Maternal and social genetic effects on average daily gain of piglets from birth until weaning
A. C. Bouwman, R. Bergsma, N. Duijvesteijn and P. Bijma

doi: 10.2527/jas.2009-2494 originally published online May 21, 2010

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://jas.fass.org/content/88/9/2883
ABSTRACT: The aim of this study was to investigate whether there is heritable social variation in ADG from birth until weaning in piglets. Nursing and the establishment of teat order are sources of social interaction among suckling piglets nursed by the same sow. If a heritable social effect is present, but ignored, the selected animals might be the most competitive ones with negative effects on growth of their group mates, resulting in less response to selection than expected. The social interaction model was extended with a maternal component to estimate genetic maternal and social effects. Four different animal models were compared: a basic model with a direct heritable effect only; a social model accounting for direct and social heritable effects; a maternal model with a heritable maternal effect in addition to the basic model; and a social-maternal model accounting for direct, social, and maternal heritable effects. Estimates of direct, maternal, and social heritability were 0.07, 0.06, and around 0.0007 (not significantly different from zero, SE = 0.0005), respectively. Total heritable variance, including direct, social, and maternal heritable variance and their covariances ranged from 0.07 to 0.15 of the phenotypic variation. Both maternal models were significantly better than equivalent nonmaternal models ($P \leq 0.005$). The social model was not significantly better than the basic model ($P = 0.102$), and the social-maternal model was also not significantly better than the maternal model ($P = 0.486$). There was no evidence for heritable social effects among piglets in a group. The generally used maternal model fit the data as well as the social-maternal model. Sufficient cross-fostering is needed to partition social and maternal variation.

Key words: average daily gain, competition, genetic parameter, maternal effect, piglet, social interaction

INTRODUCTION

Performance of populations kept in groups may be affected by social interactions among individuals (Griffing, 1967; Muir, 1996). Social interactions can be due to competition for limited resources or due to social behavior and can result in reduced growth and production, or in injuries and death (Muir, 1996, 2005). Social genetic effects, if present, should be considered during selection to obtain the desired selection response (Muir, 2005; Bijma et al., 2007b). A genetic model to account for social interactions was first proposed by Griffing (1967) and included not only the direct genetic effect, but also social genetic effects of group mates. Van Vleck and Cassady (2005) showed with simulations that REML procedures are suitable for parameter estimation of social models. Using social models, social variation has been found for ADG and other traits in pigs (Arango et al., 2005; Bergsma et al., 2008; Chen et al., 2008, 2009; Hsu et al., 2010) and for survival days in chicken (Bijma et al., 2007a; Ellen et al., 2008).

There are also social interactions among piglets, possibly affecting ADG from birth until weaning. Milk production of the sow is potentially a limiting resource causing competition among piglets because the voluntary feed intake of piglets keeps increasing, whereas milk production of the sow reaches a rather constant level after 8 to 10 d (Harrell et al., 1993). Another potential source of interaction is the establishment of a teat order. Piglets tend to position themselves in a specific order at the teats during lactation (e.g., Donald, 1937; McBride, 1963). During the first hours after birth, piglets push and bite each other to capture a teat, but competition is reduced as soon as teat order has been established (Hartsock and Graves, 1976).

Although selection of pigs in practice is not directly on ADG of piglets, ADG is related to piglet vitality;
pigs benefit later in life from a good start (Mahan and Lepine, 1991). The aim of this study was to investigate whether there is heritable social variation in ADG from birth until weaning in piglets. For the genetic analyses, the social interaction model was extended with a maternal component.

**MATERIALS AND METHODS**

Animal Care and Use Committee approval was not obtained for this study because the data were obtained from an existing database.

**Materials**

Data for this study were obtained from the experimental farm of IPG, Institute for Pig Genetics, in Beilen, the Netherlands. The farm operates strictly according to Dutch regulations on protection of animals. The farm is farrow-to-finish with 180 crossbred sows, managed under a 3-wk batch farrowing system. Lines are rotated to compare alternating combinations of 2 sire lines and 2 sow lines at any time. Every 3 wk, one sire line is replaced, and each year one sow line is replaced.

