

The Contribution of Social Effects to Heritable Variation in Finishing Traits of Domestic Pigs (*Sus scrofa*)

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ABSTRACT

Social interactions among individuals are ubiquitous both in animals and in plants, and in natural as well as domestic populations. These interactions affect both the direction and the magnitude of responses to selection and are a key factor in evolutionary success of species and in the design of breeding schemes in agriculture. At present, however, very little is known of the contribution of social effects to heritable variance in trait values. Here we present estimates of the direct and social genetic variance in growth rate, feed intake, back fat thickness, and muscle depth in a population of 14,032 domestic pigs with known pedigree. Results show that social effects contribute the vast majority of heritable variance in growth rate and feed intake in this population. Total heritable variance expressed relative to phenotypic variance was 71% for growth rate and 70% for feed intake. These values clearly exceed the usual range of heritability for those traits. Back fat thickness and muscle depth showed no heritable variance due to social effects. Our results suggest that genetic improvement in agriculture can be substantially advanced by redirecting breeding schemes, so as to capture heritable variance due to social effects.

SOcial interactions among individuals are ubiquitous both in animals and in plants, and in natural as well as domestic populations. These interactions affect both the direction and the magnitude of responses to artificial and natural selection (*e.g.*, WILHAM 1963; HAMILTON 1964; GRIFFING 1967; WADE 1977; FRANK 1998; WOLF *et al.* 1998). Social interactions, therefore, are a key factor in the design of artificial breeding programs in domestic species (DENISON *et al.* 2003; MUIR 2005) and for the outcome of evolutionary processes in natural populations (*e.g.*, HAMILTON 1964; QUELLER 1992; FRANK 1998; KELLER 1999; CLUTTON-BROCK 2002).

In agriculture, reduction of competitive behaviors is critical for improving animal well-being and productivity in confined high-intensity rearing conditions (CRAIG and MUIR 1996; KESTEMONT *et al.* 2003; MUIR 2005). Both theoretical and empirical work has shown that the relatedness among interacting individuals and the distribution of selection pressure over the individual and group levels are key factors for response to selection (GRIFFING 1967, 1976; CRAIG and MUIR 1996; MUIR 1996; BIJMA *et al.* 2007a). In evolutionary biology, the debate centers on the evolution of social behaviors such as altruism and cooperation and whether these can be explained by interactions among relatives and selection acting at multiple levels (HAMILTON 1964; MICHOD

1982; WADE 1978, 1985; FRANK 1998; WOLF *et al.* 1998; KELLER 1999).

In evolutionary biology, numerous theoretical models have been proposed for understanding the consequences of social interactions, and seemingly different models often appear to be equivalent formulations of the same process (KELLER 1999; LEHMANN and KELLER 2006; LEHMANN *et al.* 2007). There is an urgent need, however, for modeling approaches that can be applied empirically, so as to bring theory and observation into closer contact (LEIMAR and HAMMERSTEIN 2006, LEHMANN *et al.* 2007). Quantitative genetics has a strong tradition of combining theory and application (FALCONER and MACKAY 1996; LYNCH and WALSH 1998). In particular, the so-called animal model, combined with maximum-likelihood methodology, has proven to be a powerful and flexible tool for genetic analysis of complex traits in real populations (PATTERSON and THOMPSON 1971; HENDERSON 1975; SORENSON and KENNEDY 1986; LYNCH and WALSH 1998 and references therein; KRUUK 2004).

MUIR and SCHINKEL (2002) extended the animal model to analyze socially affected traits. Subsequent work, however, suggested that genetic parameters of social effects are difficult to estimate (ARANGO *et al.* 2005; VAN VLECK and CASSADY 2005; VAN VLECK *et al.* 2007). Those studies presented results from different statistical models, often with nonsignificant and unexpected results, and did not clarify the implications of observed results for genetic theory and response to

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selection. As a consequence, the magnitude of heritable social effects and its consequences for response to selection are still largely unclear.

Recently, MUIR (2005) and BIJMA *et al.* (2007a,b) presented a quantitative genetic framework for the prediction of response to selection and for statistical analyses of traits affected by social interactions. Together with the work of ELLEN *et al.* (2007), this work combines classical and socially affected traits into a single quantitative genetic framework. By adding a level of individual-by-individual interaction to the classical variance components, BIJMA *et al.* (2007a) showed that social interactions among individuals generate an additional level of heritable variation. This additional heritable variation is not part of the observed phenotypic variance, meaning that socially affected traits may possess a heritable variance exceeding observed phenotypic variance. As a consequence, response of socially affected traits to selection can be very large compared to observed variability among individuals, at least in theory. BIJMA *et al.* (2007b) show that the quantitative genetic model for socially affected traits dictates which components to include in statistical models for analyzing real data, in particular for the nonheritable component of social effects. Application to mortality due to cannibalistic behavior in domestic chickens showed that heritable variance in mortality was two- to threefold greater than classical additive genetic variance (BIJMA *et al.* 2007b; ELLEN and BIJMA 2008).

At present, still very little is known of the genetic parameters underlying socially affected traits. Here we present estimated genetic parameters for direct and social genetic effects on growth rate, feed intake, back fat thickness, and muscle depth in domestic pigs (*Sus scrofa*). Our results show that social effects contribute the vast majority of heritable variance in growth rate and feed intake in this population.

THEORY

This section summarizes the quantitative genetic theory for traits affected by social interactions presented in BIJMA *et al.* (2007a), emphasizing the consequences for heritable variance. In classical quantitative genetics, observed trait values (P) are the sum of a heritable component (A , breeding value), and a nonheritable component (E , environment); $P = A + E$ (FALCONER and MACKAY 1996; LYNCH and WALSH 1998). When trait values of individuals are affected by interactions with others, this model needs to be expanded with social effects (DICKERSON 1947; WILHAM 1963; GRIFFING 1967; CHEVERUD 1984; WOLF *et al.* 1998). When interactions take place within groups of n individuals, the trait value of each individual may be modeled as the sum of a direct effect rooted in the individual itself and the summed social effects due its $n - 1$ group members. Both direct and social effects may be decomposed into a heritable

component, A , and a nonheritable component, E , so that the trait value of individual i is

$$P_i = A_{D,i} + E_{D,i} + \sum_{j \neq i}^{n-1} (A_{S,j} + E_{S,j}) \quad (1)$$

(GRIFFING 1967), in which $A_{D,i}$ is the heritable direct effect of individual i on its own trait value, $E_{D,i}$ is the corresponding nonheritable direct effect, $A_{S,j}$ is the heritable social effect of group member j on the trait value of i , $E_{S,j}$ is the corresponding nonheritable social effect, and $\sum_{j \neq i}^{n-1}$ represents the sum taken over the $n - 1$ group members of i . Henceforth, we refer to A_D and A_S as direct and social breeding values (DBV and SBV). It follows from Equation 1 that the phenotypic variance equals

$$\sigma_P^2 = \sigma_{A_D}^2 + \sigma_{E_D}^2 + (n-1)(\sigma_{A_S}^2 + \sigma_{E_S}^2) + (n-1)r[2\sigma_{A_{DS}} + (n-2)\sigma_{A_S}^2] \quad (2)$$

(ARANGO *et al.* 2005; see the APPENDIX for derivation), in which σ^2 denotes variance, $\sigma_{A_{DS}}$ the covariance between direct and social breeding values of individuals, and r the mean additive genetic relatedness among group members (the r is twice the mean pairwise coefficient of coancestry between group members; LYNCH and WALSH 1998).

