

## LATE GESTATION HEALTH STATUS IS CORRELATED WITH LACTATION OUTCOMES FOR SOWS

L. Vargovic<sup>1</sup>, K.L. Bunter<sup>1</sup>, S. Hermes<sup>1</sup>, J. Harper<sup>2</sup> and R. Sokolinski<sup>3</sup>

<sup>1</sup>Animal Genetics & Breeding Unit\*, University of New England, Armidale, NSW, 2351 Australia

<sup>2</sup>Rivalea Australia Pty Ltd, Corowa, NSW, 2646, Australia

<sup>3</sup>PIC Australia, Grong Grong, NSW, 2652 Australia

### SUMMARY

Gilts and sows from two nucleus farms (N=1103) were recorded after transfer to the farrowing shed for a range of health-related traits and subsequent lactation outcomes. Traits recorded pre-farrowing included fight lesions (FIGHT), caliper score (CAL), udder condition (MAST), haemoglobin level (HB), respiration rate (RESP), rectal temperature (RECT) and feed refusal before farrowing (FRBF). Lactation outcomes included the number of weaned piglets (NWEAN) and lactation failure (LFAIL). The highest heritabilities ( $h^2$ ) were estimated for CAL ( $0.34 \pm 0.08$ ), FRBF ( $0.21 \pm 0.08$ ) and RESP ( $0.20 \pm 0.09$ ), while the remaining traits were lowly heritable. Antagonistic genetic (rg) and/or phenotypic (rp) correlations were estimated for NWEAN with FRBF (rg:  $-0.36 \pm 0.30$ ; rp:  $-0.10 \pm 0.03$ ) and for CAL with HB (rg:  $0.33 \pm 0.41$ ; rp:  $0.15 \pm 0.03$ ). The absence of pre-farrowing mastitis was associated with higher NWEAN both genetically ( $-0.74 \pm 0.30$ ) and phenotypically ( $-0.05 \pm 0.03$ ), indicating that selection for healthy udder led to increase in NWEAN. Sows with higher levels of HB and fewer feed refusals had increased NWEAN. Non-zero heritabilities demonstrate that health-related traits have a genetic component, but evaluation of their potential use as selection criteria to improve lactation outcomes for sows requires additional data to obtain more accurate estimates of genetic correlations.

### INTRODUCTION

Lactation outcomes can be defined by the number of weaned piglets, lactation length or removal reasons related to poor mothering ability. Selection for litter size in pigs is aimed at increasing the number of weaned piglets, which can have detrimental effects for health of both sows and piglets and lead to a poor lactation outcome. Previous studies reported genetic associations between piglet survival and traits such as body condition, fight lesions, appetite or rectal temperatures of sows (Tabuaciri *et al.* 2010). In a phenotypic study, Anil *et al.* (2008) reported negative correlations between lactation outcomes and lactation feed intake, elevated rectal temperature or health issues.

The objective of this study was to test whether health traits (haemoglobin, fight lesions, respiration rate, mastitis, rectal temperature, appetite or body condition) were heritable and accompanied by negative genetic correlations with lactation outcomes. The hypothesis was that those traits are heritable and can be considered for developing breeding goals that balance high production performance with improved health and welfare of sows and piglets.

### MATERIALS AND METHODS

**Data.** The data used in this study were recorded at two nucleus farms operated by independent companies, collected during the period October-December 2017 (Farm A, N=558 sows) and March-June 2018 (Farm B, N=545 sows). The sows recorded included both primi- and multi-parous sows and represented a total of 10 (maternal or terminal) lines across both farms. Farms differed generally in their production environment, management, housing, feeding regimes and health status, which are not described further here. Sows were transferred from gestation housing to the farrowing shed at an

\* A joint venture of NSW Department of Primary Industries and the University of New England

average gestation length of 110 days and recorded for a range of health and welfare characteristics by a single operator. Subsequently, sows farrowed naturally and were managed according to each farm's commercial protocols. The targeted lactation lengths were four (Farm A) and three weeks (Farm B).

**Late gestation characteristics.** The extent of fight lesions (FIGHT) was scored as 0: no lesions; 1: 1-5 lesions; 2: 6-10 lesions; and 3: 10+ lesions (Bunter 2017). Body condition (CAL) was measured as caliper increments, using procedures described by Knauer *et al.* (2015), with increasing value corresponding to increasing body condition. Udder health was assessed by recording pre-farrowing mastitis (MAST, 0/1), considered to be present (score=1) for sows with a hard and swollen udder, irrespective of whether this was accompanied by an elevated rectal temperature. Resting respiration rate (RESP) was recorded as the number of expirations per 30 seconds, expressed per minute. Rectal temperatures (RECT) were obtained when sows were at rest ensuring the thermometer was in contact with the bowel wall. Haemoglobin (HB) level was measured using the Hemocue H201+ (HemoVue AB, Angeloholm, Sweden) using a single drop of blood obtained from a skin prick on the sow's ear (Hermesch and Tickle 2012). Sows which farrowed prior to the measurement date or which appeared distressed at the time of procedure were not sampled for HB. Feed refusal before farrowing (FRBF) was recorded as the proportion of days observed where less than half the meal was eaten, assessed 3-4 hours after the first feed delivery in the morning. Sows were observed for FRBF for  $5.62 \pm 2.14$  days, on average.

