Survival In Laying Hens: Genetic Parameters For Direct And Associative Effects In The Reciprocal Crosses Of Two Purebred Layer Lines

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Introduction

Survival in purebred laying hens is known to be influenced by social interactions (Craig and Muir, 1996). A bird's chance to survive is highly influenced by the cannibalistic behaviour of its cage members. As these negative social interactions are undesirable from both welfare and economic perspectives (Albentosa *et al.*, 2003), measures, such as beak-trimming, are taken to minimize mortality due to cannibalism. In order to put a ban on beak-trimming in a sensible sound way, breeding programs are now aiming for the genetic improvement of laying hens so that they become more sociable. When improving traits affected by social interactions, one should add an associative component to the traditional model. The associative effect represents the heritable effect an individual has on the phenotype of another individual. When accounting for this effect, an individual's chance to survive is not only considered to be affected by the direct effect of the individual, but also by the associative effects of its cage members. When neglecting the associative effects, response to selection could move in the opposite direction (Griffing, 1967; Wade, 1976).

So far, genetic parameters for direct and associative effects have been estimated in purebred layer lines only (Ellen *et al.*, 2008). These parameters give insight in the magnitude of the associative effects. Genetic parameters estimated in purebreds, however, can not simply be extrapolated to crossbreds. Moreover, since improvement of crossbred performance is the ultimate breeding goal, the evaluation of purebred lines based on the performance of their crossbred offspring is of most interest. Therefore, we estimated the genetic parameters for direct and associative effects on survival in the reciprocal crosses of two purebred layer lines.

Material

The dataset consisted of 15,012 laying hens from the reciprocal crosses of two purebred White Leghorn layer lines, W1 and WB, of which 7,668 were a W1xWB ($\exists x$) and 7,344 were a WBxW1 ($\exists x$) cross. On average, each cross was produced using 65 purebred sires and 720 purebred dams. The animals originated from the Institut de Sélection Animale B.V., the layer division of Hendrix Genetics.

At the age of 17 weeks, the hens were placed in two laying houses. Each laying house consisted of 4-5 double rows and each row consisted of three levels. Interaction with back neighbours was possible, but interaction with side neighbours was prevented. Four hens of the same crossbred line were randomly assigned to a cage. Hens were not beak-trimmed.

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The trait of interest, 'survival days', was defined as the number of days from the start of the experiment up until death or the end of the experiment, with a maximum of 398 days. Dead laying hens were recorded and removed daily.

Methods

A Kaplan-Meier survival curve was used to gain insight in the phenotypic difference between the reciprocal crosses with regard to the number of survival days.

A bivariate animal model, either including or excluding associative effects, was used to investigate the magnitude of the associative effects and to calculate the genetic correlation between direct and associative effects within and between crosses:

$$\begin{bmatrix} \mathbf{y}_1 \\ \mathbf{y}_2 \end{bmatrix} = \begin{bmatrix} \mathbf{X}_1 & 0 \\ 0 & \mathbf{X}_2 \end{bmatrix} \begin{bmatrix} \mathbf{b}_1 \\ \mathbf{b}_2 \end{bmatrix} + \begin{bmatrix} \mathbf{Z}_1 & 0 \\ 0 & \mathbf{Z}_2 \end{bmatrix} \begin{bmatrix} \mathbf{a}_{1_D} \\ \mathbf{a}_{2_D} \end{bmatrix} + \begin{pmatrix} \begin{bmatrix} \mathbf{Z}_1 & 0 \\ 0 & \mathbf{Z}_2 \end{bmatrix} \begin{bmatrix} \mathbf{a}_{1_S} \\ \mathbf{a}_{2_S} \end{bmatrix} + \begin{bmatrix} \mathbf{V}_1 & 0 \\ 0 & \mathbf{V}_2 \end{bmatrix} \begin{bmatrix} \mathbf{cage}_1 \\ \mathbf{cage}_2 \end{bmatrix} + \begin{bmatrix} \mathbf{e}_1 \\ \mathbf{e}_2 \end{bmatrix}$$

The fixed effects include an interaction term for each laying house*row*level and a covariate for the average number of survival days in the back cage. The random effects include a direct animal effect, an associative animal effect and a cage effect.

The inclusion of an associative animal effect redefines the heritable impact of an individual i on the population, as the total breeding value (TBV) now consists of a direct breeding value (DBV) and an associative breeding value (SBV), $TBV_i = DBV_i + (n-1)SBV_i$, where n denotes the number of cage members. Consequently, the total heritable variance equals $\sigma_{TBV}^2 = \sigma_{A_D}^2 + 2(n-1)\sigma_{A_{DS}} + (n-1)^2\sigma_{A_S}^2$ (Bijma $et\ al.$, 2007) and the total "heritability" equals $T^2 = \sigma_{TBV}^2/\sigma_P^2$ (Bergsma $et\ al.$, 2008). Because response to selection equals $\Delta G = \iota \rho \sigma_{TBV}$, where ι denotes the intensity and ρ denotes the accuracy, the σ_{TBV}^2 truly represents the heritable variance relevant for response to selection.

The descriptive statistics and the determination of the fixed effects were performed by SAS v9.1 (SAS Institute Inc., 2003), while the genetic parameters were estimated using ASREML v2.0 (Gilmour *et al.*, 2006).