Because each individual interacts with $n - 1$ others, the total heritable impact of an individual on the mean trait value of the population is the sum of the individual's DBV and $n - 1$ times its SBV. BIJMA *et al.* (2007a), therefore, defined the total breeding value (TBV),

$$TBV_i = A_{D,i} + (n-1)A_{S,i}. \quad (3)$$

The TBV is a generalization of the usual breeding value, to account for heritable social effects on trait values. Analogous to classical theory, response to selection equals the change in mean TBV per generation, $\Delta \bar{P} = \Delta \overline{TBV} = \Delta \bar{A}_D + (n-1)\Delta \bar{A}_S$. The $\Delta \bar{A}_D$ represents the usual response to selection, whereas the $(n-1)\Delta \bar{A}_S$ represents the response originating from the change in mean social environment that individuals experience.

In classical theory, heritable variance in trait value is the variance of breeding values among individuals. Analogously, for socially affected traits, heritable variance is the variance of TBVs among individuals (BIJMA *et al.* 2007a),

$$\sigma_{TBV}^2 = \sigma_{A_D}^2 + 2(n-1)\sigma_{A_{DS}} + (n-1)^2\sigma_{A_S}^2. \quad (4)$$

In Equation 4, $\sigma_{A_D}^2$ represents the usual additive genetic variance, whereas the $2(n-1)\sigma_{A_{DS}} + (n-1)^2\sigma_{A_S}^2$ represents the additional heritable variance due to social effects. Equation 4 shows that heritable social effects may substantially increase heritable variance, in particular with large groups. (Although σ_{TBV}^2 may be smaller than $\sigma_{A_D}^2$ when $\sigma_{A_{DS}}$ is strongly negative.) Increased heritable variance translates directly into increased

potential for response to selection. ELLEN *et al.* (2007) show that response to selection equals $\Delta\bar{P} = \iota\rho\sigma_{TBV}$, in which ι represents the intensity of selection and ρ the correlation between the selection criterion and the TBV of individuals. This expression is fully analogous to the classical expression for response, $\Delta\bar{P} = \iota\rho\sigma_A$, in which ρ represents the correlation between the selection criterion and the classical breeding value (FALCONER and MACKAY 1996). Thus the σ_{TBV}^2 truly represents the potential of a trait to respond to selection.

In classical theory, an individual's breeding value (A) is a component of its trait value, $P_i = A_i + E_i$. As a consequence, heritable variance is smaller than phenotypic variance, and heritability is smaller than one. With heritable social effects, however, an individual's TBV is not a component of its trait value; $P_i \neq TBV_i + E_i$. The trait value of an individual contains social components originating from others (Equation 1), whereas the TBV consists entirely of heritable effects originating from the individual itself (Equation 3). Because the TBV is not a component of the trait value, phenotypic variance does not present an upper limit for heritable variance. With socially affected traits, therefore, heritable variance may exceed phenotypic variance. For example, if direct and social effects are independent and of equal magnitude, heritable effects account for half of the phenotypic variance, and groups are composed of four unrelated individuals, then heritable variance is 125% of phenotypic variance. (For example, $\sigma_{A_{DS}} = \sigma_{E_{DS}} = 0$, $\sigma_{A_D}^2 = \sigma_{E_D}^2 = 1$, $\sigma_{A_S}^2 = \sigma_{E_S}^2 = 1$, $n = 4$, and $r = 0 \rightarrow \sigma_{TBV}^2 = 10$ and $\sigma_P^2 = 8$.) This example illustrates that social effects create hidden heritable variance. Part of the heritable variance is hidden, because the TBV of an individual is spread across trait values of n distinct individuals and does, therefore, not surface in phenotypic variance.

To express heritable variance relative to phenotypic variance, we introduce

$$T^2 = \sigma_{TBV}^2 / \sigma_P^2, \quad (5)$$

which is an analogy of $h^2 = \sigma_A^2 / \sigma_P^2$, although T^2 may exceed one. Note that T^2 is not a true heritability, but represents heritable variance expressed on the scale of phenotypic variance among individuals. Comparison of T^2 and classical $h^2 = \sigma_A^2 / \sigma_P^2$ allows quick judgment of the contribution of social effects to heritable variance. For example, with $h^2 = 0.3$ and $T^2 = 0.6$, total heritable variance is two times greater than classical (direct) additive genetic variance, meaning that social effects contribute 50%. In the following, we describe the estimation of $\sigma_{A_D}^2$, $\sigma_{A_S}^2$, $\sigma_{A_{DS}}^2$, and σ_{TBV}^2 in a population of domestic pigs.

MATERIALS

Data originated from the experimental farm of the Institute for Pig Genetics, located in Beilen, the

Netherlands. This is a farrow-to-finish farm of 170 cross-bred sows and a rotational use of six sire lines in a 3-week system, with direct comparison of alternating combinations of two sire lines at any time. Five sow crosses were used as dams of the finishing pigs; two sow crosses were present at any time. To disentangle the common environment among litter mates due to the biological mother from that due to the foster mother, at least 25% of the live born piglets of each sows were cross-fostered during the weaning period (*i.e.*, before the start of the finishing period).

Data consisted of records on 14,032 finishing pigs, descending from 397 sires and 580 dams. Table 1 shows the distribution of observations (animals with slaughter record), number of sires, and number of dams over the different combinations of sire lines and sow crosses. Pens consisted of 6–12 animals of the same gender (male, female, or castrate). The penning strategy aimed at reducing variation in penning weight within pens. Due to the working method on the farm, the probability of penning litter mates together was higher than that of penning at random. As a consequence, average relatedness within pen was 0.18, ranging from 0.01 to 0.51. Relatedness was calculated using three generations of pedigree.

About one-third of the finishing pigs of each cross were fed *ad libitum*, using IVOG (INSENTEC, Marknesse, The Netherlands) feeding stations to record feed intake (Table 2). With *ad libitum* feeding, average daily eating time was ~ 1 hr per individual. Because maximum pen size was 12 individuals, feeding stations were vacant at least 50% of the day and thus available for (submissive) animals. The remaining two-thirds of the finishing pigs were fed restricted at a group level during the entire finishing period. Individual feed intake was unknown for restricted fed animals. On a pen level, restricted feed intake was $\sim 90\%$ of feed intake in *ad libitum* fed pens. For restricted fed animals, the amount of feed per pen was transported once a day to the dry feeders, which took ~ 4 hr for the entire farm and started at 8:00 AM. Per pen, only one animal at a time could use the dry feeder. A nipple drinker was mounted over the feeding pan of the dry feeder.