**Lactation outcomes.** Lactation failure (LFAIL, 0/1) was defined to occur (score=1) for any combination of: weaned piglets <7; lactation length <15 days; or if removal reasons included poor mothering ability, bad udder or no milk. A trait frequently used to describe lactation performance is the number of weaned piglets (NWEAN). Sows which weaned no piglets (due to piglet deaths) or had all piglets removed prematurely were assigned NWEAN=0. For sows which were used to foster a second litter (N=4), NWEAN was based on the first litter only. If the sow did not lactate at all (culled or died), LFAIL and NWEAN were considered missing (N=3). Records clearly identified with recording errors were excluded from analyses (N=12).

**Analyses.** Data preparation and summary statistics were obtained using R (R Core Team 2018). Raw data were firstly examined for errors and outliers, which were excluded from analyses (HB: N=4) if trait values were more than four standard deviations from the mean, within farm. The combined farm dataset was then used for analyses. Estimates of variance components were obtained by fitting a linear mixed animal model using residual maximum likelihood procedures in ASReml (Gilmour *et al.* 2014). Systematic effects fitted for all traits included parity group (4 levels: parities 1, 2, 3-4 and >4) and the interaction between breed and farm (10 levels). Estimates for heritabilities were obtained from univariate analyses. Correlations between traits were estimated using a series of bivariate analyses. Sows were progeny of 352 sires and 852 dams, and the pedigree was extended over 5 generations to contain 1261 sires and 3274 dams in total. There were 104 commercial sows without pedigree retained in the data.

## RESULTS AND DISCUSSION

**Characteristics of the data.** The incidence of sows which experienced undesirable lactation outcomes (LFAIL) was <10% (Table 1), consistent with results from a different population (Bunter *et al.* 2018). Fight lesions were observed on a relatively high percentage of sows, demonstrating aggression exists amongst group-housed sows in late gestation. The average value for HB was 106 g/l, with 2.71% of sows considered borderline anaemic (< 80 g/l). The average values for HB align with previous study by Hermesch and Tickle (2012). The extent of feed refusal was variable (CV=141%), with an average of 20% of meals observed pre-farrowing with feed refused.

**Table 1. Raw data characteristics, distribution (%×100) of scores, estimates of heritability ( $h^2$ ) and phenotypic variance ( $\sigma_p^2$ ) from univariate model, with model  $R^2$** 

Trait	N	Mean (SD)	CV%	Distribution of scores				h2 (SE)	$\sigma_p^2$	R2 (%)
				0	1	2	3			
NWEAN	1088	9.38 (2.62)	28	na	na	na	na	0.16 (0.08)	6.65	4.50
LFAIL	1100	na	na	90.2	9.8	na	na	0.09 (0.08)	0.09	2.51
CAL	1098	14.4 (2.66)	19	na	na	na	na	0.34 (0.08)	5.90	16.5
FIGHT	1103	na	na	26.5	36.3	26.2	11.0	0.14 (0.07)	0.65	29.3
MAST	1103	na	na	93.7	6.3	na	na	0.15 (0.08)	0.52	10.8
RESP	1067	25.4 (16.7)	68	na	na	na	na	0.20 (0.09)	225	19.6
RECT	1067	37.8 (0.47)	1	na	na	na	na	0.12 (0.08)	0.19	13.6
HB	960	106 (14.0)	13	na	na	na	na	0.06 (0.07)	171	12.9
FRBF	1076	0.20 (0.28)	141	na	na	na	na	0.21 (0.08)	0.80	0.56

Abbreviations: NWEAN: count of weaned piglets, LFAIL: lactation failure (0/1), CAL: caliper increments (count), FIGHT: fight lesion scores (0-3), MAST: pre-farrowing mastitis (0/1), RESP: count of expirations/minute, RECT: rectal temperature (°C), HB: haemoglobin level (g/l), FRBF: proportion of days observed where less than half the meal was eaten, na: not applicable

