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Genetic correlations between sow uterine prolapse and reproductive traits using first- and multiple-parity performance data

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Introduction

As global demand for pork increases, sow lifetime productivity is an essential factor in herd-level production and profitability. Elevated sow mortality rates lead to the loss of breeding sows and, thus, to a loss of genetic progress. Increased management and replacement costs impose heavy psychological strain on producers. Hence, sow mortality is a trait of concern for both welfare and economic reasons. In the U.S. swine industry, one of the major causes of sow mortality includes vaginal/uterine prolapse (PLS), defined as a loss of pelvic support resulting in the protrusion of the vagina or uterus (Stevens *et al*., 2021*;* Bhatia *et al*., 2023). Some suspected causes of uterine prolapse include water quality, vitamin deficiencies, mycotoxins, and genetics. Although PLS has shown relatively low heritability (~0.15 on the linear scale and ~0.22 on the threshold scale), this suggests that genetic selection could reduce its incidence (Stevens *et al.,* 2021). Many breeding programs worldwide emphasize first-parity reproductive performance to enable earlier culling decisions and shorten the generation interval (Noppibool *et al*., 2017). However, selection decisions based solely on first-parity data may have unintended consequences for later-life outcomes, potentially increasing PLS risk. We therefore hypothesize that selection using only first-parity records could inadvertently increase PLS incidence, whereas incorporating information across parities may reduce this risk by enabling a more accurate evaluation of lifetime performance. The objective of this study was to quantify the genetic relationship between PLS and sow reproductive performance in a purebred maternal line by estimating genetic correlations between PLS with sow reproductive traits and by comparing models using first-parity information with models based on lifetime performance.

Materials & Methods

Data. Data were collected from 38,175 purebred Topigs Norsvin sows from a single line across multiple U.S. farms and parities between 2014 and 2024. Records included PLS, defined at the sow level as the presence/absence of uterine and/or vaginal prolapse, with an average incidence of 4.4% across all parities (1 through 8). Reproductive performance traits included total number born (TNB), number of stillborn piglets (SB), body condition score at weaning (BCS: 1= skinny, 3= ideal, 5= fat), and sow longevity. Longevity was defined as retention to parity 2 (LGY2: 0-1) or to parity 5 (LGY5: 1-5). For reproductive traits, alternative trait definitions were created using either first-parity records only (TNB_1, SB_1, BCS_1) or lifetime performance records (TNB_{All}, SB_{All}, BCS_{All}). Summary statistics for all traits are presented in Table 1. The pedigree included 41,025 individuals traced back three generations.

Table 1. Descriptive statistics of reproduction traits and prolapse

Trait[1]	Total Number of records	Mean	Standard deviation
TNB_1	30,537	13.4	3
TNB_{All}	104,393	13.92	3.35
SB_1	30,581	0.22	0.43
SB_{All}	104,553	0.24	0.45
BCS_1	21,010	2.47	0.5
BCS_{All}	76,993	2.56	0.51
LGY2	23,468	0.88	0.32
LGY5	17,566	3.74	1.41
PLS	36,336	4.4	23.4

[1]TNB_1: Total number born at first parity; TNB_{All}: Total number born in all parities; SB_1: Number of stillborn piglets at first parity; SB_{All}: Number of stillborn piglets in all parities; BCS_1: Body condition score at weaning at first parity; BCS_{All}: Body condition score at weaning in all parities (Score: 1-5); LGY2: Sow longevity up to parity 2; LGY5: sow longevity up to parity 5; PLS: vaginal/uterine prolapse (%)

Genetic parameter estimation. Prolapse heritability on the observed scale as well as genetic correlations (r_g) between PLS and each reproductive trait were estimated from pedigree-based analyses in ASReml-R (Butler *et al*., 2017). For computational simplicity and to ensure a consistent modelling framework across traits, bivariate linear animal models were fitted, pairing PLS with each reproductive trait in turn, with PLS treated as a continuous trait. Analyses were conducted using bivariate linear animal models, pairing PLS with each reproductive trait. Fixed effects included herd × year × season interaction and parity. Random effects were additive genetic effects for both traits, and trait-specific permanent environmental and contemporary group effects for the reproductive trait only, in addition to residual effects. They were assumed to follow a multivariate normal distribution with a mean zero and the following (co)variance structure:

$$\mathrm{Var}(\mathbf{r}) = \begin{bmatrix} \mathbf{A} \otimes \boldsymbol{\Sigma}_a & \mathbf{0} & \mathbf{0} & \mathbf{0} \\ \mathbf{0} & \mathbf{I}\sigma^2_{pe,tr} & \mathbf{0} & \mathbf{0} \\ \mathbf{0} & \mathbf{0} & \mathbf{I}\sigma^2_{cg,tr} & \mathbf{0} \\ \mathbf{0} & \mathbf{0} & \mathbf{0} & \mathbf{I} \otimes \boldsymbol{\Sigma}_e \end{bmatrix} \quad (1)$$

where $\mathbf{A}$ is the numerator relationship matrix, $\boldsymbol{\Sigma}_a$is the additive genetic variance–covariance matrix for PLS and the reproductive trait, $\sigma^2_{pe,tr}$ and $\sigma^2_{cg,tr}$ are the permanent environmental and contemporary group variances for the reproductive trait, respectively, and $\boldsymbol{\Sigma}_e$ is the residual variance–covariance matrix.