All finishers were weighed individually at the start of the finishing period at ~ 27 kg. At slaughter, hot carcass weight was recorded along with back fat thickness and muscle depth using the Hennessy grading probe. Four traits were analyzed: growth rate (grams per day), back fat thickness (millimeters), muscle depth (millimeters), and feed intake (grams per day). Growth rate was calculated as calculated live weight minus penning weight, divided by the length of the finishing period. Live weight was calculated as $1.3 \times \text{Hot Carcass Weight} - 0.0025 \times \text{Hot Carcass Weight}^2 + 0.2075 \times \text{Hot Carcass Weight}$ (HANDBOEK VARKENSHOUDERIJ 2004). Feed intake was calculated as cumulative feed intake during the finishing period, divided by the length of the finishing

TABLE 1

Number of individuals with observations on slaughter traits and number of sires and dams for each combination of sire line and dam cross

Sire line	Sow cross					All
	A	B	C	D	E	
F						
Individuals	305	55	310	18	122	810
Sires	13	7	17	5	10	32
Dams	31	14	33	7	36	121
G						
Individuals	453	341	161	—	—	955
Sires	30	16	12	—	—	37
Dams	47	43	20	—	—	110
H						
Individuals	1,417	1,001	353	125	1,031	3,927
Sires	80	50	24	22	41	117
Dams	121	92	39	17	102	371
I						
Individuals	1,042	1,012	583	113	579	3,329
Sires	61	38	33	18	23	91
Dams	84	101	52	21	77	335
J						
Individuals	924	1,147	222	135	1,024	3,452
Sires	49	43	15	18	46	97
Dams	78	101	28	21	102	330
K						
Individuals	232	579	261	83	404	1,559
Sires	14	12	11	9	9	23
Dams	27	78	34	13	50	202
All						
Individuals	4,373	4,135	1,890	474	3,160	14,032
Sires	247	166	112	72	129	397
Dams	153	145	88	37	157	580

period. Table 2 shows the number of observations and means for all traits. *Ad libitum* fed animals had a higher growth rate and a somewhat higher back fat thickness and muscle depth than restricted fed animals. The number of animals with slaughter records was ~15% lower than the number of penned animals, mainly due to loss of information, such as ear tags.

METHODS

Genetic parameters were estimated using residual maximum likelihood (ReML) with an animal model (PATTERSON and THOMPSON 1971; HENDERSON 1975; LYNCH and WALSH 1998; KRUIK 2004). Three models were compared: first, the classical animal model (model 1); second, the classical animal model extended with nonheritable social effects of pen mates (model 2); and third, the classical animal model extended with both heritable and nonheritable social effects of pen mates (model 3).

Model 1 was

TABLE 2

Number of observations and means of traits per feeding strategy

	Feeding strategy		
	Restricted ^a	<i>Ad libitum</i>	All
No. of animals penned	11,469	4,965	16,434
Penning weight (kg)	27.7	27.2	27.6
No. of animals with slaughter records	9,541	4,491	14,032
Hot carcass weight (kg)	86.3	88.9	87.1
Growth rate (g/day)	823	881	841
Back fat thickness (mm)	16.6	17.6	16.9
Muscle depth (mm)	57.2	58.6	57.6
No. of animals with individual feed intake	0 ^b	4,342	4,342
Feed intake (g/day)	— ^b	2,141	2,141

^a The amount of feed was restricted per pen.

^b Individual feed intake of restricted fed animals was unknown.

$$\mathbf{y} = \mathbf{Xb} + \mathbf{Za} + \mathbf{Wc} + \mathbf{e}, \quad (6)$$

in which $\mathbf{y}$ is the vector of observations; $\mathbf{X}$, $\mathbf{Z}$, and $\mathbf{W}$ are known incidence matrices; $\mathbf{b}$ is a vector of so-called fixed effects, which account for systematic nongenetic differences between groups of individuals (see below); $\mathbf{a}$ is a vector of random additive genetic effects (breeding values), which were assumed to follow a normal distribution, $\mathbf{a} \sim N(\mathbf{0}, \mathbf{A}\sigma_a^2)$; $\mathbf{c}$ is a vector of random nongenetic effects common to individuals born in the same litter, with $\mathbf{c} \sim N(\mathbf{0}, \mathbf{I}_c\sigma_c^2)$; and $\mathbf{e}$ is a vector of residuals, with $\mathbf{e} \sim N(\mathbf{0}, \mathbf{I}_e\sigma_e^2)$. The $\mathbf{I}_c$ and $\mathbf{I}_e$ are identity matrices of the appropriate dimensions, and $\mathbf{A}$ is a matrix of additive genetic relationships among all individuals (e.g., LYNCH and WALSH 1998). Common litter effects are routinely included in genetic analyses of pig data, to account for nongenetic covariances between full sibs due to the shared maternal environment.

The fixed effects depended on the trait analyzed. For growth rate, $\mathbf{b}$ included effects of the number of pen mates, the gender of the individual (male, female, or castrate), the combination of sire line by sow cross of the parents of the animal (1..28), the feeding strategy (restricted or *ad libitum*), and the compartment in which the pen was located (1..18). In addition, $\mathbf{b}$ included a linear regression on hot carcass weight in the analyses of back fat thickness and muscle depth and a linear regression on body weight at penning in the analysis of feed intake. In pig breeding, it is common practice to adjust back fat thickness and muscle depth for carcass weight and to adjust feed intake for penning weight when known.

Model 2 accounted for nonheritable social effects ($\sigma_{E_s}^2 > 0$). Nonheritable social effects create a nongenetic covariance among pen mates equal to $\text{Cov}_{\text{penmates}} = 2\sigma_{E_{Ds}} + (n-2)\sigma_{E_s}^2$ (BIJMA *et al.* 2007b).

Bijma *et al.* (2007b) showed that even a small non-genetic covariance among pen mates may substantially bias the estimated genetic parameters, illustrating the need to account for such covariance in the statistical analysis. To account for this covariance, Bijma *et al.* (2007b) fitted a correlation between residuals of group members, which is the general solution allowing any $\text{Cov}_{\text{penmates}}$. However, when $\text{Cov}_{\text{penmates}}$ is positive, which is likely unless n is small, an equivalent but simpler solution is to fit random pen effects rather than correlated residuals within pens. It follows from the general statistical result that “covariance within groups equals variance among group means,” that the variance of the random group effect equals $\text{Var}_{\text{groupmeans}} = \text{Cov}_{\text{penmates}} = 2\sigma_{E_{\text{DS}}}^2 + (n-2)\sigma_{E_{\text{S}}}^2$. (Our simulated data confirmed equivalence of both models as long as $\text{Cov}_{\text{penmates}} \geq 0$, results not shown.) Preliminary analyses confirmed that $\text{Cov}_{\text{penmates}}$ was positive for all traits in our data. We, therefore, fitted a random group effect, which converged easier and took less computing time than fitting correlated residuals. (Note, we use “group” to refer to the animals in the same pen.) Thus model 2 was

$$\mathbf{y} = \mathbf{Xb} + \mathbf{Za} + \mathbf{Wc} + \mathbf{Vg} + \mathbf{e}, \quad (7)$$

in which $\mathbf{V}$ is a known incidence matrix for groups and $\mathbf{g}$ a vector of random group effects, with $\mathbf{g} \sim N(\mathbf{0}, \mathbf{I}_g \sigma_g^2)$. Other elements were the same as in model 1.