**Heritability estimates.** Overall, results presented in Table 1 demonstrate genetic contributions to performance (LFAIL, NWEAN), as well as feeding or interactive behaviours (FRBF, FIGHT), health or condition (MAST, CAL), and physiological traits (RESP, RECT, HB) recorded prior to farrowing. LFAIL and NWEAN were two traits for assessing sow performance as nursing sow. Heritability estimate for LFAIL was  $0.09 \pm 0.08$ , which was higher than previously reported ( $h^2 = 0.00$ ) for crossbred sows (Bunter *et al.* 2018). The heritability estimate for NWEAN was higher ( $0.16 \pm 0.08$ ) than the average of 0.07 reported in the review of Rydhmer (2000), and is potentially influenced by the minimum cross-fostering, diversity of lines, combined with phenotypes which included zero values for sows which weaned no piglets. Moderate  $h^2$  ( $0.21 \pm 0.08$ ) for FRBF suggests that when sows are observed pre-farrowing for feed refusals following fixed delivery, phenotypic differences between animals may be accurately observed, revealing differences in appetite before farrowing. Estimate of heritability for CAL was high ( $0.34 \pm 0.08$ ), consistent with similar traits like sow weight or back fat (Tabuaciri *et al.* 2010). Heritability for FIGHT was moderate ( $0.14 \pm 0.07$ ) and align with previously reported by Bunter (2017).

**Correlations.** Large genetic ( $-0.97 \pm 0.18$ ) and residual ( $-0.73 \pm 0.03$ ) correlations between NWEAN and LFAIL are consistent with the use of NWEAN to define LFAIL phenotypes (Table 2). All other correlations were of lesser magnitude. Genetic correlations were only consistent in direction or magnitude with phenotypic correlations for some trait combinations, which probably reflects relatively small sample size. The genetic correlation between NWEAN and MAST was strong ( $-0.74 \pm 0.30$ ), indicating selection for udder health could contribute to increased NWEAN. Genetic and phenotypic correlations were positive between CAL and HB, and between FRBF and RECT. Sows with lower FRBF (rg:  $-0.36 \pm 0.30$ ; rp:  $-0.10 \pm 0.03$ ) or higher HB (rp:  $0.08 \pm 0.03$ ) weaned more piglets. Iron status influences appetite and vitality of piglets at birth (cited in Hermes and Tickle (2012)). Rectal temperature, RESP and FRBF were positively correlated phenotypically, consistent with the expectations that animals with elevated body temperature will breathe faster and reduce feed intake.

## CONCLUSIONS

Traits related to health of sows (MAST, CAL, FRBF, RESP, RECT, HB) were heritable. Genetic correlations in this study were preliminary estimates, had high standard errors, and were frequently

inconsistent in magnitude or direction with phenotypic correlations. More data are required to obtain more accurate estimates of genetic correlations, particularly for trait combinations where phenotypic correlations between traits were substantial. However, negative genetic and phenotypic correlations between NWEAN and FRBF or NWEAN and MAST were implying that feed refusals and udder health have implications for current performance and for breeding programs.

**Table 2. Estimates of genetic (above diagonal), residual (1st row) and (2nd row) phenotypic (below diagonal) correlations (SE in subscript) between traits**

