Survival of Laying Hens: Genetic Parameters for Direct and Associative Effects in Three Purebred Layer Lines

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ABSTRACT Mortality due to cannibalism is a major problem in laying hens. Due to prohibition of beak-trimming in the European Union, this problem will increase in the near future. One solution to reduce mortality due to cannibalism is to use genetic selection. Mortality due to cannibalism, however, differs from conventional breeding traits, because it depends on social interactions among individuals. Selection strategies aiming to reduce cannibalism, therefore, should consider both the direct effect of an individual on its own survival and the social effect of the individual on the survival of its group members (the so-called associative effect). Traditional breeding, however, accounts for only the direct effect. Recently, methods have been proposed to estimate variance components and breeding values for both direct and associative effects. This paper presents estimated genetic parameters for direct and associative effects on survival days in 3 purebred laying lines. For the analysis, 16,780 hens with intact beaks were used. When considering only direct effects, heritabilities ranged from 2 through 10%. When considering both direct and associative effects, the total heritable variance, expressed as a proportion of phenotypic variance, ranged from 6 through 19%. These results show that heritable variation in survival days is substantially larger than suggested by conventional direct effects models. This means that prospects for reducing mortality by means of genetic selection are good and may lead to substantial reduction of 1 of the major welfare problems in egg production.

Key words: social interaction, variance component estimation, laying hen, survival, indirect genetic effect

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INTRODUCTION

Social interactions among individuals can have profound influences on the expression of performance traits like production and welfare traits in domestic livestock populations (Muir, 1996; Brichette et al., 2001; Denison et al., 2003; Muir, 2005; Bijma et al., 2007a). For instance, social interactions can reduce growth due to competition for limited resources or can result in mortality due to cannibalism. The latter is seen in laying hen production systems. Mortality due to cannibalism is an economic and welfare problem occurring in all types of commercial poultry housing systems (Albentosa et al., 2003). Furthermore, due to prohibition of beak-trimming in the European Union in the near future, this problem may increase. One of the possibilities to reduce mortality due to cannibalism is to use genetic selection (Muir, 1996; Jones and Hocking, 1999).

Mortality due to cannibalism is caused by social interactions among group members. Wolf (2003) mentioned that the environment provided by group members is often the most important component of the environment experienced by an individual in that group. Although the interaction between group members may appear to be purely environmental, they differ from other sorts of environmental influences, because they can have a genetic basis (Wolf et al., 1998; Wolf, 2003). Traditional breeding, using mass selection or selection based on information of relatives, has mainly focused on improving the direct effect of the genotype of the individual on its phenotype (except for maternal effect models). With the exception of maternally affected traits, traditional breeding has neglected the social effect of an individual on the phenotypes of its group members. This social effect is often called an associative effect (Griffing, 1967). When the objective is to improve traits affected by interactions among individuals, the use of traditional models can result in response to selection in the opposite direction (Griffing, 1967). For instance, Wade (1976) showed that individual selection for increased population size of flour beetle (*Tribolium castaneum*) decreased population size in the next generation. To improve traits affected by interactions among individuals, the usual model for a given genotype must be extended to consider not only the direct effects of its own genes but also the associative effect of the

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individual on the phenotypes of its group members (Griffing, 1967). One solution is to use group selection (Griffing, 1967). Using group selection, both Muir (1996) and Wade (1976, 1977) found a decrease in mortality due to cannibalism in, respectively, laying hens and flour beetles.

With respect to agriculture, it is important to understand how to improve traits affected by interactions among individuals so as to enhance animal well-being and productivity in confined high-intensity rearing conditions (Muir, 2005). Determining the relevance of interactions among individuals for breeding programs requires knowledge of the genetic parameters underlying the interactions (Bijma et al., 2007a). Such knowledge would allow one to quantify the potential contribution of associative effects to response to selection, to optimize poultry breeding programs, and to estimate breeding values for both direct and associative effects.

The existence of social interactions among individuals may increase the total heritable variance in a trait (Brichette et al., 2001; Wolf, 2003; Bijma et al., 2007a). Bijma et al. (2007a) found that total heritable variance in survival days expressed as proportion of phenotypic variance increased from 7 through 20% due to social interactions. This indicates that ⅔ of the heritable variation is due to interactions among individuals and is hidden from traditional analysis. These results, however, were based on a relatively small data set (n = 3,800). Until now, these are the only results that show evidence that heritable variation will increase due to social interactions. Thus, more evidence is needed to confirm the relevance of social interactions for genetic improvement of poultry populations.

