

GENETIC CORRELATIONS BETWEEN PUREBRED AND CROSSBRED PRE- AND POST-WEANING PIGLET MORTALITY

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SUMMARY

Purebred performance can be a poor predictor of future crossbred performance. This study investigated the genetic correlations between purebred and crossbred individual piglet pre- and post-weaning mortality traits. The estimates of the additive genetic correlations between purebred and crossbred performances (r_{pc}) were high (pre-weaning: $r_{pc} = 0.78 \pm 0.097$ and post-weaning: $r_{pc} = 0.94 \pm 0.112$). High correlations ($r_{pc} > 0.75$) demonstrate that survival traits of crossbred pigs will be improved using genetic evaluation and selection based on purebred records.

INTRODUCTION

Mortality traits have low heritability but still have the potential to be genetically improved in purebred populations. The development of successful breeding programs relies on whether purebred performance, recorded in the nucleus tier, accurately predicts outcomes in crossbred performance, the commercial tier (Abell *et al.* 2016). A limiting factor of the typical pyramidal structure of pig breeding systems is that purebred performance can be a poor predictor of future crossbred performance for a range of traits (Ibáñez-Escriche *et al.* 2014).

The genetic correlation between the same trait recorded in purebred vs crossbred populations (r_{pc}) is a key parameter that determines the benefit of recording additional information in crossbred populations (Kramer *et al.* 2021). A high r_{pc} , for example greater than 0.75, indicates that the additive genetic component of the trait was essentially the same in the two populations and the value for selection of recording additional crossbred data may be limited (Kramer *et al.* 2021). Generally, recording systems and performance test records for crossbred animals are insufficient. This limits genetic evaluation and published genetic parameters, particularly for mortality traits. The hypothesis was that the genetic correlation between purebred and crossbred individual piglet mortality traits is high when recorded in the same production environments.

MATERIALS AND METHODS

Data. Purebred and crossbred mortality records were available for this study, from single sire matings producing purebred and crossbred progeny in a common environment, with some overlap of common parentage. The data included individual piglet mortality (0=alive, 1=dead) for pre- (PREw) and post-weaning (POSTw). All piglets were pedigreed from purebred matings within two maternal (genotype A and B) or one terminal (genotype C) selection line, or crossbred, from first-cross progeny (F1 progeny: genotype A \times B and B \times A) and commercial-cross progeny (F1 \times C: F1 sows \times C boars). Mortality data was collected as part of a commercial breeding operation located in southern New South Wales, Australia, between January 2011 and December 2019, with a shortened period for crossbred piglets being recorded from August 2015 to December 2019. A total of 477,780 progeny represented 1398 sires, 19,807 biological dams and 19,706 nurse sows, extending over 10

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generations, born in 43,094 litters. Of these 590 sires, 2321 dams and 4627 nurse sows were common to purebred and corresponding crossbred progeny.

The trait phenotypes and animal management have been outlined in Harper *et al.* (2019). Sows and piglets of all genotypes and crosses recorded during the same time-period were managed identically within the same production environment pre- and post-weaning. Purebred piglets were generally fostered within line to nurse sows who had purebred litters. Crossbred piglets were fostered to either purebred sows with first-cross progeny or crossbred sows who had commercial-cross progeny. Piglets in post-weaning, were reared in the same facility with similar health challenges, however purebred piglets were generally penned separately to crossbred progeny.

Each mortality trait was measured on purebred (PurePREw; PurePOSTw) and crossbred (CrossPREw; CrossPOSTw) progeny, defined as different traits for analysis. In this study, purebred animals had their own purebred traits but were given missing values for the corresponding crossbred traits, and vice versa.

