Genetic Determination Of Components Of Piglet Survival In A Two-Generation Crossbreeding Selection Experiment

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Introduction

Piglet mortality is of substantial animal welfare concern and of great economic importance, in particular under the circumstances where continuous genetic improvement of litter size is expected to result in higher mortality (Baxter et al. 2009; Su et al., 2008). As a consequence, genetic improvement of piglet survival will be one of the main challenges to be faced by pig breeding. The main cause of postnatal mortality is crushing of piglets by the mother. Crushing is expected to be mainly a maternal genetic effect but it may also be affected by the genetic potential of the piglet for e.g. vitality, alertness. Furthermore, crushing may be influenced differently under indoor than under outdoor conditions. Roehe et al. (2009, 2010) found for other piglet mortality traits higher direct and maternal heritabilities under outdoor conditions than under indoor conditions. Genetic correlations between survival by avoiding crushing and other survival traits or birth weight will give further insight into the genetic improvement of this trait, with the overall aim to improve sow productivity. Therefore, the objectives of this study were to estimate the direct and maternal genetic determination of survival by avoiding crushing, its genetic correlations with other survival traits and birth weight as well as its genetic response, from data of a two-generation selection experiment under outdoor conditions.

Material and methods

Selection experiment. Data on 21835 individual piglets from a two-generation selection experiment for improvement of postnatal piglet survival under outdoor conditions were available to estimate the genetic determination of crushing. In the first generation, Landrace sires (from a dam line) were selected for high or average (avg) maternal estimated breeding values (EBVs) of postnatal survival of piglets kept indoors and randomly mated with commercial crossbred sows. Female progeny from these parents were raised and mated to Large White boars (from a sire line) selected for high and average direct EBV of postnatal survival of piglets kept indoors to produce the piglets of the second generation. The parent

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sows were kept over three parities. Litters from 1st parity sows (n = 514) were from matched matings of sows from high (avg) maternal EBV sires with boars of high (avg) direct EBV, whereas litters from 2nd and 3rd parity sows (n = 432, 403, respectively) were mated to represent all combinations of sows from high or average maternal EBV sires with high or average direct EBV boars. Farrowing huts were checked each morning by staff of the farm and a trained scientist. Piglets found dead were examined for their cause of death. Criteria for stillbirth were placental membranes covering the face and/or lungs that did not float in water. For piglets that died during the lactation period, the cause of piglet death was identified following post mortem examination and categorized as crushed, low vitality, starved, scour, savaged and others. Crossfostering was low at 8.2% of the piglets (Roehe et al., 2010).

Statistical analyses. Genetic parameters and EBVs were estimated in a multiple trait Bayesian analysis, using a threshold model for survival at birth (SVB), survival during the nursing period (SVNP), survival of piglets by avoiding crushing (SVAC) and a linear Gaussian model for individual birth weight (IBW). The model includes the systematic effects of farm-year-month-parity at farrowing, gestation length, gender of piglets and, additional for SVNP and SVAC, the crossfostering effect. As random effects, the additive and maternal genetic effects, common litter effects and residual effects were fitted in the model.

Results and discussion

The means of SVB, SVNP, SVAC and IBW including weight of dead-born piglets were 96.4%, 88.5%, 92.3% and 1.59 kg, respectively. This indicates that crushing accounts for most of the postnatal mortalities with 7.7% in comparison to 3.8% of other reasons. Generally genetic parameters of direct genetic effects of survival traits and individual birth weight presented in Table 1 were higher than those presented in the literature (e.g. Grandinson et al., 2002; Arango et al., 2006). Fitting different models to analyze the causes of these high direct genetic heritabilities, Roehe et al. (2010) suggested that this piglet's direct genetic effect on its survival is due the challenging conditions of outdoor husbandry.