Records were available from piglets weaned between September 2001 and December 2007. Stillborn piglets (7.0%) and piglets that died within 24 h after birth (7.3%) were discarded. The cross-fostering percentage at the experimental farm was 28.8%, which is greater than the average cross-fostering percentage on commercial farms of 8.6% (Straw et al., 1998). This was done to disentangle the common environment among litter mates due to the biological dam from that due to the foster dam. Piglets were cross-fostered within 24 h after birth. Cross-fostering was not done randomly. Mainly the smallest and biggest piglets per litter were transferred to reduce variation in piglet BW within groups. To reach the desired cross-fostering percentage (>25%), some additional randomly chosen piglets were cross-fostered.

When social interactions are considered, the group of animals that interact has to be defined. For fattening pigs, a group consists of pigs that are kept in the same pen. Considering ADG from birth until weaning, piglets nursed by the same dam during the same period was defined as a group of interacting piglets. Because of cross-fostering, most dams nursed mixed litters composed of their own piglets and fostered piglets. Thus, a group consisted of offspring born alive by the sow minus piglets cross-fostered to another sow plus piglets fostered by the sow minus her piglets that died within 24 h after birth. Not all groups were affected by cross-fostering; 8.7% of the groups consisted of the birth litter. In another 14% of the groups, piglets were fostered away from the birth litter but none were added to the group.

The sow can be the biological dam or the foster dam of the piglets, but all piglets in the group are nursed by the sow; therefore, the sow is referred to as group dam. All the piglets nursed by the same dam and still alive 24 h after birth were considered as group mates contributing to social interactions among piglets. Piglets that died between d 2 after birth and weaning were also considered to have contributed to social interactions because they were present during establishment of teat order.

To estimate the social genetic effect of an individual, the row in the design matrix corresponding to the ADG of an individual includes all its group mates, which relates effects of the group mates to the ADG of the individual. The social effect of an individual is based on the records of its group mates, not on its own record. If a piglet does not have an ADG record itself, it is thus possible to estimate the social effect this individual has on ADG of its group mates. Identification of all group mates, with or without individual ADG, was included for a record of an individual because interactions were among all group mates, not just those with measured ADG. Deleting individual records before assigning group mates to individual records would have led to fewer group mates than there really were, which is undesirable because there has been interaction with this deleted individual. Thus, only complete groups were deleted during further editing. Groups containing piglets with unreliable or missing pedigree were discarded. The main reasons for considering pedigrees unreliable were reinsemination of sows within approximately 18 to 24 h with sperm of a different boar than the one used for the first insemination and when DNA information (if available) disagreed with the pedigree. Groups containing boars from a boar taint experiment were discarded. Only groups of 7 through 14 piglets were considered, so that at least 10 groups per group size class were present in the data. From the original data set, 28% of the groups were discarded. Information from these piglets, stillborn piglets, and piglets that died within 24 h after birth was not included in the final data set (42% of all piglets).

After editing, there were 17,053 crossbred piglets in the data set, and 15,602 of these piglets had ADG records. Piglets were female and castrated males from 1,858 litters from matings of 542 sows and 435 boars. They were nursed in 1,604 groups by 528 group dams. Most group dams (75%) nursed 2 or more litters and offspring were assumed to be zero because none of the dams had ADG records or were group mates. Piglets were sometimes cross-fostered to groups originating from other lines. This means that piglets within a group can originate from different lines. Besides some tests with other lines, there were 5 main sire-lines and 3 main dam-lines (Table 1). The other lines appeared in the data set at low frequency and were therefore combined, together with piglets with unknown lines, into a separate line (XX, Table 1) of 1,361 piglets (~8%).
Creep feed was offered to the piglets, but intake was not recorded.

Individual BW of piglets were recorded within 24 h after birth and at weaning when also age at weaning was recorded. The ADG was calculated as \( \text{ADG (g/d)} = \frac{\text{(weaning weight (kg)} - \text{birth weight (kg)}) \times 1,000}{\text{age at weaning (d)}} \).