In this paper, we present estimated genetic parameters for 2 models, the usual direct effects model and a model combining direct and associative effects. For this, we use data on survival days in 3 purebred layer lines.

MATERIALS AND METHODS

Genetic Stock

Three purebred White Leghorn layer lines from the Institut de Sélection Animale B.V., a Hendrix Genetics company, were used in this study. The 3 lines were coded: W1, WB, and WF. The lines W1 and WB were expected to have high mortality with intact beaks. The line WF was chosen, because it was characterized as a high-feather-pecking line in earlier experiments (Riedstra and Groothuis, 2002; Van Hierden et al., 2002; Rodenburg et al., 2003).

Housing Conditions and Management

For each strain, observations on a single generation were used. Chickens were hatched in 2 batches, and each batch consisted of 3 lines. Furthermore, each batch consisted of 4 age groups, differing by 2 wk each. After hatching, chickens were sexed, wing-banded in the right wing, and vaccinated for Marek's disease and infectious bronchitis. Chickens had intact beaks. Chickens of the same line and age group were allocated to rearing cages of 60 individuals per cage. Rearing cages were composed at random with respect to family. From wk 5 onwards, chickens were housed with 20 individuals per cage.

Table 1. Mean light intensity (lx) and SD in laying house 1 and 2

	Laying house 1		Laying house 2	
Level	L[1]	B[2]	L	B
Top	240 ± 180	63 ± 42	353 ± 7	22 ± 2
Middle	177 ± 150	105 ± 185	282 ± 23	27 ± 2
Bottom	81 ± 99	54 ± 116	122 ± 8	27 ± 3

[1]L = underneath strip light.
[2]B = in between 2 strip lights.

When the hens were on average 17 wk old, they were transported to 2 laying houses with traditional 4-bird battery cages. Each batch was placed in another laying house. In both laying houses, the 17-wk-old hens were allocated to laying cages, with 4 birds of the same line and age in a cage. The individuals making up a cage were combined at random. Due to chance, some of the cages contained full or half sibs, but most cages contained unrelated individuals only. Due to lost wing bands, hens were wing-banded in the left wing as well, to avoid loss of data.

In both laying houses, rows were grouped into 8 double rows. Individuals could have contact only with the back neighbors, because the back wall of the cages consisted of mesh allowing limited contact between back neighbors, whereas adjacent cages in the same row were separated by a closed wall. In between each double row, there was a corridor through which the employees could access the cages. Each row consisted of 3 levels (top, close to the light; middle; and bottom). Hens in laying house 2 were placed only in the middle and bottom level. Each level was divided into blocks of 10 cages; each block consisted of the same line and age. In general, the same line and age were also housed in the corresponding back cages. A feeding trough was in the front of the cages, and each pair of back-to-back cages shared 2 drinking nipples. A standard commercial layer diet and water were provided ad libitum.

In both laying houses, the hens started with a light period of 9 h/d. The light period was increased 1 h/wk until 16 h/wk was reached when the hens were on average 26 wk of age. In laying house 1, alongside the first and the last row, there were windows, giving an effect of daylight. In laying house 2, there was no daylight. On average, light intensity was higher in laying house 2 than in laying house 1 (Table 1). Light intensity in laying house 1, however, depended predominantly on the weather conditions outside and was therefore highly variable.

Pedigree

For both laying houses, almost the same sires were used; for laying house 2, a few sires could not be used

Table 2. Breeding scheme of the 3 layer lines per laying house

	Laying house 1		Laying house 2	
Line	Sires	Dams	Sires	Dams
W1	36	287	32	250
WB	35	276	33	261
WF	20	159	18	135

because of mortality (Table 2). The dams used were different for both laying houses. For all 3 lines, sires and dams were mated at random. Each sire was mated to approximately 8 dams, and each dam contributed on average 12.3 female offspring. Five generations of pedigree were included in the calculation of the relationship matrix (**A**). To ensure correct pedigree, hens with unknown identification or double identification were coded as having unknown pedigree (n = 101). The observations on these hens were included in the analysis but did not contribute to estimates of genetic (co)variances.