Model 3 accounted for both heritable and nonheritable social effects,

$$\mathbf{y} = \mathbf{Xb} + \mathbf{Z}_D \mathbf{a}_D + \mathbf{Z}_S \mathbf{a}_S + \mathbf{Wc} + \mathbf{Vg} + \mathbf{e}, \quad (8)$$

in which $\mathbf{Z}_D$ and $\mathbf{Z}_S$ are known incidence matrices for direct and social genetic effects, and $\mathbf{a}_D$ and $\mathbf{a}_S$ are vectors of random direct and social genetic effects, with

$$\begin{bmatrix} \mathbf{a}_D \\ \mathbf{a}_S \end{bmatrix} \sim \text{MVN}(\mathbf{0}, \mathbf{C} \otimes \mathbf{A}),$$

in which

$$\mathbf{C} = \begin{bmatrix} \sigma_{A_D}^2 & \sigma_{A_{DS}} \\ \sigma_{A_{DS}} & \sigma_{A_S}^2 \end{bmatrix}$$

and $\otimes$ indicates the Kronecker product of matrices. Other elements were the same as in model 2. In model 3, the $\mathbf{Z}_S \mathbf{a}_S$ accounts for heritable social effects, whereas the $\mathbf{Vg}$ accounts for the nonheritable social effects. The $\mathbf{Z}_D$ -matrix in model 3 is identical to the $\mathbf{Z}$ -matrix in models 1 and 2; in model 3, we included the subscript D to emphasize the difference from $\mathbf{Z}_S$.

All models were fitted using ReML as implemented in the ASREML software (GILMOUR *et al.* 2002). Traits were analyzed univariately. Thus four separate analyses were done, one for each trait. All penned animals were included in the analyses, even when their slaughter

records were missing. Because maximum group size was 12 animals, the design matrix $\mathbf{Z}_S$ had 11 columns, one for each group member. For groups <12 animals, $\mathbf{Z}_S$ contained a 1 for each of the n group members, while the remaining $(12 - n)$ elements of $\mathbf{Z}_S$ were set to missing. The matrix of additive genetic relationships, $\mathbf{A}$, was calculated using information on three generations of pedigree. A total of 19,674 animals were included in the pedigree. Animals in the pedigree originated from 13 genetic groups, each representing a particular boar or sow line. To account for a possible effect due to genetic groups, groups were accounted for in the calculation of the $\mathbf{A}$ -matrix (THOMPSON 1979).

Validation focused on growth rate. To validate our results, we performed three additional analyses. First, we extensively tested alternative models, so as to identify nongenetic factors confounded with heritable social effects, thus causing false positive results (see the APPENDIX). Second, we evaluated the predictive ability of estimated classical breeding values *vs.* estimated direct and social breeding values. For this purpose, the observation on growth rate of every 10th animal was omitted from the data, but the animal remained in the pedigree file. Next, ASREML was used to estimate either classical breeding values or direct and social breeding values for all animals, including those whose records had been set to missing. Subsequently, values of the records set to missing were predicted using the estimated fixed effects and either the estimated classical breeding values or the estimated direct and social breeding values. Analysis of variance was used to evaluate the predictive ability of the estimated classical breeding values *vs.* the estimated direct and social breeding values (using PROC GLM of SAS). Third, we used independent data on 13,168 individuals of a different population descending from the same genetic lines, collected on a different farm, to obtain an independent estimate of the genetic parameters. These data did not overlap with the data described above, but contained information on growth rate only; independent data on the other traits were not available. Pen size was 10 animals. Data were analyzed using model 3.

RESULTS

For all traits, heritabilities from model 1 were in line with the literature, although the estimate for feed intake was in the upper range (Table 3; CLUTTER and BRASCAMP 1998). For growth rate and feed intake, a likelihood-ratio test strongly favored model 2 over model 1 ($P \ll 0.001$). Results from model 2 revealed a substantial variance of the pen effect for growth rate and feed intake, whereas estimates for back fat thickness and muscle depth were small (Table 4). Pen effects contributed 27% of phenotypic variance in growth rate and even 42% of phenotypic variance in feed intake. Inclusion of pen effects

TABLE 3
Estimates from the classical approach

Trait	$\hat{\sigma}_A^2$	$\hat{\sigma}_c^2$	$\hat{\sigma}_e^2$	$\hat{\sigma}_P^2$	$\hat{h}^2$
Growth rate (g/day)	2,583 $\pm$ 249	868 $\pm$ 70	3,820 $\pm$ 141	7,272 $\pm$ 133	0.36 $\pm$ 0.03
Back fat thickness (mm)	2.83 $\pm$ 0.23	0.28 $\pm$ 0.05	4.67 $\pm$ 0.14	7.78 $\pm$ 0.13	0.36 $\pm$ 0.03
Muscle depth (mm)	7.94 $\pm$ 0.76	1.09 $\pm$ 0.21	23.07 $\pm$ 0.52	32.10 $\pm$ 0.48	0.25 $\pm$ 0.02
Feed intake (g/day)	41,275 $\pm$ 3,384	15,201 $\pm$ 2,019	39,749 $\pm$ 6,050	96,226 $\pm$ 2,982	0.41 $\pm$ 0.04

Estimates were obtained using model 1 (Equation 6); $\pm$ indicates standard errors of estimates.

reduced estimated genetic, common litter, and residual variances (Table 4 *vs.* Table 3). As a result, heritability dropped from 0.36 to 0.25 for growth rate and from 0.41 to 0.18 for feed intake. This shift indicates a partial confounding of pen and pedigree, which agrees with the above-average relatedness among pen mates (see MATERIALS). Due to the relatedness among pen mates, covariances among pen mates are fitted as heritable variance when pen effects are omitted from the model.

Accounting for pen effects is not common in pig breeding, because physical differences among pens are usually minor and pen number is often not recorded. Also in our data, physical differences among pens were negligible, apart from effects accounted for in the model such as restricted *vs.* *ad libitum* feeding. The pen effects, therefore, seemed to originate from the individuals within the pen, rather than from external factors, suggesting substantial social effects. Thus our results suggest that including pen effects in the model may be essential to avoid biased estimates of genetic parameters, even when pens are fully standardized.

