5 days prior to farrowing, until 35 days post-farrowing (N=1008). As with other species, primiparous sows frequently have more periparturient and post-farrowing difficulties than multi-parous sows, and several medication categories were grouped together over this time period. The heritability estimate for a medication event in this data was zero.

Models for analyses were developed using ASReml software (Gilmour *et al.* 2006). Univariate analyses were used to identify significant fixed effects and to obtain initial estimates of genetic parameters under either an animal (continuous traits) or sire (binary traits) model. Parameter estimates for binary traits were estimated on the underlying scale using a logit link function. Systematic effects for all traits included year/month of farrowing (levels: 19) and sow line (2 levels). In contrast to Bunter *et al.* (2009) lactation length was excluded as a covariate for lactation intake in this study, so that lactation length could be examined as a trait. Correlations between specific traits were estimated in a series of bivariate analyses. Bivariate analyses of trait combinations involving any binary trait were all performed using a sire model for both traits.

RESULTS AND DISCUSSION

Characteristics of the data. Focusing on less common traits, feed intake in the first three days of lactation was more variable (CV=37%) than LFI (CV=22%) despite consistent $3\times$ daily feed delivery schedules in the postfarrowing period (Table 1). Unmedicated sows ate significantly more feed per day in the first three days after farrowing and over the complete lactation than medicated sows, but lactation lengths were similar (Table 2). This is consistent with subjective observation that animals with health issues have reduced intake (Weary *et al.* 2009) but can also indicate that low feed intake was a cue for initiating treatment. Variability in lactation traits was also 17 to 25% higher in the medicated data set relative to the unmedicated data set. The incidence of shortened lactations in primiparous sows was 13%; many of these affected sows were not given an opportunity to re-mate. Seventy three percent of farrowed sows had a second parity. However, this is an underestimate as some sows were transferred after their first parity, and were generally confirmed pregnant at transfer.

Heritability estimates. The estimate of heritability for LFI (0.18 ± 0.04) was similar to the estimate of 0.16 ± 0.04 from Bunter *et al.* (2009) where LACL was used as a covariate for LFI, and comparable to other studies (Bergsma *et al.* 2008; Hermes 2007). In this study, low estimates of heritability were also obtained for FID3 (0.06 ± 0.04) and LACL (0.06 ± 0.03), whereas lactation length was not heritable in the data of Bergsma *et al.* (2008) or Hermes (pers. comm.). Heritabilities for LFI or FID3 were higher in the subset of medicated sows; genetic variation for appetite variation may be better expressed during lactation under more challenging environmental conditions (Table 2, subset 2 vs 3).

The frequency of progression to the second parity and shortened lactations were lowly to

moderately heritable (0.06 ± 0.07 and 0.15 ± 0.09) when estimated on the underlying liability scales, although standard errors of estimates were large. For comparison, heritability estimates for TB and sow survival to parity 2 were 0.13 ± 0.02 and 0.05 ± 0.02 in the study of Bergsma *et al.* (2008), the latter estimated on the observed scale from a population with an incidence of 85.1%. Genetic parameters for TB and ABW were consistent with averages from the review of Rothschild and Bidanel (1998).

Table 1. Characteristics of the data after editing for outliers, along with parameter estimates

| Trait | Abbreviation | N | Mean (SD) | Min-Max | h^2 | σ_n |
|--|--------------|------|-------------|---------------|----------------|------------|
| Total born (N) | TB | 2163 | 11.6 (3.20) | 2-21 | 0.14 ± 0.01 | 2.82 |
| Average piglet birth weight (kg) | ABW | 2099 | 1.41 (0.24) | 0.63-2.42 | 0.33 ± 0.03 | 0.24 |
| Lactation feed intake (kg/day) | LFI | 2031 | 4.99 (1.10) | 0.50-9.00 | 0.18 ± 0.04 | 0.94 |
| Total feed intake in first 3 days (kg) | FID3 | 2020 | 11.4 (4.20) | 0-27 | 0.06 ± 0.04 | 3.73 |
| Lactation length (days) | LACL | 2038 | 27.3 (7.47) | 0-35 | 0.06 ± 0.03 | 7.35 |
| Litter gain to 10 days (kg) | LG10 | 1885 | 10.0 (6.08) | -12.5 to 32.5 | 0.09 ± 0.04 | 5.98 |
| Shortened lactation (0/1, event=1) | SL | 2047 | 13% | 0 and 1 | 0.15 ± 0.09 | 1.85 |
| Farrowed in parity 2 (0/1, event=1) | FP2 | 2040 | 73% | 0 and 1 | 0.06 ± 0.07 | 1.83 |