### Methods

The F-test from the GLM procedure (SAS Inst. Inc., Cary, NC) was used to determine which fixed effect should be included in the model \((P \leq 0.20)\). For all 4 models the fixed effects included an interaction between line (16 classes) and batch (105 classes; \(P < 0.001\)), parity of the biological dam \((P = 0.015)\) and the group dam \((P < 0.001\); parity 1 to 5 and \(\geq 6\)), line of the group dam (4 classes; \(P = 0.085\)), and group size (7 to 14; \(P < 0.001\)). Table 2 gives the distribution of data for some of these fixed effects. Lines of the group mates were included in the model as social fixed effects (using the “and” statement in ASReml) because multiple lines were present in a group due to cross-fostering over lines to account for social effects being different among lines. So, for example, a group was composed of 10 individuals, 3 from line A, 3 from line B, and 4 from line C. The ADG of an individual from line A in this group was corrected for 2 times line A, 3 times line B, and 4 times line C \((\gamma_i = 2 \times A + 3 \times B + 4 \times C)\). There was no significant \((P = 0.36)\) difference between the females and castrated males; therefore, sex effects were not included in the model.

Subsequently, ADG was analyzed with univariate mixed models. All models were fitted using the REML procedure in ASReml (Gilmour et al., 2006). The social interaction model with a random group effect (Arango et al., 2005; Bergsma et al., 2008; Chen et al., 2008) was used and extended with a maternal genetic component from the group dam. Initially the maternal models were analyzed, including the biological dam and the group dam as maternal genetic effects. However, these models did not converge at standard ASReml convergence criteria (Gilmour et al., 2006). Alternative convergence levels were not tested. Apparently, it was not possible to separate both genetic effects. Initial analyses of maternal models, including the genetic effect of either the biological dam or the group dam, showed that variation due to genetic effects of the group dams was larger than that of biological dams (results not shown), as expected because the group dam produces the milk necessary for growth of piglets. Therefore, the maternal effects were assumed to be due to group dams.

Four linear animal models were compared: a basic model \((1)\) with a direct heritable effect; a social model \((2)\) accounting for heritable social effects in addition to the basic model; a maternal model \((3)\) with the group dam as heritable maternal effect in addition to the basic model; and a social-maternal model \((4)\) accounting for direct, social, and maternal heritable effects. All 4 models included nongenetic effects due to a common prenatal environment from biological dam, a common postnatal environment from group dam and a permanent environment associated with the group dam. The models were

\[
\text{model 1}_{\text{basic}}: \quad y = Xb + Z_D a_D + Wl + V_g + U_p + e, \\
\text{model 2}_{\text{social}}: \quad y = Xb + Z_D a_D + Z_S a_S + Wl + V_g + U_p e = e,
\]

### Table 1. Number of piglets per sire and dam line

<table>
<thead>
<tr>
<th>Sire line</th>
<th>Dam line</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>F</td>
</tr>
<tr>
<td>A</td>
<td>309</td>
</tr>
<tr>
<td>B</td>
<td>731</td>
</tr>
<tr>
<td>C</td>
<td>980</td>
</tr>
<tr>
<td>D</td>
<td>484</td>
</tr>
<tr>
<td>E</td>
<td>1,105</td>
</tr>
<tr>
<td>X</td>
<td></td>
</tr>
</tbody>
</table>

\(1\) A to E are different sire lines; F to H are different dam crosses; XX are piglets from unknown lines or lines with low frequency in the data set.

### Table 2. Number of observations (n) per fixed effect class for some fixed effects

<table>
<thead>
<tr>
<th>Parity of biological dam</th>
<th>Parity of group dam</th>
<th>Group size</th>
<th>Line of group dam</th>
</tr>
</thead>
<tbody>
<tr>
<td>Class</td>
<td>n</td>
<td>Class</td>
<td>n</td>
</tr>
<tr>
<td>1</td>
<td>3,331</td>
<td>1</td>
<td>3,536</td>
</tr>
<tr>
<td>2</td>
<td>3,173</td>
<td>2</td>
<td>3,207</td>
</tr>
<tr>
<td>3</td>
<td>2,733</td>
<td>3</td>
<td>2,708</td>
</tr>
<tr>
<td>4</td>
<td>2,307</td>
<td>4</td>
<td>2,202</td>
</tr>
<tr>
<td>5</td>
<td>2,229</td>
<td>5</td>
<td>2,156</td>
</tr>
<tr>
<td>(\geq 6)</td>
<td>3,280</td>
<td>(\geq 6)</td>
<td>3,184</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