Data

All hens were observed daily. Dead hens were removed, and wing band number, cage number, and cause of death were recorded. Determination of cause of death was done subjectively without dissection. Removed hens were not replaced. When the hens were on average 75 wk old, the study was terminated. For each hen, information was collected on survival and number of survival days. Survival was defined as alive or dead (0/1) at the end of the study. From this data, survival rate was calculated as the percentage of laying hens still alive at the end of the study. Survival days were defined as the number of days from the start of the study (day of transport to laying houses, when the hens were on average 17 wk old) till either death or the end of the study, with a maximum of 447 d. For the statistical analysis, 16,780 records were used: 6,276 records of W1, 6,916 records of WB, and 3,588 records of WF (Table 3).

Data Analysis

Model. First, the data on survival days were analyzed using the GLM procedure of the SAS statistical program (SAS, 1996). This program was used to decide which fixed effects to include in the model for estimating genetic parameters. The data were analyzed separately for each line. The initial model included a fixed effect for each laying house-row-level combination and for average survival days in the back cage to account for a possible effect of the back neighbors. Age was fully confounded with laying house and row and, therefore, not included as a separate fixed effect.

Second, genetic parameters on survival days were estimated using a linear animal model as implemented in the ASReml software package (Gilmour et al., 2002). The traditional direct effects model was used to estimate genetic parameters for the direct effect:

$$\mathbf{y} = \mathbf{Xb} + \mathbf{Za} + \mathbf{e}, \quad [1]$$

where $\mathbf{y}$ = a vector of observed survival days; $\mathbf{b}$ = a vector of fixed effects, with incidence matrix $\mathbf{X}$ linking observations to fixed effects; $\mathbf{a}$ = a vector of the usual breeding values, with incidence matrix $\mathbf{Z}$ linking observations on individuals to their breeding value; and $\mathbf{e}$ = a vector of random residuals. The fixed effects in $\mathbf{b}$ account for systematic nongenetic differences among observations. Covariance structures of model terms are: Var**[a]** = $\mathbf{A}\sigma^2_A$, where $\mathbf{A}$ = a matrix of coefficients of relatedness between individuals and σ^2_A = the genetic variance, and Var**[e]** = $\mathbf{I}\sigma^2_e$, where $\mathbf{I}$ = an identity matrix and σ^2_e = the residual variance.

To estimate genetic parameters for both direct and associative effects, the model of Bijma et al. (2007a) was used, the direct-associative effects model:

$$\mathbf{y} = \mathbf{Xb} + \mathbf{Z}_D\mathbf{a}_D + \mathbf{Z}_S\mathbf{a}_S + \mathbf{e}, \quad [2]$$

where $\mathbf{a}_D$ = a vector of direct breeding values, with incidence matrix $\mathbf{Z}_D$ linking observations on individuals to their direct breeding value; $\mathbf{a}_S$ = a vector of associative breeding values, with incidence matrix $\mathbf{Z}_S$ linking observations on individuals to the associative breeding values of their group members (i.e., individuals in the same cage); and $\mathbf{e}$ = a vector of residuals. When there are no social interactions among individuals, the term $\mathbf{Z}_S\mathbf{a}_S$ equals 0, $\mathbf{Z}_D\mathbf{a}_D$ reduces to $\mathbf{Za}$, and equation [2] is identical to equation [1].

The covariance structure of genetic terms is $\text{Var}\begin{bmatrix}\mathbf{a}_D\\ \mathbf{a}_S\end{bmatrix} = \mathbf{C} \otimes \mathbf{A}$, where $\mathbf{C} = \begin{bmatrix}\sigma^2_{A_D} & \sigma_{A_{DS}}\\ \sigma_{A_{DS}} & \sigma^2_{A_S}\end{bmatrix}$ and where $\sigma^2_{A_D}$ = the direct genetic variance; $\sigma^2_{A_S}$ = the associative genetic variance; and $\sigma_{A_{DS}}$ = the direct-associative genetic covariance. The residual term in equation [2] is actually the direct environmental effect of the individual plus the sum of environmental effects of its group members: $e_i = E_{D,i} + \sum_{j\neq i}^{n-1} E_{S,j}$. The covariance structure of the residual term, $\mathbf{e}$, is given by $\text{Var}(\mathbf{e}) = \mathbf{R}\sigma^2_e$, where $R_{ij} = 1$ when $i = j$ and $R_{ij} = \rho$ when i and j are in the same cage ($i \neq j$), but $R_{ij} = 0$ otherwise, with $\sigma^2_e = \sigma^2_{E_D} + (n-1)\sigma^2_{E_S}$ Bijma et al., 2007a). The residuals of the group members may be correlated due to nongenetic interactions among cage members. The correlation equals $\rho = [2\sigma_{E_{DS}} + (n-2)\sigma^2_{E_S}]/\sigma^2_e$ (Bijma et al., 2007a). The value of ρ is estimated in the analysis.