Table 1: Posterior means of genetic, litter and residual parameters \pm s.e. of piglet survival traits and birth weight (n = 21835 individual piglet observations)^a

Trait	SVB	SVNP	SVAC	IBW				
	————Direct genetic or residual effects————							
SVB	0.22 ± 0.04	0.11 ± 0.14	0.08 ± 0.14	0.18 ± 0.07	-0.35±0.11			
SVNP	0.30 ± 0.09	0.18 ± 0.03	0.53 ± 0.08	0.18 ± 0.08	-0.43±0.09			
SVAC	0.28 ± 0.10	0.90 ± 0.01	0.18 ± 0.03	0.06 ± 0.08	-0.42±0.09			
IBW	0.40 ± 0.03	0.42 ± 0.02	0.35 ± 0.03	0.35±0.02	-0.35±0.05			
]	$Var(L)/s_p^2$						
SVB	0.15 ± 0.02	0.09 ± 0.10	0.08 ± 0.10	0.06 ± 0.07	0.17 ± 0.02			
SVNP	0.19 ± 0.08	0.11 ± 0.01	0.51 ± 0.07	0.16 ± 0.06	0.08 ± 0.01			
SVAC	0.18 ± 0.08	0.59 ± 0.04	0.12 ± 0.01	0.09 ± 0.06	0.08 ± 0.01			
IBW	0.13 ± 0.05	0.21 ± 0.05	0.16 ± 0.05	0.29 ± 0.02	0.16±0.01			

 $^{^{\}alpha}$ Heritabilities on the diagonal and bold, direct or maternal genetic correlations above diagonals, residual or litter correlations below the diagonals; $r_{g(m,d)}$ = genetic correlation between direct and maternal genetic effects of each trait; $Var(L)/s_p^2$ = phenotypic proportion of the litter variance.

Total postnatal survival (SVNP) showed the same heritability as postnatal survival of piglets by avoiding crushing (SVAC), which may be expected because of the high proportion of crushing in the postnatal mortality. However, surprisingly the genetic correlation between SVNP and SVAC is only moderate at 0.53, whereas the residual environmental correlation is very high at 0.90. A moderate negative correlation between direct and maternal effects of -0.42 was found for SVAC, which may indicate competition of piglets for limited maternal resources may cause this undesirable association. SVAC was non-significantly genetically correlated with IBW, so that indirect selection for birth weight may not give any improvement in reduction of piglets being crushed. In contrast, SVNP showed a low significant correlation with IBW, which may suggest that the part of SVNP not caused by crushing may be much more highly genetically correlated with birth weight. The phenotypic fraction of environmental effects common to all piglets in a litter was substantially smaller than both direct and maternal heritability of SVAC. Interestingly, under outdoor conditions, the maternal heritability is lower than the direct heritability for SVAC, but consistently also for all other analyzed traits.

Table 2: Response to selection in direct and maternal genetic effects \pm s.e. in different parities after selection for SVNP^{α}

Selection	SVB (%)		SVNP (%)		SVAC (%)				
parity ——Total response as sum of direct and maternal response——									
High:Avg 1 st	1.82±0.10		2.84 ± 0.27		2.63 ± 0.12				
High:Avg 2 nd	1.16 ± 0.14		1.18±0.26		1.28 ± 0.18				
High:Avg 3 rd	0.78 ± 0.15		2.37±0.27		3.27 ± 0.14				
	Direct	Maternal	Direct	Maternal	Direct	Maternal			
	Second parity and second generation								
HighD:HighM	1.10 ± 0.13	0.06 ± 0.15	-0.46±0.23	1.64 ± 0.30	-0.94 ± 0.15	2.20 ± 0.21			
HighD:AvgM	0.98 ± 0.12	0.05 ± 0.14	-2.04 ± 0.23	0.87 ± 0.29	-1.60±0.15	0.48 ± 0.20			
AvgD:HighM	0.40 ± 0.12	0.61 ± 0.14	0.84 ± 0.23	0.71 ± 0.29	0.36 ± 0.15	1.26±0.20			
AvgD:AvgM	0	0	0	0	0	0			
	————Third parity and second generation———								
HighD:HighM	0.77±0.13	0.01 ± 0.16	0.49 ± 0.24	1.88 ± 0.30	0.41 ± 0.15	2.86 ± 0.21			
HighD:AvgM	0.40 ± 0.13	-0.05±0.14	-0.36±0.23	1.62 ± 0.29	0.09 ± 0.15	1.84 ± 0.20			
AvgD:HighM	0.68 ± 0.13	0.14 ± 0.16	0.30 ± 0.23	1.90 ± 0.29	0.23 ± 0.15	2.26±0.20			
AvgD:AvgM	0	0	0	0	0	0			