\(1\) F to H are different dam crosses, and X are unknown dam crosses or dam crosses with low frequency.
model 3_{maternal} : \begin{align*} 
\mathbf{y} &= \mathbf{Xb} + \mathbf{Z}_D \mathbf{a}_D + \mathbf{Z}_M \mathbf{a}_M \\
&\quad + \mathbf{Wl} + \mathbf{Vg} + \mathbf{Upe} + \mathbf{e}, \end{align*}

where \( \mathbf{y} \) is the vector of observations; \( \mathbf{X}, \mathbf{Z}_D, \mathbf{Z}_S, \mathbf{Z}_M, \mathbf{W}, \mathbf{V}, \mathbf{U} \) are known incidence matrices relating observations to fixed and random effects; \( \mathbf{b} \) is a vector of fixed effects; \( \mathbf{a}_D \) is a vector of random direct additive genetic effects; \( \mathbf{a}_S \) is a vector of random social additive genetic effects; \( \mathbf{a}_M \) is a vector of random maternal additive genetic effects; \( \mathbf{l} \) is a vector of random nongenetic effects common to individuals born in the same litter (common prenatal environment from biological dam), referred to as litter effect, with \( 1 - N(0, \sigma_l^2) \); \( \mathbf{g} \) is a vector of random nongenetic effects common to individuals nursed in the same group (common postnatal environment from group dam), referred to as group effect, with \( \mathbf{g} \sim N(0, \sigma_g^2) \); \( \mathbf{pe} \) is a vector of random nongenetic effects due to a permanent environment of the group dam with \( \mathbf{pe} \sim N(0, \sigma_{pe}^2) \); and \( \mathbf{e} \) is a vector of residuals with \( \mathbf{e} \sim N(0, \sigma_e^2) \). The augmented \( \mathbf{a}_D, \mathbf{a}_S, \) and \( \mathbf{a}_M \) vectors have a multivariate normal distribution with expected values of zero and covariance matrix \( \mathbf{C} \otimes \mathbf{A} \), where

\[
\mathbf{C} = \begin{bmatrix} 
\sigma_{\mathbf{a}_D}^2 & \sigma_{\mathbf{a}_D\mathbf{a}_S} & \sigma_{\mathbf{a}_D\mathbf{a}_M} \\
\sigma_{\mathbf{a}_D\mathbf{a}_S} & \sigma_{\mathbf{a}_S}^2 & \sigma_{\mathbf{a}_S\mathbf{a}_M} \\
\sigma_{\mathbf{a}_D\mathbf{a}_M} & \sigma_{\mathbf{a}_S\mathbf{a}_M} & \sigma_{\mathbf{a}_M}^2 
\end{bmatrix},
\]

\( \otimes \) indicates the Kronecker product of \( \mathbf{C} \) and \( \mathbf{A} \), the \( \mathbf{I} \) are appropriate identity matrices, and \( \mathbf{A} \) is the augmented numerator relationship matrix. The pedigree contained 5 generations and in total 49,645 animals. The rows of \( \mathbf{Z}_D \) contain a single 1 for the record of the observed individual itself. The rows of \( \mathbf{Z}_S \) contain a 1 for each group mate of the observed individual. The rows of \( \mathbf{Z}_M \) contain a single 1 for the group dam of the observed individual.