For growth rate and feed intake, a likelihood-ratio test with 2 d.f. strongly favored model 3 over model 2 ($P \ll 0.001$). Model 3 yielded highly significant social genetic variances for growth rate and especially for feed intake, whereas estimates for back fat thickness and muscle depth were small and nonsignificant (Table 5). Estimated direct genetic variances were little affected by including heritable social effects in the model. When judged by their absolute values, estimates of σ_{As}^2 for growth rate and feed intake may seem small. However, because an individual's SBV affects each of its $n - 1$ pen mates, small absolute values of σ_{As}^2 may still contribute

substantially to heritable variance (σ_{TBV}^2 , Equation 4). Heritable variance was 71% of phenotypic variance for growth rate and 70% of phenotypic variance for feed intake (T^2 , Table 5). These values are well outside the usual range of heritabilities for those traits (CLUTTER and BRASCAMP 1998), indicating that social effects create additional heritable variance. Comparing Tables 4 and 5 shows that heritable variance expressed relative to phenotypic variance, $T^2 = \sigma_{TBV}^2 / \sigma_P^2$, was almost threefold greater than classical heritability for growth rate and almost fourfold greater than classical heritability for feed intake. These results show that social effects contribute the vast majority of heritable variance in growth rate and feed intake in this population. The standard error of T^2 for feed intake was large compared to that for other traits. This is due to the smaller number of observations and the large contribution of social genetic effects, which were estimated with lower precision.

Estimated genetic correlations between direct and social genetic effects were positive but mostly nonsignificant (r_{Ads} , Table 5). This result suggests absence of conflict between self interest and interest of others, indicating that heritable interactions were not competitive, but rather neutral or slightly cooperative. When this is the case, classical mass selection for growth rate or feed intake would not increase competition among animals.

Including heritable social effects reduced estimated pen effects (Table 4 *vs.* Table 5). As argued above, pen effects seemed to originate from social interactions among individuals, rather than from physical differences among pens. In Table 4, pen effects originate from both heritable and nonheritable social effects,

TABLE 4
Estimates from the classical approach including random pen effects

Trait	$\hat{\sigma}_A^2$	$\hat{\sigma}_c^2$	$\hat{\sigma}_g^2$	$\hat{\sigma}_e^2$	$\hat{\sigma}_P^2$	$\hat{h}^2$
Growth rate (g/day)	1,780 $\pm$ 172	259 $\pm$ 43	1,929 $\pm$ 90	3,057 $\pm$ 101	7,023 $\pm$ 122	0.25 $\pm$ 0.02
Back fat thickness (mm)	2.79 $\pm$ 0.23	0.18 $\pm$ 0.05	0.44 $\pm$ 0.05	4.37 $\pm$ 0.14	7.78 $\pm$ 0.13	0.36 $\pm$ 0.02
Muscle depth (mm)	7.69 $\pm$ 0.74	0.86 $\pm$ 0.21	1.03 $\pm$ 0.18	22.44 $\pm$ 0.51	32.02 $\pm$ 0.47	0.24 $\pm$ 0.02
Feed intake (g/day)	17,678 $\pm$ 3,244	2,689 $\pm$ 1,092	41,018 $\pm$ 3,346	35,780 $\pm$ 1,986	97,165 $\pm$ 3,573	0.18 $\pm$ 0.03

Estimates were obtained using model 2 (Equation 7); $\pm$ indicates standard errors of estimates.

TABLE 5
Estimates when including heritable social effects

Trait	$\sigma_{A_0}^2$	$\sigma_{A_s}^2$	$\sigma_{A_{hs}}^2$	σ_{TBV}^2	σ_c^2	σ_g^2	σ_e^2	$\sigma_p^{2,b}$	$\hat{\tau}^2$	$\hat{\tau}_{A_{hs}}^e$
Growth rate (g/day)	1,522 ± 157	51 ± 9	56 ± 27	5,208 ± 669	236 ± 42	1,371 ± 102	3,221 ± 96	7,324 ± 162	0.71 ± 0.08	0.20 ± 0.10
Back fat thickness (mm)	2,75 ± 0.23	0.009 ± 0.004	-0.003 ± 0.023	3.19 ± 0.38	0.16 ± 0.05	0.38 ± 0.06	4.36 ± 0.15	7.79 ± 0.13	0.41 ± 0.04	-0.02 ± 0.15
Muscle depth (mm)	6.68 ± 0.51	0.027 ± 0.015	0.14 ± 0.07	10.35 ± 1.33	0.85 ± 0.21	0.66 ± 0.28	23.13 ± 0.53	32.17 ± 0.49	0.32 ± 0.04	0.33 ± 0.21
Feed intake (g/day)	16,950 ± 3,247	596 ± 240	1,215 ± 695	68,687 ± 17,990	2,620 ± 1,087	28,909 ± 4,043	37,080 ± 1,918	98,535 ± 4,096	0.70 ± 0.17	0.38 ± 0.22
Growth rate, restricted ^c	1,608 ± 188	71 ± 13	59 ± 35	6,466 ± 642	203 ± 57	1,131 ± 125	3,343 ± 120	7,593 ± 195	0.85 ± 0.11	0.18 ± 0.11
Growth rate, <i>ad libitum</i> ^d	1,784 ± 271	62 ± 16	51 ± 51	6,014 ± 1,141	209 ± 83	1,104 ± 215	2,617 ± 163	6,854 ± 214	0.88 ± 0.22	0.15 ± 0.14

Estimates were obtained using model 3 (Equation 8); ± indicates standard errors of estimates.

^aFrom Equation 4 with pen size (n) of 8.5.

^bFrom an extension of Equation 2, $\sigma_p^2 = \sigma_{A_0}^2 + \sigma_{L_0}^2 + (n-1)(\sigma_{A_s}^2 + \sigma_{E_s}^2) + (n-1)r[2\sigma_{A_{hs}} + (n-2)\sigma_{A_s}^2] + \sigma_c^2 + \sigma_g^2$, with pen size (n) of 8.5 and average relatedness within pens (r) of 0.18.

^cSubset of data with restricted fed animals only, 9541 observations.

^dSubset of data with *ad libitum* fed animals only, 4491 observations.

^eEstimated genetic correlation between direct and social effects.

whereas in Table 5 heritable social effects are included in $\sigma_{A_s}^2$ and $\sigma_{A_{hs}}^2$, thus reducing estimated pen effects.

In the statistical analyses of growth rate, feeding strategy was included as a fixed effect, which accounts for differences in mean growth rate between both treatments. However, a different feeding strategy may create differences not only in mean but also in variance. We, therefore, split the data into two subsets, one for each feeding strategy, and analyzed both subsets separately (last two rows in Table 5). Residual variance for growth rate differed significantly between feeding strategies, being largest with restricted feeding. Apparently, competition for limited resources in restricted fed pens increases differences in growth rate among individuals.

