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GENETIC PARAMETER ESTIMATES FOR PRE- AND POST-WEANING PIGLET MORTALITY

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SUMMARY

Alternative models for genetic evaluation of pre- and post-weaning mortality traits were investigated. For pre-weaning mortality, the best model accounted for direct piglet effects, common litter effects of both the nurse sow and biological dam, repeated records of the nurse sow and the maternal nurse sow genetic effects. For post-weaning mortality, the most parsimonious model included only direct piglet effects and the common litter effects of both the nurse sow and biological dam. After accounting for systematic effects, genes of the piglet contribute to both pre- and post-weaning mortality (direct $h^2 = 0.02 \pm 0.002$ for pre- and post- weaning), whereas the nurse maternal genes only contribute to pre-weaning (maternal $m^2 = 0.01 \pm 0.002$). While heritabilities were low, there is potential for genetic improvement of both pre- and post-weaning mortality traits.

INTRODUCTION

Selection for efficient, lean growth and increased litter size can increase piglet pre-weaning mortality (Bunter 2009), with recent pre-weaning mortality rates reported as high as 18% in Australian herds (Australian Pig Industry Benchmarking Report, 2018). Therefore, breeding values for survival have become an important component of breeding programs. It is possible to make improvements by genetically enhancing a piglet's ability to survive (Mesa *et al.* 2006), while also improving litter size, although an antagonistic relationship occurs between the two traits (Bunter 2009). Piglet survival involves different phenotypes and genes, including that of the piglet's biological dam, the sow nursing the piglet, and the genotype of the piglet itself (Knol *et al.* 2002). In addition, piglets born and/or nursed within a common litter have common environmental effects contributing to their mortality (Bunter 2009). In the review of Bunter (2009), heritability estimates were on average 0.05 at the piglet level, and 0.11 at the sow level, indicating that both direct and maternal components should be considered. The purpose of this study was to investigate alternative models for genetic evaluation of piglet pre- and post-weaning mortality, treated as a trait of the piglet.

MATERIALS AND METHODS

Data. Data on individual piglet mortality (alive = 0, dead = 1) before weaning or post-weaning and other related traits, were recorded at a commercial piggery located in southern New South Wales, Australia. Data included 466,012 individually identified pedigreed piglets born between 2009 and 2018, from two Maternal (Large White and Landrace) and one Terminal (Duroc) selection line. This data set represented progeny of 1,535 sires, 19,867 dams and 28,228 nurse sows, which were included in the pedigree, extending over 10 generations, born in 43,462 litters. Piglets were individually identified within 24 hours from birth, with individual birth weights and sex recorded. Cross fostering occurred after identification, and all movements and deaths of individual piglets were recorded, along with corresponding dates. A piglet was recorded as a pre-weaning death if it was born alive and died before weaning (average of 26 days). A post weaning death was recorded if the piglet had been weaned and was less than 70 days of age at death. Piglets with a pre-weaning record equal to 1 do not have a post-weaning mortality record.

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Statistical Analysis. Preliminary analyses confirmed that the following fixed effects significantly ($P<0.0001$) contributed to mortality outcomes: piglet breed; gender (2 levels: male and female); piglet fostering status (un-fostered = 0, fostered by day 2 = 1, fostered after day 2 = 2), which was concatenated with sow (birth-nurse) parities and farrowing farm (totalling 48 levels); and birthweight class (6 levels: 0.60-1.21, 1.22-1.39, 1.40-1.54, 1.55-1.69, 1.70-1.90, 1.91-3.00 kilograms). Additionally, gestation length (6 levels: 105-114, 115, 116, 117, 118-125 days); total born group (5 levels: 1-5, 6-10, 11-15, 16-20, 20-25) and birth year quarter (40 levels: accounting for the managerial and seasonal differences) were accounted for in models for analysis. Additional factors for post-weaning mortality included age group when individual piglets were weaned (5 levels: 0-14, 15-21, 22-28, 29-35 and 36-60 days), and the farm that piglets were weaned into (7 levels).