Results and Discussion

The Kaplan-Meier survival curve shows a significantly higher survival rate for the W1xWB cross when compared to the WBxW1 cross (Figure 1), with a final survival rate of 61% and 51%, respectively.

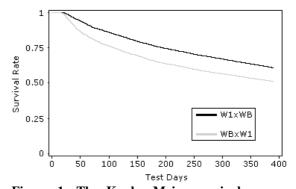


Figure 1: The Kaplan-Meier survival curve. Plotting the number of test days (0-398) against the survival rate (0-1) for the reciprocal crosses of two purebred layer lines.

Table 1: Estimated parameters for survival days, based on a direct and a direct associative animal model.

	Direct animal model		Direct-associative animal model	
_	W1xWB	WBxW1	W1xWB	WBxW1
$\sigma_{cage}^{^{2}}{}^{lpha}$	$2,765 \pm 213$	$2,918 \pm 265$	$1,991 \pm 260$	$2,366 \pm 307$
$\sigma_{a_{\scriptscriptstyle D}}^{\scriptscriptstyle 2}{}^{\scriptscriptstyle eta}$	710 ± 179	$1,\!280 \pm 278$	541 ± 153	987 ± 224
$\sigma_{\scriptscriptstyle a_{\scriptscriptstyle DS}}^{ \gamma}$			-196 ± 92	-723 ± 140
$\sigma_{a_{\scriptscriptstyle S}}^{\scriptscriptstyle 2}{}^{\delta}$			529 ± 108	772 ± 148
$\sigma_{_{e}}^{^{2\epsilon}}$	$12,325 \pm 270$	$17,112 \pm 392$	$11,728 \pm 298$	$15,657 \pm 460$
h^2 - T^2 ζ	0.04 ± 0.01	0.06 ± 0.01	0.26 ± 0.06	0.17 ± 0.05
$r_{DS}^{ \ \eta}$			-0.37 ± 0.17	-0.83 ± 0.10
$\sigma_{a_D}^{ heta}$	763 ± 251		682 ± 208	
$\sigma_{a_{\scriptscriptstyle S}}^{ \iota}$			265 ±	172
$r_{\!\scriptscriptstyle D}^{\kappa}$	0.80 ± 0.22		0.93 ± 0.23	
$r_{_S}^{\lambda}$			0.41 ± 0.25	
r^{μ}			0.63 ± 0.31	

r cage variance; β direct variance; γ direct-associative covariance within crosses; δ associative variance; ϵ error variance; β heritability and total heritability, respectively; β genetic correlation between the DBVs and SBVs of a specific cross; β direct covariance between crosses; β associative covariance between crosses; β genetic correlation between the DBVs of both crosses; β genetic correlation between the TBVs of both crosses.

The associative genetic variance was large and strongly significant in both crosses. The estimates are two to six times larger than those found by Ellen *et al.* (2008) in the purebred parental lines. The genetic correlation between DBVs and SBVs was negative, and differed considerably between crosses. The strong negative genetic correlation of -0.83 for the WBxW1 cross is exceptional when compared to the W1xWB cross (-0.37) or the purebred W1 (0.18) and WB (-0.31) line (Ellen *et al.*, 2008). For socially affected traits, the realized heritability in case of mass selection equals $[\sigma_{A_D}^2 + (n-1)\sigma_{A_{DS}}]/\sigma_P^2$ (Griffing, 1967), which

equals to 0.00 for W1xWB and -0.06 for WBxW1. Hence, despite clear heritable variance, W1xWB would fail to respond to mass selection and WBxW1 would show increased mortality. Overall, the difference between h² and T² indicates once again the importance of associative effects. The bivariate analyses also gives the direct and associative correlations between the reciprocal crosses. The genetic correlation between DBVs was high (0.93), while the genetic correlation between SBVs was only moderate (0.41). Moreover, the genetic correlation between TBVs (0.63) shows that both crosses are not as similar as suggested by the direct animal model (0.80).

The difference between the reciprocal crosses indicates that it matters which parental line provides the sire and which provides the dam. This difference could have multiple underlying causes, such as imprinting, (cytoplasmatic) maternal effects or sex-chromosome linked effects. Imprinting is assumed to be a phenomenon exclusive for placental mammals, though there are indications that it could be present in chickens as well (Tuiskula-Haavisto *et al.*, 2004). (Cytoplamatic) maternal effects, *e.g.* their influence on early growth or the transmission of antibodies through the egg (Fairfull and Gowe, 1986), could influence the number of survival days. The difference between both crosses could also be attributed to the Z-chromosome, which, in comparison with the W-chromosome, caries more genetic information (Gowe and Fairfull, 1995). Additional analysis will be performed in order to understand the origin of the difference between the reciprocal crosses.

Conclusion

The associative genetic variance was large and strongly significant in both crosses, and contributed up to 87% of total heritable variance. Compared to the purebred parental lines, the associative genetic variance in the crossbreds was considerably larger. The genetic correlation between direct and associative effects was negative to highly negative, and differed between crosses. The genetic correlation between TBVs for both crosses was moderate, indicating that the selection of purebred parents, when aiming to increase crossbred performance, should be different for both crosses. Overall, the results clearly indicate the need to account for associative effects when breeding for increased survival in non beak-trimmed crossbreds.

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