Analyses always converged to the same results, irrespective of starting values used in ASREML (unless starting values were so extreme that convergence failed totally). Details on model comparisons are in the APPENDIX; here we summarize main results. In mammals, confounding of genetic and environmental effects occurs mostly via the dam. We, therefore, fitted a sire model, so that information on genetic parameters comes entirely via paternal relationships (LYNCH and WALSH 1998). Compared to Table 5, the sire model yielded similar direct genetic variance and higher social genetic variance in growth rate (66 *vs.* 51). The full data used for Table 5 were a mix of individuals descending from different sire and dam lines (see MATERIALS). To investigate a potential bias due to this mixture of lines, we analyzed the subset of individuals descending from the single largest sire line (line H, Table 1). Compared to Table 5, this analysis yielded a slightly higher social genetic variance in growth rate (71 *vs.* 51). In the full data, pen size varied from 6 through 12. To investigate a potential effect of varying pen size, we analyzed the data subset for the most frequent pen size ($n = 8$). Compared to Table 5, this analysis yielded a higher social genetic variance (73 *vs.* 51). In summary, all models investigated yielded a highly significant social genetic variance in growth rate, mostly close to that in Table 5, but occasionally higher.

Analysis of variance was used to evaluate the ability of the estimated classical breeding values *vs.* estimated direct and social breeding values to predict missing records (see METHODS). When estimated DBV and SBV were not included in the model, estimated classical breeding values were highly significant ($P < 0.0001$). This result shows that estimated classical breeding values were meaningful in the absence of estimated DBV and SBV. However, when fitting both estimated classical breeding values and estimated DBV and SBV, classical breeding values were no longer significant ($P = 0.09$), whereas estimated DBV and SBV were highly significant ($P = 0.0018$ for DBV; $P < 0.0001$ for SBV). These results confirm significance of social genetic effects.

The analysis of independent data yielded the following results for growth rate: $\hat{\sigma}_{A_D}^2 = 1319 \pm 175$, $\hat{\sigma}_{A_S}^2 = 41 \pm 7$, $\hat{\sigma}_{A_{DS}} = -21 \pm 25$, $\hat{\sigma}_{TBV}^2 = 3294 \pm 492$, and $\hat{\sigma}_P^2 = 7012 \pm 134$, so that $\hat{h}_D^2 = \sigma_{A_D}^2 / \sigma_P^2 = 0.19$ and $\hat{T}^2 = 0.47 \pm 0.06$. This shows that T^2 was more than twofold greater than classical heritability, which is in line with Table 5.

DISCUSSION

Our results demonstrate that social effects may contribute the vast majority of heritable variation in some quantitative traits in mammals. Heritable variances in growth rate and feed intake were more than twofold greater than suggested by classical heritability. Estimated social genetic variances for growth rate and feed intake were highly significant, which was confirmed by extensive model comparison and independent data. Our results, therefore, demonstrate that heritable social effects are not merely of theoretical interest, but have significant biological relevance in a real population. Because response to selection is proportional to standard deviation in TBV, potential response in growth rate and feed intake in this population is substantially larger than suggested by classical heritability ($\Delta P = ip\sigma_{TBV}$; ELLEN *et al.* 2007). The increases in heritabilities found are in line with large responses to selection found by CRAIG and MUIR (1996).

Growth rate and feed intake are strongly influenced by social interactions while back fat thickness and muscle depth show only a small increase in heritability. Since carcass weight is part of the statistical model for back fat thickness and muscle depth, these traits describe carcass composition rather than quantity. Our results therefore indicate that carcass composition is little affected by social interactions.

Previous results: Few studies have reported genetic variance in social effects. BIJMA *et al.* (2007b) and ELLEN *et al.* (2008) reported significant social genetic variance in laying hens. ARANGO *et al.* (2005) attempted estimation of direct and social genetic parameters for growth rate in a population of 4946 female finishing pigs. However, due to the data structure in that study, accurate estimation of social genetic variance was impossible, resulting in a nearly flat likelihood and spurious convergence. VAN VLECK *et al.* (2007) estimated direct and social genetic effects for growth rate in a population of 1882 feed lot bulls. For most of their results, social genetic effects were nonsignificant, which is not surprising given their small data set of a few large pens. With few pens, it is difficult to discriminate between heritable and nonheritable effects, because heritable and nonheritable social genetic covariances among individuals are fully confounded within pen. CHEN *et al.* (2006) estimated genetic parameters of direct and social genetic effects for growth rate in a

population of 11,235 pigs, kept in pens of 15 individuals. Although the authors did not report significance levels, log-likelihood values presented in their results suggest significant social genetic variance. As in the present study, CHEN *et al.* (2006) observed a substantial increase in estimated direct heritability when group effects were omitted from the model, suggesting that their groups consisted partly of relatives.

The contribution of social effects to heritable variance is often misjudged. Both VAN VLECK *et al.* (2007) and CHEN *et al.* (2006) judged their estimated $\sigma_{A_S}^2$ as small, not realizing its substantial contribution to total heritable variance. When comparing total heritable variance calculated from their results to classical heritabilities, the following results are obtained: $T^2 = 1.42$ *vs.* $h_D^2 = 0.14$ (VAN VLECK *et al.* 2007, Table 3, period 1, models 1 and 5) and $T^2 = 0.58$ *vs.* $h_D^2 = 0.29$ (CHEN *et al.* 2006, Table 3, scheme 1a). Although those results were not always significant, such values are large rather than small.

Remarkably, some studies referred to social effects as “competitive effects,” even when estimated genetic correlations between direct and social genetic effects were positive (ARANGO *et al.* 2005; CHEN *et al.* 2006; VAN VLECK *et al.* 2007). A positive correlation, however, indicates that individuals with positive direct effects also have positive social effects on average, indicating mutual benefit rather than competition.

Estimability: VAN VLECK and CASSADY (2005) used simulated data to investigate estimability of social genetic effects. They showed that estimates depended heavily on whether or not group effects were included in the model and whether group effects were treated as fixed or random. Analyses without group effects yielded substantially overestimated social genetic variance, but the cause of this phenomenon was left unclear. In METHODS, we show that group effects take account of nonheritable social effects ($\sigma_{E_S}^2 > 0$). Our simulations confirmed that the expected between-group variance equals $2\sigma_{E_{DS}} + (n - 2)\sigma_{E_S}^2$, illustrating that nonheritable social effects translate into between-group variance (results not shown). Because heritabilities are rarely close to 100%, it is unlikely that social effects are fully heritable (*i.e.*, $\sigma_{A_S}^2 > 0$ while $\sigma_{E_S}^2 = 0$). Therefore, when analyzing social genetic effects, one should always account for a nonheritable component, either by allowing residuals to be correlated within groups or by including random group effects when n is large.

Because between-group variance originates from nonheritable social effects, which are random effects, group effects should ideally be fitted as random rather than fixed. VAN VLECK *et al.* (2007) and CHEN *et al.* (2006) observed that analysis with groups included as a fixed effect failed, which also occurred in our analysis. Our simulations, in contrast, showed that genetic parameters are identifiable and estimates are unbiased when groups are treated as fixed and are composed fully

at random with respect to family (results not shown). In our data, however, groups were partly composed of family members, which probably explains failure when including group as a fixed effect.

When group members are related, social genetic variance may not be identifiable. For example, the Appendix of BIJMA *et al.* (2007b) shows that the data structure used by WOLF (2003) prohibits identification of the social genetic variance. Estimation of social genetic effects seems most powerful when populations consist of many small groups of unrelated individuals, but more research on optimum designs is needed.