To estimate genetic parameters for pre- and post-weaning mortality, an univariate analysis was performed using linear models in ASReml (Gilmour *et al.* 2015), where either nurse sow, biological dam models or elements of both were investigated. Random effects tested included terms for the animal (piglet), the common litter effect of either nurse litter, birth litter or a combination of both, the permanent environment of the nurse sow or the biological dam and the maternal genetic effect of either nurse sow or biological dam. The mixed model is represented by:

$$\mathbf{y= X\beta + Z_1a + Z_2c + Z_3mpe + Z_4m + e} \quad (1)$$

where **y** is the vector of the observations; **X** is the incidence matrix for the vector of fixed effects in **β;** **Z** are incidence matrices associated with vectors of random effects including additive genetic effect (piglet) in **a**, common nurse and/or biological litter effect in **c**, nurse sow or biological dam permanent environment effect in **mpe**, nurse sow or biological dam maternal genetic effect in **m** and **e** is a vector residuals. Effects were distributed as $Var(\mathbf{a}) = \mathbf{A}\sigma_a^2$, where **A** is a matrix describing the relationships between animals, and the remaining effects: $Var(\mathbf{c})=\mathbf{I}\sigma_c^2$, $Var(\mathbf{mpe})=\mathbf{I}\sigma_{mpe}^2$ and $Var(\mathbf{m})=\mathbf{I}\sigma_m^2$, where **I** is an identity matrix. The Akaike Information Criteria (AIC test) was used to test if the inclusion of additional random effects was significant, with the preferred model having the lowest AIC value (Mendenhall *et al.* 1996).

RESULTS AND DISCUSSION

To avoid double counting, models for piglet mortality traits accounted for sow traits already used as selection criteria, such as birth weight and litter size, and also accounted for fostering status, which is typically ignored in other studies. Accounting for these systematic effects might have reduced estimates of heritabilities in this relative to other studies (Table 1).

Pre-weaning mortality. On average, 87.4% of piglets were nursed by their biological dam, based on fostering events in the first two days of birth, but 19.3% of litters contained piglets with different parentage. Based on the AIC values, the nurse sow model (N3b) fit the data significantly better than the corresponding biological sow model (D3). Model N3b included the common litter effects of both the nurse sow and the biological dam, accommodating the effects of both the gestation and lactation environment experienced by the piglets. The model was improved substantially by the addition of maternal effects (mpe or m), accommodating repeated records and maternal genetic effects of the nurse sow. Maternal genetic effects reflect heritable traits like uterine nutrient supply and capacity, milk quality and quantity and general maternal care (Kaufmann *et al.* 2000). Removal of the model terms related to gestation length, birth weight and litter size (model N3c), which are typically considered as sow traits, created an increase in the phenotypic variance for pre-weaning mortality along with increases in maternally mediated variance ratios (cnl^2 and m^2), demonstrating that these maternal factors influence pre-weaning mortality. Direct heritability of piglets was improved with a nurse sow model (N1 vs D1), as the survival of piglets from different litters were compared within common nurse litters. Direct ($h^2 = 0.002 \pm 0.002$) and maternal ($m^2 = 0.01 \pm 0.002$) heritabilities for piglet mortality

estimated from model N3b were low in comparison to average literature estimates (Bunter 2009). Knol *et al.* (2002) reported that direct heritabilities were 0.03 for dam lines and 0.22 for maternal heritabilities, where the genetic effects of the biological dam, maternal permanent environmental and common birth litter effects were accounted for. Roehe *et al.* (2010) reported a direct heritability of 0.24 and maternal heritability 0.14 using a biological dam model, without accounting for nurse sow effects, making direct comparisons with the literature difficult.

Post-weaning mortality. The best model for post-weaning mortality was D6b based on AIC values, which was a biological sow model including common nurse litter effects. However, in contrast to pre-weaning mortality, variances for permanent environmental and maternal genetic effects were much lower and contributed no substantial improvement to the model fit (for example model N4b vs N6b; D4 vs D6a). Furthermore, the ratios for maternal permanent environment and direct maternal effects were very small, indicating sows only contribute to post-weaning outcomes through additive genetic effects, and any carry over from prior gestation and lactation periods was accounted for in the common litter effects. This was also inferred using model N4c, where removing gestation length, total born and piglet birth weight terms from the model resulted in no changes to variance estimates. Since sampling correlations between direct and biological dam effects are high (not shown) and taking these results into account, a more parsimonious model should be used, leading to model N4b being the best model for the data. This model is a nurse sow model accounting for common nurse and biological litter effects, which resulted in direct heritability estimates to be 0.02 ± 0.002, with no literature available for direct comparison of post-weaning mortality estimates in pigs.