The phenotypic variances assumed for each model were (see Appendix for derivation).

model 1: \( \sigma_p^2 = \sigma_{\mathbf{a}_D}^2 + \sigma_{\mathbf{g}}^2 + \sigma_{\mathbf{pe}}^2 + \sigma_{\mathbf{e}}^2 \),

model 2: \( \sigma_p^2 = \sigma_{\mathbf{a}_D}^2 + (n-1)\overline{r}_i \left[ 2\sigma_{\mathbf{a}_D\mathbf{a}_S} + (n-2)\sigma_{\mathbf{a}_S}^2 \right] \\
+ (n-1)\sigma_{\mathbf{a}_S}^2 + \sigma_{\mathbf{g}}^2 + \sigma_{\mathbf{pe}}^2 + \sigma_{\mathbf{e}}^2 \),

model 3: \( \sigma_p^2 = \sigma_{\mathbf{a}_D}^2 + 2\overline{r}_i \sigma_{\mathbf{a}_D\mathbf{a}_M} + \sigma_{\mathbf{a}_M}^2 + \sigma_{\mathbf{i}}^2 \\
+ \sigma_{\mathbf{g}}^2 + \sigma_{\mathbf{pe}}^2 + \sigma_{\mathbf{e}}^2 \); and

model 4: \( \sigma_p^2 = \sigma_{\mathbf{a}_D}^2 + (n-1)\overline{r}_i \left[ 2\sigma_{\mathbf{a}_D\mathbf{a}_S} + (n-2)\sigma_{\mathbf{a}_S}^2 \right] \\
+ (n-1)\sigma_{\mathbf{a}_S}^2 + 2\overline{r}_i \sigma_{\mathbf{a}_D\mathbf{a}_M} + 2(n-1)\overline{r}_i \sigma_{\mathbf{a}_M}^2 \\
+ \sigma_{\mathbf{g}}^2 + \sigma_{\mathbf{pe}}^2 + \sigma_{\mathbf{e}}^2 \),

where \( n \) denotes the number of individuals in a group, \( \overline{r}_i \) denotes the mean relatedness among piglets in the same group, and \( \overline{r}_m \) denotes the mean relatedness between a group and their group dam.

Nonheritable effects generate a covariance among residuals of group mates equal to \( 2\sigma_{pe}^2 + (n-2)\sigma_{s}^2 + \sigma_{e_m}^2 \), with \( n \) individuals in a group (modified from Bijma et al., 2007a, see Appendix). This nonheritable covariance among group mates can be accounted for by fitting a correlation among residuals of group mates (Bijma et al., 2007a). Equivalently, a simpler method is to fit random group effects because the covariance within groups equals the variance among group means (Van Vleck and Cassady, 2005; Bergsma et al., 2008). Because variances are nonnegative, this is appropriate only when the covariance among group mates is greater than zero, which is likely unless \( n \) is small. Excluding random group effects can lead to a bias in the estimates of variance components.

Heritable Variance and Response to Selection

An interesting question is how much response to selection can be obtained from the combination of direct, maternal, and social genetic effects. When traits are affected by direct effects only, response to selection can be expressed as

\[
\Delta G = \nu \rho \sigma_A,
\]

where \( \Delta G \) is the genetic change in mean trait value, \( \nu \) the intensity of selection, \( \rho \) the accuracy of selection, and \( \sigma_A \) the additive genetic SD. This is a very powerful expression; it is valid for any selection strategy and partitions response into clearly distinct components. Selection intensity and accuracy depend on the breeders and express the quality of a breeding design on a standardized scale. Inspection of \( \nu \) and \( \rho \) reveals the prospects for improvement of a breeding design. When \( \nu \) approaches a value of \( \approx 3 \) and \( \rho \) a value of 1, for example, prospects for further improvement of a breeding schemes are extremely limited. The additive genetic variance, in contrast, is a biological parameter of the population, which is outside control of the breeders. It expresses the intrinsic potential of a population to respond to selection.

The above expression for response can be extended to social and maternal effects as follows. Trait affected by direct, social, and maternal effects may be modeled as

\[
P_i = A_{D,i} + \sum_{j=1}^{n-1} A_{S,j} + A_{M, dam(i)} + e_i,
\]

where \( P_i \) is the phenotype of individual \( i \), \( A_{D,i} \) its direct breeding, \( A_{S,j} \) the social breeding value of its group mate \( j \), the sum is taken over its \( n - 1 \) group mates.
because their social effects contribute to the phenotype of the individual, $A_{M,dam(i)}$ is the breeding value for maternal effect of its dam, and $e_i$ is a nonheritable residual. From taking the average phenotypic value of all individuals in the current and the next generation, it follows immediately that response to selection (i.e., the genetic change in mean trait value), equals

$$\Delta \bar{G} = \Delta A_D + (n-1)\Delta A_S + \Delta A_{M,i}.$$  

In this expression, the $(n-1)\Delta A_S$ appears because each phenotype contains $(n-1)$ social effects, one from each of the $(n-1)$ group mates of an individual. The maternal effect, in contrast, appears only once, because each phenotype contains a single maternal effect originating from the dam of the individual.