Statistical analysis. The nurse sow models identified in Harper *et al.* (2019) formed the basis for the bivariate analyses performed in this study, using linear models in ASReml (Gilmour *et al.* 2021). For PREw, the best model for random effects accounted for direct effects of the animal (piglet), common litter effects of both the nurse sow and biological dam, along with the permanent environment and maternal genetic effect of the nurse sow. Biological dam and nurse sow effects (genetic and permanent environment) were fitted across purebred and crossbred traits to accommodate common sows producing or nursing both purebred and crossbred piglets. For POSTw, the most parsimonious model included only direct animal (piglet) effects along with common litter effects of both the nurse sow and biological dam. The fixed effects fitted for both traits included gender, gestation length group, total born group, birthweight group and birth year quarter. The concatenation of biological dam and nurse sow parity grouping, fostering status of the piglet and farrowing farm described the rearing environment. Additionally, wean age group and weaning farm were fitted for POSTw. Levels for piglet genotype were extended to accommodate crossbred data (purebred traits, contained three levels: A, B and C; and crossbred traits contained three levels: $A \times B$, $B \times A$ and $F1 \times C$). Genotype was nested within the purebred or crossbred traits as the latter were only present in the second half of the project time-period. Genetic correlations were then estimated, with both common parents and ancestors providing genetic links between purebred and crossbred individual piglets.

RESULTS AND DISCUSSION

The design of this study provided good cross classification of purebred and crossbred piglet performance for sires and dams. Trait means for PurePREw and CrossPREw were 17.8% (A: 20.6%; B: 15.4%; C: 17.3%) and 16.8% ($A \times B$: 18.2%; $B \times A$: 15.3%; $F1 \times C$: 17.0%), respectively. Suggesting that in this population, raised in a common environment, commercial crossbred piglets with 100% direct and maternal heterosis were no better at surviving than purebred piglets. For PurePOSTw and CrossPOSTw, a proportionally larger difference was seen, 5.59% (A: 5.46%; B: 6.11%; C: 4.67%) and 3.01% ($A \times B$: 3.88%; $B \times A$: 3.15%; $F1 \times C$: 2.43%), respectively. The increased crossbred piglet survival may have been the result of increased direct heterosis relative to purebreds, which is beneficial when piglets are weaned and reared separately from their dams. Phenotypic variances reflected (dis)similarity in incidence, being similar for PurePREw (13.6%) and CrossPREw (13.2%), but approximately halved between PurePOSTw (5.17%) and CrossPOSTw (2.86%). It is important to note that there was no difference in the environment and health status experienced between genotypes as all piglets were reared in the same facility under the same management.

Table 1. Variance component estimates ($\times 100$), resulting ratios of variance components (\pm standard error) and correlations (\pm standard error) between random effects for each bivariate model; purebred pre-weaning mortality (PurePREw) and crossbred pre-weaning mortality (CrossPREw); purebred post-weaning mortality (PurePOSTw) and crossbred post-weaning mortality (CrossPOSTw).

Pre-weaning mortality Models						
Variance component estimates ($\times 100$)						
	σ^2_a	σ^2_{cnl}	σ^2_{cbl}	σ^2_{mpe}	σ^2_m	σ^2_p
PurePREw	0.24	0.46	0.64	0.12	0.16	13.6
CrossPREw	0.20	0.41	0.34	0.12	0.16	13.2
Ratios of variance components (\pm standard error)						
	$h^2 \pm SE$	$cnl^2 \pm SE$	$cbl^2 \pm SE$	$mpe^2 \pm SE$	$m^2 \pm SE$	
PurePREw	0.02 ± 0.002	0.03 ± 0.003	0.05 ± 0.003	0.01 ± 0.001	0.01 ± 0.001	-
CrossPREw	0.02 ± 0.003	0.03 ± 0.005	0.03 ± 0.005	0.01 ± 0.001	0.01 ± 0.001	-
Correlations (\pm standard error) between traits within random effects						
	$r_a \pm SE$	$r_{cnl} \pm SE$	$r_{cbl} \pm SE$	$r_{mpe} \pm SE$	$r_m \pm SE$	
Correlation	0.78 ± 0.097	0.98 ± 0.210	n.e.	n.e.	n.e.	-
Post-weaning mortality Models						
Variance component estimates ($\times 100$)						
	σ^2_a	σ^2_{cnl}	σ^2_{cbl}	σ^2_{mpe}	σ^2_m	σ^2_p
PurePOSTw	0.10	0.11	0.16	-	-	5.17
CrossPOSTw	0.03	0.08	0.05	-	-	2.86
Ratios of variance components (\pm standard error)						
	$h^2 \pm SE$	$cnl^2 \pm SE$	$cbl^2 \pm SE$	$mpe^2 \pm SE$	$m^2 \pm SE$	
PurePOSTw	0.02 ± 0.002	0.02 ± 0.003	0.03 ± 0.003	-	-	-
CrossPOSTw	0.01 ± 0.002	0.03 ± 0.005	0.02 ± 0.005	-	-	-
Correlations (\pm standard error) between traits within random effects						
	$r_a \pm SE$	$r_{cnl} \pm SE$	$r_{cbl} \pm SE$	$r_{mpe} \pm SE$	$r_m \pm SE$	
Correlation	0.94 ± 0.112	0.77 ± 0.264	n.e.	n.a.	n.a.	-