Animal breeding: Animal breeders can utilize heritable social effects to increase response in their selection programs. GRIFFING (1967, 1976) showed that breeding schemes need to be adapted to improve socially affected traits (see also MUIR 2005). One strategy to fully utilize heritable variance is to use groups composed of family members when recording phenotypic data. With this strategy, one may either keep selection candidates themselves in family groups, such as in classical group selection (*e.g.*, GRIFFING 1976), or keep relatives of selection candidates in family groups and select among candidates on the basis of performance of their relatives. For example, ELLEN *et al.* (2007) showed that accuracy of selection based on information of progeny kept in family groups has a maximum of 100%, whereas maximum accuracy was lower or even negative when progeny were kept in groups of unrelated individuals. Thus, animal breeders may use “artificial kin selection” to improve socially affected traits. Selection based on sib or progeny information does not require knowledge of direct and social genetic variances. When direct and social genetic variances are known, however, such as for the current pig population, selection on best linear unbiased predictions (BLUP) (HENDERSON 1975) of TBVs may yield a higher response or allow for different population structures. For example, MUIR (2005) used simulated data to show that selection using BLUP yielded higher responses than group selection with groups composed of full sibs.

Social behaviors may depend on environmental circumstances. With restricted feeding on a pen level, competition for a fixed total amount of feed creates negative correlations between individual intakes within pens, causing a negative correlation between direct and social effects. Unfortunately, individual feed intake was not recorded with restricted feeding. Because growth rate and feed intake are highly genetically correlated ($r_g = \sim 0.65$; CLUTTER and BRASCAMP 1998), one might expect restricted feeding to cause a negative genetic correlation between direct and social effects on growth rate. For both feeding strategies, however, genetic correlations were nonsignificant, suggesting that competition for finite resources on the phenotypic level does not necessarily translate to the genetic level.

Long-term selection: Classical traits not affected by social interactions often continue to respond to selection for many generations, indicating that selection does not exhaust heritable variance (*e.g.*, DUDLEY and LAMBERT 2004). It is unclear whether this observation extends to socially affected traits. Once social effects are on a “sufficient” level, further improvement of social behaviors may not translate into response in trait value. For example, once tree breeders would manage to breed individuals maintaining equal height, thus canceling competition for daylight, further increase in productivity by decreasing competition for daylight seems difficult. In quantitative genetic analysis, this phenomenon would surface as gradually decreasing social genetic variance. On the one hand, this would be undesirable because it reduces opportunities for further genetic improvement. On the other hand, however, a reduction in social genetic variance would increase uniformity of individuals, which is often desirable but has been difficult to achieve in livestock (SANCRISTOBAL-GAUDY *et al.* 1998; HILL and ZHANG 2004).

Social effects on kin: Hamilton proposed kin selection as a mechanism for the evolution of social behaviors (HAMILTON 1964). Kin selection may cause individuals to behave more cooperatively toward relatives compared to nonrelatives, because helping relatives has inclusive fitness benefits (*e.g.*, FRANK 1998). To investigate whether relatedness among individuals affected trait values in the current population, we included a linear regression of growth rate on mean additive genetic relatedness within pen in model 3 (Equation 8). The estimated regression coefficient equaled 29 g/day ($P < 0.10$), meaning that a pen of full sibs ($r = \frac{1}{2}$) shows 15 g/day higher growth rate than a pen of nonrelatives. The 15 g/day correspond to ~ 0.2 phenotypic standard deviation, indicating a moderate effect. We are currently investigating the origin of this effect. Including a regression on relatedness hardly affected estimated direct and social genetic parameters (results not shown).

Natural populations: For natural populations, collecting sufficient data involves substantial effort and often yields data structures that are difficult to analyze. Animal and plant breeders have developed flexible statistical tools, such as the so-called animal model, which may be useful for studying natural populations (*e.g.*, KRUIK 2004). Statistical methods presented in MUIR (2005) and BIJMA *et al.* (2007b) do not require balanced designs or specific family relationships within groups. Moreover, those methods enable estimating social genetic variance without the need to record the behavior *per se*. For example, as illustrated in this article, those methods allow estimating social genetic variance in growth rate without recording behavioral interactions among group members. Compared to behavioral studies, this represents a substantial saving of labor, which may be used to collect information on a larger

number of individuals, to enable quantitative genetic analysis of meaningful accuracy. Application of an animal model requires (i) knowledge of which individuals interact with each other (*i.e.*, the identification of groups), (ii) phenotypic records on the trait of interest, and (iii) additive genetic relatedness among all individuals. When pedigrees are unknown, either additive genetic relatedness may be estimated directly from molecular markers (LYNCH and RITLAND 1999; TORO *et al.* 2002; OLIEHOEK *et al.* 2006) or the pedigree may be reconstructed using molecular markers (BLOUIN 2003). When DNA samples are available on multiple generations, pedigree reconstruction provides information on the number of offspring of individuals, *i.e.*, on their fitness. Such information may be used to estimate total heritable variance in fitness, the contribution of social effects to total heritable variance, and the genetic correlation between direct and social genetic effects on fitness. Knowledge of this correlation would reveal the strength of heritable competition within species and the degree to which such competition constrains evolutionary success of the species.

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APPENDIX

Phenotypic variance: From Equation 1, $\text{Var}(P) = \text{Var}(A_{D,i} + E_{D,i} + \sum_{j \neq i}^{n-1} (A_{S,j} + E_{S,j}))$. With $\text{Cov}(A, E) = 0$ for all i, j , and $\text{Cov}(E_{D,i}, E_{S,j}) = 0$ when $i \neq j$, it follows that $\text{Var}(P) = \sigma_{A_D}^2 + \sigma_{E_D}^2 + \text{Var}(\sum_{j \neq i}^{n-1} A_{S,j}) + \text{Var}(\sum_{j \neq i}^{n-1} E_{S,j}) + 2 \text{Cov}(A_{D,i}, \sum_{j \neq i}^{n-1} A_{S,j})$. With $\text{Cov}(A_{S,j}, A_{S,j'}) = r_{jj'} \sigma_{A_S}^2$ in which $r_{jj'}$ is relatedness between individuals j and j' , it follows that $\text{Var}(\sum_{j \neq i}^{n-1} A_{S,j}) = (n-1)\sigma_{A_S}^2 + (n-1)(n-2)r\sigma_{A_S}^2$, r denoting mean relatedness within groups. Furthermore, with $\text{Cov}(E_{S,j}, E_{S,j'}) = 0$ when $j \neq j'$,

$\text{Var}(\sum_{j \neq i}^{n-1} E_{S,j}) = (n-1)\sigma_{E_S}^2$. Finally, $\text{Cov}(A_{D,i}, \sum_{j \neq i}^{n-1} A_{S,j}) = (n-1)r\sigma_{A_{DS}}$. Collecting terms yields $\sigma_P^2 = \sigma_{A_D}^2 + \sigma_{E_D}^2 + (n-1)(\sigma_{A_S}^2 + \sigma_{E_S}^2) + (n-1)r[2\sigma_{A_{DS}} + (n-2)\sigma_{A_S}^2]$, which is Equation 2.