The above shows that response equals the per generation change in mean $A_D + (n-1)A_S + A_{M,i}$. Following Bijma et al. (2007b) and Eaglen and Bijma (2009), this quantity is referred to as a total breeding value (TBV),

$$\text{TBV}_i = A_{D,i} + (n-1)A_{S,i} + A_{M,i},$$

so that response to selection equals the per generation change in mean TBV. The “weights” on components of the TBV (i.e., the one for direct and maternal effect and the $n-1$ for social effect), are not arbitrary or economic weights, but follow directly from taking the average phenotypic value of all individuals. The TBV, therefore, is not an arbitrary construct, but defines an individuals breeding value as the average impact of its genes on the mean trait value of the population. This is precisely what matters for genetic improvement because it is the genes that are transmitted to the next generation.

Next, an expression for response by regressing the TBV on the selection criterion was obtained, $C$, giving $\Delta \bar{G} = b_{\text{TBV},C}i\sigma_C$, where $b_{\text{TBV},C}$ is the regression coefficient of TBV of an individual on its $C$-value, and $i\sigma_C$ is the selection differential. Substituting $b_{\text{TBV},C} = \text{Cov}(C_i, \text{TBV}_i) / \sigma_C^2$ yields

$$\Delta \bar{G} = ip\sigma_{\text{TBV}}, \quad [1a]$$

where $p$ is the accuracy of selection for TBV,

$$p = \text{Corr}(\text{TBV}, C) = \frac{\text{Cov}(\text{TBV}, C)}{\sigma_C \sigma_{\text{TBV}}}, \quad [1b]$$

and $\sigma_{\text{TBV}}$ is the SD among TBV of individuals, which is the square root of

$$\sigma_{\text{TBV}}^2 = \sigma_{A_D}^2 + 2(n-1)\sigma_{A_{BS}}^2 + (n-1)^2\sigma_{A_S}^2 + 2\sigma_{A_{DM}} + 2(n-1)\sigma_{A_{SM}} + \sigma_{M}^2. \quad [1c]$$

This result is derived with minimal assumptions and applies to any selection strategy. The derivation merely involves taking an average and applying the definitions of regression, correlation, and (co)variance.

Equation [1a] is completely analogous to the classical expression for response given above. It partitions response into selection intensity and accuracy, which are design parameters, and the genetic SD, $\sigma_{\text{TBV}}$, which is the biological parameter reflecting the ability of a population to respond to selection. Thus $\sigma_{\text{TBV}}$ is the analogy of $\sigma_A$. The $\sigma_{\text{TBV}}^2$ in Eq. [1c], therefore, summarizes genetic parameters for direct, social, and maternal effects into a single parameter describing the ability of a population to respond to selection. Moreover, the accuracy as defined in Eq. [1b] provides a single scale-free measure of the quality of a breeding scheme aiming to improve traits affected by direct, social, and maternal effects. The use of a single accuracy helps to identify breeding schemes that optimally utilize the available genetic variance. For socially affected traits, for example, Ellen et al. (2007) show that an accuracy approaching 100% can be obtained by using groups composed of family members in progeny testing schemes.