Abbreviations: 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 attributed to nurse sow; σ^2_m = maternal genetic variance attributed to nurse sow; σ^2_p = phenotypic variance; h^2 = heritability estimate; cnl^2 = proportion of phenotypic variance attributed to common nurse litter effect; cbl^2 = proportion of phenotypic variance attributed to common biological litter effect; mpe^2 = proportion of phenotypic variance attributed to permanent environmental effect of nurse sow; m^2 = proportion of phenotypic variance attributed to maternal genetic variance of nurse sow; r_a = genetic correlation; r_{cnl} = common nurse litter correlation; r_{cbl} = common birth litter correlation; r_{mpe} = permanent environmental effect of the biological dam correlation; r_m = biological dam genetic correlation; n.a. = not applicable = effect not fitted; n.e. = not estimable; SE = standard error.

The estimates of the additive genetic correlations between purebred and crossbred performances (r_{pc}) within trait were high (PREw: $r_{pc} = 0.78 \pm 0.097$ and POSTw: $r_{pc} = 0.94 \pm 0.112$; Table 1). High correlations ($r_{pc} > 0.75$) indicate that these two traits were largely controlled by the same genes and that obtaining crossbred data may be of limited use to improve accuracy of breeding values for mortality traits, other than through enabling more data. Estimates of the correlation between common nurse litter effects (r_{cnl}) within both PREw and POSTw were also very high ($r_{cnl} = 0.98 \pm$

0.21 and $r_{\text{cni}} = 0.77 \pm 0.26$ respectively) and were not significantly different from one based on the log-likelihood ratio test, indicating that the environment provided by the nurse sow was the same regardless of the piglet genotype, which was expected.

The variance due to nurse sow effects (maternal: $m^2 = 0.01 \pm 0.001$ and permanent environment: $mpe^2 = 0.01 \pm 0.001$) for PREw were similar in magnitude to previous estimates from purebred data only (Harper *et al.* 2019). Correlations were not estimable for the maternal genetic or permanent environmental components of the nurse sow, as convergence failed when these were fitted within trait due to poor data structure. Correlations between common birth litter effects were also not estimable, as birth litters could only be either purebred or crossbred.

Very few studies evaluate purebred and crossbred performance for any trait in the same production environment. For mortality traits, literature tends to only be available for purebred populations; or only on three-way crossbred piglets (F1 sows crossed with terminal sire lines) in commercial production environments (Cecchinato *et al.* 2008, Dufrasne *et al.* 2013, Strange *et al.* 2013); or the combination of purebred and crossbred piglets that are with different environments and management practices, such as a high health nucleus or commercial production farm (Cecchinato *et al.* 2010, Abell *et al.* 2012). All of these scenarios may act to reduce the resemblance in performance between purebred and crossbred progeny, reducing the correlations between these representations of the same trait, making direct comparisons with literature difficult. Overall, r_{pc} here tended to be higher than present in other studies.

CONCLUSIONS

For genetic improvements in the commercial tier of the breeding pyramid to be made, it is important that genetic improvement in the nucleus is reflected by corresponding changes in crossbred performance. The current study demonstrates that survival traits of crossbred pigs will be improved using genetic evaluation and selection based on purebred records due to the high genetic correlations between these traits. Therefore, selection to reduce mortality in purebred populations are expected to deliver downstream improvements for crossbred populations.

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