^aHigh (Avg)D/M represents the selection groups selected for high (average) direct (D) and maternal (M) EBV of postnatal survival, respectively. Selection response was predicted as least square estimates of EBVs of different selection groups.

The accumulated direct and maternal responses in SVNP and its component SVAC were similar and not significantly different for the first two parities. However, in the third parity significantly higher total response in improvement of piglets not crushed was obtained. This may indicate that the selection for postnatal survival has a long-term effect on later parities. Interestingly, there was a reduction in response in the postnatal survival in the second parity, which may be at least partly due to the cross-classified matings carried out in the second and third parity. In this second parity a negative direct response occurred in SVNP and SVAC

even when selection was for direct genetic effects, mostly likely due to limited maternal genetic effects such as nutrition, mothering ability. The selection pressure (determined as difference of direct and maternal EBVs of sires) on direct genetic effects was in this parity at least twice as high as on the maternal genetic effects. Therefore, the high selection pressure on direct genetic effects associated with traits such as vitality, growth, immune-competence may increase competition within litter for limited maternal genetic resources, and thus even result in a negative direct response. The maternal response for SVAC is, for the maternal selection groups, consistently higher than that for SVNP. This may indicate that selection mainly improved maternal behavior of sows to avoid crushing, but to a lower extent traits associated with low vitality, starvation, scour, savaging. The maternal response to avoid crushing is consistently higher in the third parity compared with that of total postnatal survival. However, the negative direct response was not obtained for SVAC and not significant for SVNP. This may indicate that, with maturity of sows, the limitation of maternal effects may not be as severe as in second-parity sows, when selection pressure for direct genetic effects is higher than for maternal genetic effects.

Conclusion

The genetic analysis indicates that genetic determination of avoiding crushing has a substantial direct and maternal genetic foundation under outdoor conditions. The challenging circumstances of piglet survival under outdoor conditions may have caused these high direct heritabilities. Breeding organizations may use this information to more accurately estimate direct breeding values for piglet survival traits when using outdoor facilities. Although crushing was the main cause of postnatal mortality, the genetic correlation between postnatal survival and survival by avoiding crushing was only of moderate magnitude, suggesting its low genetic associations with other causes of piglet mortality. The maternal selection response of SVAC was substantially higher than that for the entire postnatal survival, suggesting that maternal behavior to avoid crushing has mainly been improved. Because selection was on EBVs for survival based on indoor performance, whereas the selection responses were estimated based on performance in outdoor conditions, the released selection responses suggest that genotype by environment interactions were of minor importance for the selection criteria of postnatal piglet survival. Selection for postnatal survival has been shown to be a robust genetic tool for improvement of avoiding crushing of piglets, but should be used with similar selection pressure on direct and maternal genetic effects of piglet survival to maximize total response of these effects.

References

Arango, J., Misztal, I., Tsuruta, S. et al. (2009). Livest. Sci., 101:208-218.

Baxter, E.M., Jarvis, S., Sherwood, L. et al. (2009). Livest. Sci., 124:266-276.

Grandinson, K., Lund, M.S., Rydhmer, L. et al. (2002). Acta Agric. Scand., 124:266–276.

Roehe, R., Shrestha, N.P., Mekkawy, W. et al. (2009). Livest. Sci., 121:173-181.

Roehe, R., Shrestha, N.P., Mekkawy, W. et al. (2010). J. Anim. Sci., 88:1276-1285.

Su, G., Sorensen, D., and Lund, M.S. (2008). Animal, 2:184-189.