Heritable Variation. When there are interactions among individuals, each individual interacts with $n-1$ group members. The total heritable effect of an individual on the population, called its total breeding value (**TBV**), equals the sum of its direct breeding value and $n-1$ times its associative breeding value: $\text{TBV}_i = \text{A}_{D,i} + (n-1)\,\text{A}_{S,i}$. The total heritable variation equals the variance of

Table 3. Number of birds (n), survival rate (%) with SE, and average survival days (d) with SE of the 3 layer lines and the fixed effects

	Laying house 1			Laying house 2		
Item	n	Survival rate[1]	Survival days[2]	n	Survival rate	Survival days
Line						
W1	3,900	53.6 ± 1.3	344 ± 3.6	2,376	64.6 ± 1.5	366 ± 4.3
WB	3,796	50.2 ± 1.3	323 ± 3.6	3,120	56.3 ± 1.5	329 ± 4.1
WF	2,004	74.1 ± 1.1	376 ± 2.9	1,584	75.1 ± 1.2	370 ± 3.3
Laying house	9,700	56.5 ± 0.5	342 ± 1.3	7,080	63.3 ± 0.8	350 ± 2.1
Level						
Top	3,212	52.5 ± 1.2	330 ± 3.2	—	—	—
Middle	3,236	58.0 ± 1.2	348 ± 3.2	3,540	63.8 ± 1.1	353 ± 3.2
Bottom	3,252	59.0 ± 0.9	350 ± 2.3	3,540	62.8 ± 0.8	348 ± 2.3
Row						
1	1,208	61.3 ± 2.0	353 ± 5.2	872	73.5 ± 2.3	364 ± 6.4
2	1,220	64.5 ± 2.0	359 ± 5.2	884	65.1 ± 2.3	341 ± 6.4
3	1,172	66.9 ± 2.0	368 ± 5.3	880	66.7 ± 2.3	355 ± 6.4
4	1,216	68.8 ± 2.0	373 ± 5.2	880	60.6 ± 2.3	343 ± 6.4
5	1,224	50.7 ± 2.0	330 ± 5.2	896	63.4 ± 2.3	358 ± 6.3
6	1,224	50.8 ± 2.0	333 ± 5.2	872	59.3 ± 2.3	351 ± 6.4
7	1,212	43.2 ± 2.0	306 ± 5.2	896	58.2 ± 2.3	346 ± 6.3
8	1,224	46.1 ± 1.4	318 ± 3.7	900	59.9 ± 1.6	346 ± 4.5

[1]Survival rate = percentage of laying hens still alive at the end of the study.

[2]Survival days = average number of days from the start of the study (on average 17 wk old) till death, with a maximum of 447 d.

the TBV among individuals, $\sigma^2_{TBV} = \sigma^2_{A_D} + 2(n-1)\sigma_{A_{DS}} + (n-1)^2\sigma^2_{A_S}$ (Bijma et al., 2007a,b). The σ^2_{TBV} represents the total heritable variation that can be utilized to generate response to selection (ΔG). Thus, response to selection per generation is given by $\Delta G = \iota\rho\sigma_{TBV}$, where ι = the selection intensity; ρ = the accuracy; and σ_{TBV} = the SD of total breeding value. When there are no interactions among individuals, σ_{TBV} reduces to the usual σ_A (Ellen et al., 2007). It follows from equation [2] that the total phenotypic variance equals $\sigma^2_P = \sigma^2_{A_D} + (n-1)\sigma^2_{A_S} + \sigma^2_e$. The total heritable variance expressed as a proportion of the phenotypic variance (T^2) equals $\frac{\sigma^2_{TBV}}{\sigma^2_P}$.