Results & Discussion

Heritability of PLS was estimated at 0.13 (± 0.02), which is consistent with the estimate reported by Stevens *et al*. (2021) (0.15 ± 0.02), obtained using a linear animal model on data from the same genetic line. While both studies used linear models and similar phenotype definitions (presence/absence of prolapse at the sow level), the present study differs in data structure and size, whereas Stevens *et al*. (2021) analyzed data from a more limited set of populations and focused on validation of EBVs. These differences in data composition may contribute to minor differences in estimated heritability. In contrast, Bhatia *et al.* (2023), also using data from this genetic line, reported a higher heritability estimate (0.35 ± 0.02) when

incorporating genomic information and fitting a logit (threshold) model on the liability scale. Taken together, these results indicate that while heritability estimates for PLS vary depending on incidence rate, model, and data type, they consistently demonstrate the presence of a genetic component. This suggests that genetic selection can be effective in reducing PLS incidence, even though environmental factors play a substantial role in its expression. Estimates of genetic correlations between PLS and reproductive traits (under both first-parity and lifetime performance definitions) are presented in Table 2. Overall, these results indicate that the genetic relationship between PLS and reproductive performance is more appropriately captured when traits are defined across the lifetime rather than based solely on first-parity information. The genetic correlation between TNB_1 and PLS was essentially zero (0.06 ± 0.04), suggesting no genetic association when selection is based solely on first-parity performance. In contrast, TNB_{All} showed a favourable, negative correlation with PLS ($r_g = -0.16 \pm 0.03$), indicating that sows with higher lifetime TNB tend to have lower genetic susceptibility to PLS. A similar pattern was observed for longevity, where increased retention to parity 5 was associated with reduced PLS ($r_g = -0.22 \pm 0.06$). For SB and BCS, correlations with PLS were small and remained stable across trait definitions ($SB_1 = 0.19$ vs. $SB_{All} = 0.18$; $BCS_1 = -0.08$ vs. $BCS_{All} = -0.10$), suggesting that the underlying genetic relationships of these traits with PLS are relatively insensitive to whether first-parity or lifetime information is used. To our knowledge, no previous studies have contrasted genetic relationships between PLS and reproductive performance using first-parity versus lifetime trait definitions. The differences observed here—particularly for TNB—suggest that first-parity-only selection may underestimate the potential for indirect improvement in PLS incidence achievable when lifetime performance information is incorporated. This is notable given evidence of relatively high genetic correlations between early and later parities for reproductive traits, which is interpreted as shared genetic control across parities (Roehe and Kennedy, 1995; Fernández *et al*., 2008). In the present study, however, incorporating all-parity information suggests a low but favourable association between TNB and PLS, highlighting the value of lifetime performance assessment in selection strategies aimed at reducing prolapse incidence.

Table 2. Genetic correlations (r_g) and associated standard errors (SE) between reproductive traits, defined using lifetime performance data (All) vs. first parity data only (1), with vaginal/uterine prolapse (PLS).

Trait[1]	r_g	SE	t-value[2]
TNB_1	0.06	0.04	4.40***
TNB_{All}	-0.16	0.03	
SB_1	0.19	0.05	0.16 (n.s)
SB_{All}	0.18	0.04	
BCS_1	-0.08	0.04	0.40 (n.s)
BCS_{All}	-0.10	0.03	
LGY2	-0.09	0.07	1.41 (n.s)
LGY5	-0.22	0.06	

[1]TNB_1: Total number born at first parity; TNB_{All}: Total number born in all parities; SB_1: Number of stillborn piglets at first parity; SB_{All}: Number of stillborn piglets in all parities; BCS_1: Body condition score at weaning at first parity; BCS_{All}: Body condition score at weaning in all parities; LGY2: Sow longevity up to parity 2; LGY5: sow longevity up to parity 5

[2]Significance level of t-value between the correlations ($_1$ vs. $_{All}$): *** highly significant, n.s not significant

Conclusions

Selection for higher TNB based on lifetime performance information, rather than first-parity data alone, could reduce genetic susceptibility to PLS as a correlated response. In addition, these results suggest that restricting selection to first-parity reproductive performance may mask favourable genetic associations with lower PLS risk and, consequently, limit the potential for indirect reductions in PLS incidence over time. In conclusion, our current selection strategy, which includes selection for enhanced reproductive performance using lifetime performance information, as well as selection for enhanced sow longevity, is an effective breeding strategy to improve lifetime productivity while reducing mortality due to vaginal/uterine prolapse and other causes of sow mortality.

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