Table 2. Characteristics and heritability estimates from data subsets, along with genetic (1st row) and phenotypic (2nd row) correlations (se_{subset}) (all parameter estimates and $se \times 100$)

| Trait | LFI | | | | FID3 | | | | LACL | | | | |
|--------------------|--------------------------|-------------------------|--------------------------|--------------------------|-------------------------|-------------------------|------------------------|-------------------------|--------------------------|-------------------------|-------------------------|--------------------------|---|
| | Subset | ALL | 1 | 2 | 3 | ALL | 1 | 2 | 3 | ALL | 1 | 2 | 3 |
| N | 2031 | 1778 | 1029 | 1002 | 2020 | 1778 | 1021 | 999 | 2038 | 1778 | 1030 | 1008 | |
| Mean _{SD} | 4.99 _{1.1} | 5.19 _{0.9} | 5.16 _{1.0} | 4.81 _{1.2} | 11.4 _{4.2} | 11.6 _{4.1} | 12.3 _{3.9} | 10.4 _{4.3} | 27.3 _{7.5} | 29.9 _{2.9} | 27.9 _{7.1} | 26.6 _{7.8} | |
| h^2 | 18 ₄ | 9 ₄ | 6 ₆ | 23 ₈ | 6 ₄ | 4 ₄ | 0 ₅ | 19 ₈ | 6 ₃ | 11 ₄ | 9 ₆ | 5 ₆ | |
| σ_p | 0.94 | 0.72 | 0.74 | 0.80 | 3.73 | 3.63 | 3.49 | 3.90 | 7.35 | 2.66 | 6.97 | 7.70 | |
| Correlations | | | | | | | | | | | | | |
| TB | -14 ₁₃ | 9 ₁₉ | 22 ₂₂ | -26 ₁₄ | -6 ₂₀ | 3 ₂₄ | NE | -31 ₁₇ | -46 ₂₀ | -27 ₁₆ | -21 ₂₁ | -66 ₃₇ | |
| | 3 ₂ | 8 ₂ | 4 ₃ | 3 ₃ | 0 ₂ | 1 ₂ | -1 ₃ | 1 ₃ | -3 ₂ | 2 ₂ | -4 ₃ | -2 ₃ | |
| ABW | 33 ₁₂ | 42 ₁₈ | 66 ₂₄ | 19 ₁₄ | -24 ₂₀ | -46 ₂₉ | NE | -17 ₁₆ | 49 ₁₉ | 44 ₁₆ | 50 ₂₂ | 43 ₃₀ | |
| | 2 ₂ | -1 ₂ | 2 ₃ | 1 ₃ | -10 ₂ | -11 ₂ | -8 ₃ | -12 ₃ | 7 ₂ | 5 ₃ | 6 ₃ | 8 ₃ | |
| LG10 | 55 ₁₈ | 4 ₃₂ | 18 ₃₃ | 66 ₂₄ | 16 ₃₅ | -21 ₄₅ | NE | 21 ₃₀ | 138 ₃₄ | 55 ₂₅ | 79 ₂₇ | NR | |
| | 34 ₂ | 23 ₂ | 33 ₃ | 30 ₃ | 6 ₂ | 6 ₂ | 2 ₃ | 6 ₃ | 46 ₂ | 22 ₂ | 47 ₃ | 36 ₃ | |
| SL | -78 ₁₉ | -72 ₃₆ | -65 ₃₁ | -90 ₂₃ | -52 ₅₁ | -35 ₇₂ | NE | -25 ₄₂ | NE | NE | NE | NE | |
| | -49 ₁ | -33 ₄ | -38 ₂ | -52 ₂ | -11 ₁ | -1 ₄ | 8 ₁ | -10 ₂ | NE | NE | NE | NE | |
| FP2 | 42 ₄₁ | 69 ₇₃ | -55 ₇₀ | 116 ₄₇ | 39 ₇₂ | 30 ₉₇ | NE | 78 ₆₁ | 73 ₄₀ | 88 ₅₅ | 72 ₆₉ | 122 ₅₆ | |
| | 31 ₁ | 15 ₂ | 25 ₂ | 27 ₂ | 10 ₁ | 7 ₂ | 6 ₂ | 10 ₂ | 42 ₁ | 9 ₂ | 37 ₂ | 35 ₂ | |

Trait abbreviations are in Table 1. NE= Not estimated; NR: Not reported ($se \times 100 > 100$). Estimates are in bold if significantly ($P < 0.05$) different to zero.