RESULTS

Survival

Line WF showed the highest survival rate of 74.6% and the highest survival days of 373 d, whereas line WB showed the lowest survival rate of 52.9% and the lowest survival days of 326 d (Table 3). Both survival days and survival rate differed significantly between lines. Laying house 2 showed significantly higher survival rate over the whole study period (63.3%) and a slightly higher number of survival days (350 d) than laying house 1 (56.5% and 342 d; Table 3). A difference in survival rate and survival days was found between the 3 levels and between the 8 corridors. All fixed effects included in the model were significant.

Line WB showed the lowest survival rate in both laying houses (Figure 1). At the end of the laying period, ranking of the lines was the same for both laying houses. In laying house 2, however, line W1 showed until 260 d the highest survival rate, whereas from 260 d onwards, line WF showed the highest survival rate.

Genetic Parameters

The estimated genetic parameters on survival days in the 3 layer lines, using the direct effects model, are given in Table 4. The lowest additive genetic SD (σ_A) was found in line WF and the highest in line WB, ranging from 16 through 44 d. Heritabilities ranged from 2% in line WF (not significantly different from 0) through 10% in line WB (significantly different from 0).

In Table 5, results of the direct-associative effects model are given. For the line WF, all results are not significantly different from 0. Both the direct genetic variance ($\sigma^2_{A_D}$) and the associative genetic variance ($\sigma^2_{A_S}$) were highest in line WB and lowest in line WF. The $\sigma^2_{A_D}$ ranged from 246 through 1,917 d^2, and $\sigma^2_{A_S}$ ranged from 60 through 273 d^2. The covariance between direct and associative effect ($\sigma_{A_{DS}}$) was negative in line WB and positive in line W1 and WF, ranging from –228 through 62 d^2 (W1). The SD

Table 4. Estimates of genetic parameters[1] with SE for direct effect on survival days in 3 layer lines using a traditional linear animal model

Parameter	Unit	W1	WB	WF
σ_A	d	30 ± 4	44 ± 5	16 ± 5
σ^2_P	d^2	12,814 ± 239	20,066 ± 367	13,936 ± 333
h^2		0.07 ± 0.02	0.10 ± 0.02	0.02 ± 0.01

[1]σ_A = the additive genetic SD; σ^2_P = the phenotypic variance: $\sigma^2_P = \sigma^2_A + \sigma^2_e$; h^2 = the heritability: $h^2 = \sigma^2_A/\sigma^2_P$.

Table 5. Estimates of genetic parameters[1] with SE for direct and associative effect on survival days in 3 layer lines using the linear animal model of Bijma et al. (2007a)

Parameter	Unit	W1	WB	WF
$\sigma^2_{A_D}$	d^2	915 ± 218	1,917 ± 394	246 ± 159
$\sigma^2_{A_S}$	d^2	134 ± 51	273 ± 85	60 ± 61
$\sigma_{A_{DS}}$	d^2	62 ± 76	−228 ± 132	13 ± 69
σ_{TBV}	d	50 ± 8	55 ± 9	30 ± 21
σ^2_P	d^2	12,847 ± 245	20,111 ± 374	13,999 ± 343
T^2		0.19 ± 0.06	0.15 ± 0.05	0.06 ± 0.06
r_A		0.18 ± 0.21	−0.31 ± 0.18	0.11 ± 0.55
ρ		0.08 ± 0.02	0.08 ± 0.02	0.10 ± 0.02

[1] $\sigma^2_{A_D}$, $\sigma^2_{A_S}$, and $\sigma_{A_{DS}}$ = estimates of direct genetic variance, associative genetic variance, and direct-associative genetic covariance; σ_{TBV} = the SD of the total breeding value: $\sigma^2_{TBV} = \sigma^2_{A_D} + 2(n-1)\sigma_{A_{DS}} + (n-1)^2\sigma^2_{A_S}$; σ^2_P = the phenotypic variance: $\sigma^2_P = \sigma^2_{A_D} + (n-1)\sigma^2_{A_S} + \sigma^2_e$; T^2 = the total heritable variance relative to the phenotypic variance: $T^2 = \sigma^2_{TBV}/\sigma^2_P$; r_A = the genetic correlation between direct breeding value and associative breeding value; ρ = the correlation between the residuals of the group members.

of the TBV (σ_{TBV}) ranged from 30 d (WF) through 55 d (WB). Line WF showed the lowest total heritable variance in survival days expressed as proportion of phenotypic variance (T^2), whereas line W1 showed the highest T^2; ranging from 6 through 19%. The T^2 expresses the total heritable variance relative to the phenotypic variance and is, therefore, a generalization of the conventional h^2 to account for social interactions. The genetic correlation between direct breeding value and associative breeding value (r_A) was positive but not significantly different from 0 in line W1 (0.18) and in line WF (0.11) and negative and significantly different from 0 in line WB (–0.31). Furthermore, the estimates of the correlation between the residuals of the group members (ρ), ranged from 0.08 in line W1 and WB through 0.10 in line WF and were highly significant.