CONCLUSIONS

This large data set enabled separation of direct from maternal effects, along with common litter effects and permanent environmental effects of the sow and fostering status. Failing to separate all of these effects could lead to overestimates of direct or maternal heritability but is complicated due to high sampling correlations for mortality traits. Nevertheless, the genetic parameters presented in this study suggest that there is potential for genetic improvement of pre- and post-weaning mortality traits in commercial breeding programs, independent of other important traits such as birth weight, gestation length and litter size.

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Table 1. Variance component estimates (×100) and resulting ratios of variance components under different models for pre- and post-weaning mortality

Level	Model	σ^2_a	σ^2_{cnl}	σ^2_{cbl}	σ^2_{mpe}	σ^2_m	σ^2_p	$h^2 \pm SE$	$cnl^2 \pm SE$	$cbl^2 \pm SE$	$mpe^2 \pm SE$	$m^2 \pm SE$	AIC
Pre-wean	N1	0.93	1.31	-	-	-	14.1	0.07±0.003	0.09±0.001	-	-	-	-471314.3
Nurse	N2	0.58	1.14	-	0.26	-	14.0	0.04±0.003	0.08±0.002	-	0.02±0.001		-471547.46
	N3a	0.50	1.14	-	0.13	0.17	14.0	0.04±0.003	0.08±0.002	-	0.01±0.002	0.01±0.001	-471669.45
	N3b	0.29	0.70	0.53	0.13	0.20	14.0	0.02±0.002	0.05±0.002	0.04±0.002	0.01±0.002	0.01±0.002	-472115.44
	N3c	0.27	0.82	0.40	0.11	0.23	14.8	0.02±0.002	0.06±0.002	0.03±0.002	0.007±0.002	0.02±0.002	
Pre-wean	D1	0.67	-	1.25	-	-	14.0	0.05±0.003	-	0.09±0.001	-	-	-469850.36
Dam	D2	0.32	-	1.09	0.27	-	14.0	0.02±0.002	-	0.08±0.001	0.02±0.001	-	-470075.54
	D3	0.26	-	1.09	0.14	0.17	14.0	0.02±0.002	-	0.08±0.001	0.01±0.002	0.01±0.001	-470193.32
Post-wean	N4a	0.14	0.24	-	-	-	5.09	0.03±0.002	0.05±0.001	-	-	-	-756059.93
Nurse	N4b	0.11	0.10	0.16	-	-	5.09	0.02±0.002	0.02±0.002	0.03±0.002	-	-	-756306.33
	N4c	0.11	0.12	0.16	-	-	5.11	0.02±0.002	0.02±0.002	0.03±0.002	-	-	
	N5	0.13	0.24	-	0.001	-	5.09	0.03±0.002	0.05±0.001	-	0.002±0.001	-	-756061.24
	N6a	0.13	0.24	-	0.0004	0.0007	5.09	0.02±0.002	0.05±0.001	-	0.000±0.001	0.001±0.001	-756064.37
	N6b	0.10	0.09	0.17	0.0006	0.0009	5.09	0.02±0.002	0.02±0.002	0.03±0.002	0.002±0.001	0.002±0.001	-756317.81
Post-wean	D4	0.11	-	0.25	-	-	5.09	0.02±0.002	-	0.05±0.001	-	-	-756199.49
Dam	D5	0.09	-	0.24	0.02	-	5.09	0.02±0.002	-	0.05±0.001	0.004±0.001	-	-756213.85
	D6a	0.09	-	0.24	0.01	0.01	5.09	0.02±0.002	-	0.05±0.001	0.002±0.001	0.003±0.001	-756225.55
	D6b	0.09	0.11	0.15	0.01	0.01	5.09	0.02±0.002	0.02±0.002	0.03±0.002	0.002±0.001	0.003±0.001	-756332.69

Abbreviations: N = nurse sow model; D = biological dam model; σ^2_a = additive genetic variance; σ^2_{cnl} = common nurse litter variance; σ^2_{cbl} = common biological litter variance; σ^2_{mpe} = maternal permanent environmental variance either attributed to nurse or biological dam; σ^2_m = maternal genetic variance either attributed to nurse or biological dam; σ^2_p = phenotypic variance; h^2 = heritability estimate; cnl^2 = proportion of phenotypic variance attributed to nurse litter effect; cbl^2 = proportion of phenotypic variance attributed to biological litter effect; mpe^2 = proportion of phenotypic variance attributed to permanent environmental effect (either nurse or biological dam); m^2 = proportion of phenotypic variance attributed to maternal genetic variance (either nurse or biological dam); AIC = Akaike Information Criterion