Correlations between traits. LFI had positive genetic correlations (r_a) with ABW, LG10 and FP2 (range 0.33 to 0.55) and the direction of correlations was generally similar across data subsets. Phenotypic correlations (r_p) between LFI and LG10 or FP2 were also moderate (around 0.30) supporting the positive associations typically observed between these traits. However, similar to Bergsma *et al.* (2008) a negative genetic correlation was estimated between LFI and FP2 in data from unmedicated sows. This outcome might arise under a positive relationship between feed intake and elevated maintenance requirements. In contrast, the estimate (r_a) over all data was positive although not significantly different to zero. Correlations (r_a and r_p) between LFI and SL

were moderately negative indicating sows that had high intake were less likely to have shortened lactations. The magnitude of correlations between LFI and SL or FP2 was largest in the medicated data set. The pattern of correlations between FID3 and LG10 or FP2 were similar to that for LFI, but lower magnitude. However, the phenotypic correlations between FID3 and SL were opposite in sign in data subsets 2 vs 3. Correlations between FID3 and ABW were negative indicating that sows farrowing heavier piglets ate less initially, despite genetic potential to consume more feed over the complete lactation. Correlations between LACL and ABW, LG10 and FP2 were consistently positive. High ABW and LG10 may promote initiation and continuation of lactation, by virtue of increased suckling stimulus of more robust piglets, and a longer lactation can assist with rebreeding performance (eg. see Tholen *et al.* 1996). Some estimates of genetic correlations involving LG10 or FP2 exceeded the upper boundary of the parameter space, predominantly in the medicated data subset.

Phenotypic correlations between LFI or LACL with LG10, SL or FP2 were of smaller magnitude in the data subset containing sows with lactation lengths longer than 21 days. This implies that the stronger phenotypic correlations observed in subsets 2 and 3 arise partly from partial lactation data for the 13% of sows which did not achieve the targeted lactation length. There were generally no significant correlations between TB and lactation traits, with the exception that for sows with normal lactation lengths (subset 1) the association between TB and LFI was low and positive. Bivariate analyses involving LACL and SL generally failed to converge, since SL was described as subsets of LACL.

CONCLUSIONS

Relative to unmedicated sows, LFI and FID3 of medicated sows was lower, more variable and more heritable, and correlations between feed intake traits and the prevalence of shortened lactations or survival to the second parity were of larger magnitude. Therefore, high lactation feed intake is relatively more important to sow and piglet survival in the presence of sow health issues, which was not a heritable trait in itself in this data. Standard errors of genetic correlations were relatively large such that only phenotypic correlations significantly differed between these data subsets. Recording individual health status should be considered important for studies involving feed intake; a larger study will provide more accurate estimates of genetic correlations under different health circumstances.

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REFERENCES

- Bergsma, R., Kanis, E., Verstegen, M.W.A. and Knol, E.F. (2008) *J. Anim. Sci.* **86**:1067.
Bunter, K.L., Luxford, B.G., Smits, R., and Hermes, S. (2009) *Proc. Assoc. Advmt. Anim. Breed. Genet.* **18**:203.
Hermesch, S. and Jones, R.M. (2007) *Proc. Aust. Pig Sci. Assoc.* **11**:196.
Hermesch, S. (2007) *Proc. Assoc. Advmt. Anim. Breed. Genet.* **17**:61.
Rothschild, M.F. and Bidanel, J.P. (1998) In "The Genetics of the Pig", p. 313, Eds. M.F. Rothschild and A. Ruvinsky, CAB International.
Gilmour, A.R., Cullis, B.R., Welham, S.J. and Thompson, R. (2006) "ASReml User Guide", Release 2.0 VSN International Ltd, Hemel Hempstead, HP1 1 ES, UK.
Tholen, E., Bunter, K.L., Hermesch, S. and Graser, H.-U. (1996) *Aust. J. Agric. Res.* **47**:1261.
Weary, D.M., Huzzey, J.M. and Keyserlingk, M.A.G. von (2009) *J. Anim. Sci.* **87**:770.