DISCUSSION

In this paper, we showed that it is possible to estimate genetic parameters for both direct and associative effect on survival days in 3 purebred layer lines. Furthermore, we showed that including associative effects in the model resulted in a substantially larger heritable variation than was found when using the traditional linear animal models. This result demonstrates the relevance of associative effects for poultry breeders and indicates that prospects for genetic improvement of survival in laying hens are substantially better than suggested by traditional heritabilities.

In our study, survival rate ranged from 52.9 through 74.6% between lines. In other studies, survival rates were found ranging from 69.4 through 94.2% (Craig and Muir, 1989; Kjaer and Vestergaard, 1999). In both studies, however, the hens were beak-trimmed and were kept in larger groups. The fact that we used birds that had intact beaks explains the, on average, lower survival rates in our study.

In our study, survival rate was different between the 2 laying houses. Survival rate was lowest in laying house 1, which could be due to the effect of daylight. Furthermore, survival rate was lowest in the top level (laying house 1), which could be due to higher light intensity (Table 1). Difference in light intensity, however, did not change the ranking of the lines; it only influenced the level of the survival rate. In other studies, it was also found that high light intensity resulted in a decrease in survival rate (Hughes and Duncan, 1972; Kjaer and Vestergaard, 1999).

In poultry breeding, the trait survival days are more important than the trait survival rate, because survival days show when a laying hen died (i.e., in the beginning or at the end of the laying period). That is why, in this study, the trait survival days were chosen. No literature, however, was found that showed heritabilies for survival days using the direct effects model. Estimated heritabilities were found only for survival as a binary trait. Using the direct effects model, estimated heritabilities for survival days were comparable with heritabilities found for survival as a binary trait, ranging from 3.2 through 9.9% (Robertson and Lerner, 1949; Craig and Muir, 1989; Mielenz et al., 2005). Furthermore, we found that heritabilities, using the direct effects model, for survival as a binary trait ranged also from 3 through 12% (data not shown).

In a simulation study, Van Vleck and Cassady (2005) showed that ignoring a cage effect biases estimates of genetic parameters. In this study, we accounted for nonheritable social effects by fitting a correlation (ρ) between the residuals of cage members (see also Bijma et al., 2007a). Fitting a correlated residual allows cage members to be either similar or dissimilar, corresponding to either a positive or a negative correlation. When cage members are similar due to nonheritable social effects, fitting a random cage effect instead of a correlated residual yields the identical variance. In other words, when cage members are similar, one can fit either a variance between cages or a covariance within cages. The relationship between both models is that $\sigma^2_{cage} = \rho\sigma^2_e$. The equivalence of both models is, however, limited to the situation in which cage members are similar, because σ^2_{cage} cannot be negative. Whether cage members are similar or not is unknown a priori. The covariance between residuals of cage mem-