Validation: To evaluate robustness of our estimates, we performed additional analyses for growth rate (Table A1). The “Basic” row in Table A1 corresponds to results in Table 5. We distinguished four possible sources of bias:

1. Due to imperfection of the statistical model, genetic and nongenetic effects might be confounded.
2. Our data were a mix of individuals from a large number of crosses (Table 1), which might affect results.
3. For part of our data, all animals within a pen were slaughtered on the same day, whereas for the remaining part of the data, delivery decisions were based on individual body weight. As a result, the interval between delivery of the first and the last animal within a pen lasted up to 4 weeks.
4. Pen size varied between 6 and 12 individuals, which may affect estimates.

Confounding: Early life experience may affect social behaviors later in life. As a consequence, individuals born in the same litter may show similar social effects, leading to nongenetic covariances between social effects of full sibs. When not accounted for in the statistical analyses, such effects would inflate estimated social genetic variance. To reduce this risk, we performed two additional analyses. First, we applied a sire model, so that covariances between pen mates of full

TABLE A1

Estimates ($\pm$ standard error) for growth rate from different models

Model	$\hat{\sigma}_{A_D}^2$	$\hat{\sigma}_{A_S}^2$	$\hat{\sigma}_{A_{DS}}$	$\hat{\sigma}_e^2$	$\hat{\sigma}_{\text{foster}}^2$	$\hat{\sigma}_{\text{common-social}}^2$	$\hat{\sigma}_g^2$	$\hat{\sigma}_e^2$
Basic	1522 $\pm$ 157	51 $\pm$ 9	56 $\pm$ 27	236 $\pm$ 42	—	—	1371 $\pm$ 102	3221 $\pm$ 96
Sire model ^a	1698 $\pm$ 267	66 $\pm$ 14	117 $\pm$ 43	573 $\pm$ 49	—	—	1625 $\pm$ 83	3947 $\pm$ 54
Basic including social common environment	1525 $\pm$ 157	49 $\pm$ 9	54 $\pm$ 27	229 $\pm$ 42	—	12 $\pm$ 6	1318 $\pm$ 104	3205 $\pm$ 96
Foster instead of common	1609 $\pm$ 151	50 $\pm$ 9	49 $\pm$ 27	—	290 $\pm$ 38	—	1377 $\pm$ 102	3096 $\pm$ 95
Both common and foster	1506 $\pm$ 155	49 $\pm$ 9	52 $\pm$ 27	106 $\pm$ 45	249 $\pm$ 41	—	1365 $\pm$ 102	3131 $\pm$ 95
Only 1 sire line, ♂H (3927 observations)	1653 $\pm$ 384	71 $\pm$ 21	34 $\pm$ 59	259 $\pm$ 118	—	—	1149 $\pm$ 210	3399 $\pm$ 229
Different error variances per sire line	1514 $\pm$ 156	50 $\pm$ 9	58 $\pm$ 27	238 $\pm$ 42	—	—	1374 $\pm$ 102	3212 ^b
Different error variances per cross	1531 $\pm$ 156	50 $\pm$ 9	56 $\pm$ 27	223 $\pm$ 41	—	—	1369 $\pm$ 102	3224 ^b
Delivery based on weight (8129 observations)	1648 $\pm$ 200	44 $\pm$ 10	37 $\pm$ 33	169 $\pm$ 48	—	—	1007 $\pm$ 114	2906 $\pm$ 122
Only eight-animal pens (5523 observations)	1684 $\pm$ 247	73 $\pm$ 18	67 $\pm$ 47	188 $\pm$ 84	—	—	932 $\pm$ 173	3189 $\pm$ 158

^aFor the sire model, genetic variance components were calculated as four times the corresponding sire component (LYNCH and WALSH 1998).

^bWeighed average.

sibs do not contribute to estimated social genetic variance. Results of the sire model strongly supported the presence of social genetic variance ($P < 0.001$, "Sire model," Table A1). Second, we fitted a nongenetic social effect common to individuals born in the same litter. For this purpose, we fitted the litter identification of an individual as a nongenetic effect in the records of its pen mates. This is an analogy of a classical common environmental effect, but in this case it refers to social effects rather than direct effects. Results showed a small but significant variance for the common social effect of litter mates ($P < 0.05$, Basic including social common environment row, Table A1), suggesting that early life experiences affect social behaviors. Estimated social genetic variance was not affected by including social common environment effects.

The basic model included a common environment among littermates. However, because at least 25% of individuals were cross-fostered, common environment due to the foster sow might affect estimates. Therefore, a model including common environment due to the foster sow, instead of the biological mother, was fitted ("Foster instead of common" row, Table A1) as well as a model including both effects ("Both common and foster" row, Table A1). Both effects were highly significant, but estimated genetic variance components were unaffected.

To account for a within-group nongenetic correlation, we included a random pen effect (see METHODS). We also evaluated an alternative model in which pens were treated as fixed effects, while omitting number of pen mates, gender, feeding strategy, and compartment, because of full confounding. This analysis converged, but variance components for social effects could not be estimated due to singularity (see DISCUSSION).

Multiple crosses: The full data consisted of crossbred individuals descending from multiple sire and dam lines. A sire model ignores existence of multiple dam

lines. Results of the sire model, therefore, indicate that significant social genetic variance was not an artifact caused by multiple dam lines underlying the observed data (Sire model row, Table A1). To further investigate the effect of multiple parental lines, we analyzed the subset of data originating from the largest cross using model 3 ($\text{♂H} \times \text{♀A}$; $n = 1417$; Table 1). However, this analysis did not converge, probably due to the small number of observations. We, therefore, analyzed the next best alternative, which was the subset of individuals descending from the single largest sire line, but from multiple dam lines ("Only 1 sire line" row, ♂H ; $n = 3927$, Table A1). Results confirmed the previous finding.

Analyses thus far assumed that residual variances did not depend on the cross. We investigated two alternatives allowing for heterogeneous residual variances, the first allowing for different residual variances per sire line ("Different error variances per sire line" row, Table A1) and the second allowing for different residual variances per cross ("Different error variances per cross" row, Table A1). Results were nearly identical to those in Table 5.

Delivery strategy: To investigate a potential effect due to delivery per pen *vs.* delivery based on individual weight, we analyzed the subset of data on individuals delivered on the basis of individual weight ("Delivery based on weight" row, Table A1). Estimated genetic parameters were in line with Table 5.

Pen size: To investigate a potential effect due to varying pen size, we analyzed the subset of data of the single most frequent pen size ("Only eight-animal pens" row, Table A1). Estimated social genetic variance was greater than in Table 5 (73 *vs.* 51), but the difference was not significant.

In conclusion, all of the above analyses strongly support presence of heritable variance due to social effects.