	NWEAN	LFAIL	CAL	FIGHT	MAST	RESP	RECT	HB	FRBF
NWEAN		-0.97 <sub>(0.18)</sub>	-0.56 <sub>(0.29)</sub>	-0.03 <sub>(0.37)</sub>	-0.74 <sub>(0.30)</sub>	0.89 <sub>(0.37)</sub>	-0.16 <sub>(0.40)</sub>	-0.69 <sub>(0.69)</sub>	-0.36 <sub>(0.30)</sub>
LFAIL	-0.73 <sub>(0.03)</sub>		0.65 <sub>(0.41)</sub>	-0.04 <sub>(0.47)</sub>	0.53 <sub>(0.42)</sub>	-0.48 <sub>(0.52)</sub>	0.20 <sub>(0.50)</sub>	0.62 <sub>(0.76)</sub>	0.47 <sub>(0.41)</sub>
CAL	-0.75 <sub>(0.01)</sub>	0.17 <sub>(0.08)</sub>	-0.18 <sub>(0.07)</sub>	-0.43 <sub>(0.26)</sub>	0.07 <sub>(0.46)</sub>	-0.42 <sub>(0.26)</sub>	-0.04 <sub>(0.29)</sub>	0.33 <sub>(0.41)</sub>	-0.13 <sub>(0.23)</sub>
FIGHT	0.001 <sub>(0.03)</sub>	-0.02 <sub>(0.03)</sub>	-0.004 <sub>(0.07)</sub>		-0.62 <sub>(0.34)</sub>	-0.21 <sub>(0.33)</sub>	-0.21 <sub>(0.41)</sub>	-0.33 <sub>(0.54)</sub>	-0.47 <sub>(0.34)</sub>
MAST	0.02 <sub>(0.07)</sub>	-0.01 <sub>(0.06)</sub>	-0.09 <sub>(0.03)</sub>	0.09 <sub>(0.07)</sub>		-0.03 <sub>(0.35)</sub>	0.16 <sub>(0.43)</sub>	-0.25 <sub>(0.58)</sub>	0.21 <sub>(0.32)</sub>
RESP	0.02 <sub>(0.03)</sub>	-0.02 <sub>(0.03)</sub>	0.13 <sub>(0.06)</sub>	0.06 <sub>(0.03)</sub>	-0.07 <sub>(0.07)</sub>		-0.12 <sub>(0.43)</sub>	-0.69 <sub>(0.76)</sub>	-0.49 <sub>(0.35)</sub>
RECT	-0.16 <sub>(0.07)</sub>	0.05 <sub>(0.07)</sub>	0.20 <sub>(0.08)</sub>	-0.07 <sub>(0.07)</sub>	-0.07 <sub>(0.07)</sub>			0.98 <sub>(0.64)</sub>	0.20 <sub>(0.34)</sub>
HB	0.02 <sub>(0.03)</sub>	-0.01 <sub>(0.03)</sub>	0.04 <sub>(0.03)</sub>	-0.09 <sub>(0.03)</sub>	-0.06 <sub>(0.03)</sub>	0.30 <sub>(0.06)</sub>			
FRBF	-0.01 <sub>(0.07)</sub>	0.02 <sub>(0.06)</sub>	0.14 <sub>(0.07)</sub>	-0.02 <sub>(0.06)</sub>	-0.05 <sub>(0.07)</sub>	0.24 <sub>(0.03)</sub>	0.19 <sub>(0.07)</sub>	-0.05 <sub>(0.06)</sub>	-0.08 <sub>(0.50)</sub>
	-0.03 <sub>(0.03)</sub>	0.04 <sub>(0.03)</sub>	0.10 <sub>(0.03)</sub>	-0.05 <sub>(0.03)</sub>	-0.02 <sub>(0.03)</sub>	0.24 <sub>(0.03)</sub>	0.04 <sub>(0.03)</sub>		
	0.16 <sub>(0.07)</sub>	-0.13 <sub>(0.06)</sub>	0.13 <sub>(0.07)</sub>	-0.06 <sub>(0.06)</sub>	-0.02 <sub>(0.07)</sub>	0.01 <sub>(0.03)</sub>	0.04 <sub>(0.03)</sub>		
	0.08 <sub>(0.03)</sub>	-0.06 <sub>(0.03)</sub>	0.15 <sub>(0.03)</sub>	-0.08 <sub>(0.03)</sub>	-0.04 <sub>(0.03)</sub>	0.24 <sub>(0.07)</sub>	0.12 <sub>(0.07)</sub>	0.15 <sub>(0.06)</sub>	
	-0.04 <sub>(0.07)</sub>	0.04 <sub>(0.07)</sub>	0.06 <sub>(0.08)</sub>	0.04 <sub>(0.07)</sub>	-0.12 <sub>(0.07)</sub>	0.10 <sub>(0.03)</sub>	0.13 <sub>(0.03)</sub>	0.12 <sub>(0.06)</sub>	
	-0.10 <sub>(0.03)</sub>	0.10 <sub>(0.03)</sub>	0.01 <sub>(0.03)</sub>	-0.04 <sub>(0.03)</sub>	-0.06 <sub>(0.03)</sub>				

For trait name abbreviations see Table 1.

#### ACKNOWLEDGEMENT

This research was funded by the Australian Pork CRC under the project 2A-116. The first author is supported by UNE via International Postgraduate Research Award (UNE IPRA).

#### REFERENCES

- Anil S.S., Anil L. and Deen J. (2008) *Can J Anim Sci* **88**: 381.  
 Bunter K.L. (2017) *Proc. Assoc. Advmt. Anim. Breed. Genet.* **22**: 229.  
 Bunter K.L., Vargovic L., Athorn R.Z., Henman D. and Luxford B.G. (2018) In *Proc. World Congr. Genet. App. Livest. Breed* **11**: 992.  
 Gilmour A., Gogel B., Cullis B., Welham S. and Thompson R. (2014) ASReml User Guide Release 4.1 VSN International Ltd, Hemel Hempstead, HP1 1ES, UK.  
 Hermes S. and Tickle K.M. (2012) In *Pig Genetic Workshop Notes*. Armidale, Australia. pp. 31:38.  
 Knauer M.T. and Baitinger D.J. (2015) *Appl. Eng. Agric.* **31**: 175.  
 R Core Team (2018) *R Foundation for Statistical Computing*, Vienna, Austria.  
 Rydhmer L. (2000) *Livest. Prod. Sci.* **66**: 1:12.  
 Tabuaciri P., Bunter K.L. and Graser H.U. (2010) *Proc. World Congr. Genet. App. Livest. Breed* **9**: 684.