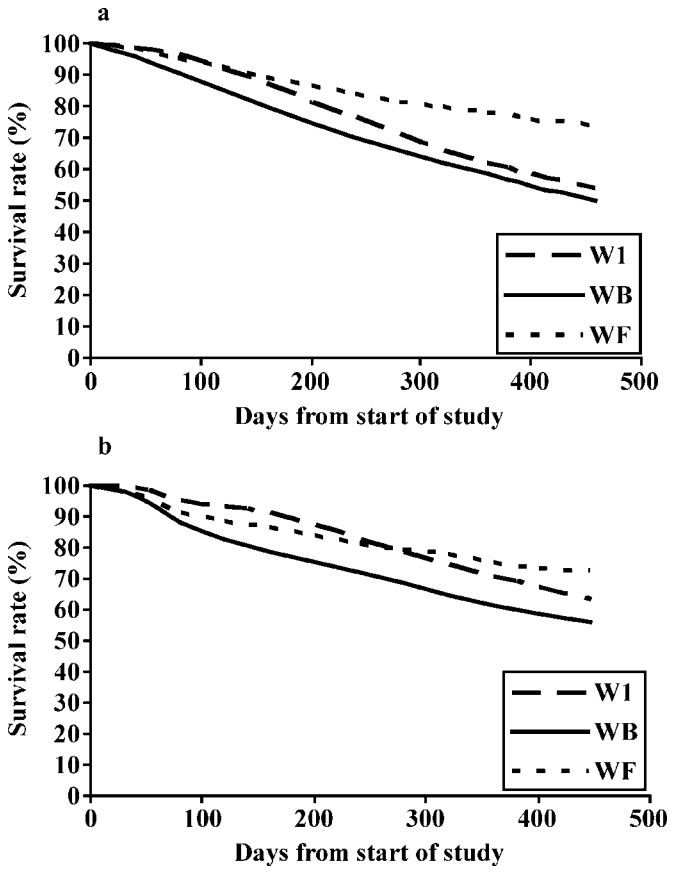

Figure 1. Survival curve of the 3 layer lines, W1, WB, and WF, of laying house 1 (a) and laying house 2 (b).

bers is equal to $2\sigma_{IE_{DS}} + (n - 2)\sigma^2_{E_S}$ and can be either positive or negative (Bijma et al., 2007a). The general solution to account for nonheritable social effects is, therefore, to fit a correlation between residuals of cage members, not to fit a random cage effect. Moreover, fitting both a correlated residual and a random group effect means that 2 variables are fitted to account for a single unknown, which overspecifies the variance structure and does not yield a unique solution.

Including associative effects in the model, the total heritable variance in survival days expressed as proportion of phenotypic variance (T^2) was 1.5- through 3-fold greater than when using the direct effects model. Line W1 showed the same T^2, of 19%, as found by Bijma et al. (2007a) on 50% of the data used in the present study. The underlying genetic parameters were, however, slightly different between the 2 studies. The present results, therefore, confirm the preliminary results of Bijma et al. (2007a).

For growth in mussel cultures, it was found that the genetic correlation between direct and associative effect was negative; individuals getting more food or space would deprive their group members (Brichette et al., 2001). Based on the results of the survival rates, it was expected that correlations between direct and associative effect for survival in hens would be negative because of strong competition. It was expected that dominant animals may kill others and, as a consequence, survive themselves. For line WB, indeed a negative (significantly different from 0) correlation was found between direct and associative effect for survival. However, for line W1 and WF, a positive genetic correlation between direct and associative effect for survival days was found, suggesting that individuals benefit from not harming others (Bijma et al., 2007a). The results of line W1 and WF, however, are not significantly different from 0; it could be that the genetic correlation between direct and associative effect was positive by coincidence. Furthermore, survival rate in line WF is high, which reduced the accuracy of estimated genetic parameters.

Genetic parameters are usually estimated by a linear model in which the dependent variables and the random variables are assumed to be normally distributed. In this study, genetic parameters of survival data were also estimated using a linear animal model. Survival data is, however, heavily skewed (Kachman, 1999). Furthermore, for hens still alive at the end of the study, only a lower bound of the exact survival days will be available. These data are called censored data (Kalbfleisch and Prentice, 1980). To analyze survival data, the appropriate method would be survival analysis, which can be done using the survival kit (Ducrocq and Sölkner, 1998). Until now, however, it is not possible to estimate genetic parameters for both direct and associative effect using that software package. When using survival analysis including associative effects, we would, however, expect that the proportion of heritable variation will even be higher than when using a linear animal model including associative effects.

In conclusion, it is possible to estimate genetic parameters for direct and associative effects on survival in laying hens. The results of this study show that including associative effects in the model will give substantially higher heritable variation than when using the conventional direct effects model. When designing a breeding program, estimation of the genetic parameters for all lines is needed. Furthermore, environmental factors, like group size and light intensity, are important, because they can have an effect on the genetic parameters. Theoretical work shows that prospects for reduction of mortality using the direct-associative effects model are good (Bijma et al., 2007b; Ellen et al., 2007). Genetic selection targeting both direct and associative effects is expected to substantially reduce 1 of the major welfare problems in egg production.

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