At first review, the expression for $\sigma_{\text{TBV}}^2$ seems to suggest that heritable variance increases with group size, and becomes very large with large groups, which is clearly incorrect. In large groups, however, the intensity of social interactions among individual animals is likely to be less than in small groups. As a consequence, $\sigma_A^2$ will be smaller in larger groups. Hence, it is not realistic to assume that a $\sigma_A^2$ estimated from data with a certain group size is valid for other group sizes as well. The issue, therefore, is not in the $(n-1)$ and $(n-1)^2$ terms in Eq. [1c], but in the dependency of $\sigma_A^2$ on group size. This is an empirical rather than theoretical issue. The square root of Eq. [1c], therefore, truly expresses the genetic differences among individuals that can be utilized for genetic improvement, provided that the $\sigma_A^2$ refers to the group size used in Eq. [1c]. In other words, in the expression for $\sigma_{\text{TBV}}^2$, one should use the group size found in the data from which $\sigma_A^2$ was estimated. To avoid confusion about the relationship between $\sigma_{\text{TBV}}^2$ and group size, it may be written that

$$\sigma_{\text{TBV}}^2 = \sigma_{A_D}^2 + 2\sigma_{A_{DM}} + \sigma_{A_{MS}}^2 + 2(n-1)\sigma_{A_{BS}}^2 + 2(n-1)\sigma_{A_{AS}}^2 + (n-1)^2\sigma_{A_S}^2,$$

which emphasizes that $\sigma_{A_{BS}}^2$, $\sigma_{A_{MS}}^2$, and $\sigma_{A_S}^2$ depend on group size. The relationship between the genetic parameters for social effects and group size can be estimated empirically when there is sufficient variation in group size in the data (Canario et al., 2010).
The relative magnitude of additive genetic variance is often expressed in terms of heritability, \( h^2 = \sigma_A^2 / \sigma_P^2 \), which is a scale-free parameter and therefore easier to interpret than \( \sigma_A^2 \). This definition can be generalized to traits affected by maternal and social effects (Bergsma et al., 2008; Eaglen and Bijma, 2009), giving

\[
T^2 = \frac{\sigma_{TBV}^2}{\sigma_P^2}.
\]

The \( T^2 \) expresses the heritable variance that can be used to generate response to selection relative to phenotypic variance. Thus \( T^2 \) is expressed on the same scale as the ordinary \( h^2 \). This is convenient because comparison of \( T^2 \) to \( h^2 \) immediately reveals the contribution of social effects to the genetic variance that breeders can use for improvement. When \( h^2 = 0.3 \) and \( T^2 = 0.6 \), for example, then the presence of social effects double heritable variance, indicating that social effects are very relevant for genetic improvement in this population.

Judging the relevance of social genetic effects based on \( \sigma_{APS}^2 / \sigma_P^2 \) rather than \( T^2 \) can be very misleading, particularly when estimates come from data with large groups. When groups are large, \( \sigma_{APS}^2 / \sigma_P^2 \) will be a small value, falsely suggesting that social effects are not important. However, because their contribution to the genetic variance that breeders can use for improvement is proportional to \( (n-1)^2 \), social effects can still contribute substantially to response even when \( \sigma_{APS}^2 / \sigma_P^2 \) is very small (Bergsma et al., 2008).

For the mixed models used here (see above), total heritable variance is given by

- model 1\(_{\text{basic}}\): \( \sigma_{TBV}^2 = \sigma_A^2 \);
- model 2\(_{\text{social}}\): \( \sigma_{TBV}^2 = \sigma_A^2 + 2(n-1)\sigma_{ADM} + (n-1)^2 \sigma_{L}^2 \);
- model 3\(_{\text{maternal}}\): \( \sigma_{TBV}^2 = \sigma_A^2 + 2\sigma_{L} + 2\sigma_{SM} + \sigma_{AM}^2 \); and
- model 4\(_{\text{social-maternal}}\): \( \sigma_{TBV}^2 = \sigma_A^2 + 2(n-1)\sigma_{ADSM} + (n-1)^2 \sigma_{L}^2 + 2\sigma_{L} + 2(n-1)\sigma_{SM} + \sigma_{AM}^2 \).

### RESULTS

Table 3 shows descriptive statistics of the data. Animals gained 251 g/d on average. Groups were on average composed of 10.8 (SD = 1.4) piglets, with a mean relatedness of 0.336 (SD = 0.12) among group mates and 0.375 (SD = 0.12) between piglets and their group dam.

Figures 1, 2, and 3 show estimates of effects of group size and parity of biological dam and group dam with model 3\(_{\text{maternal}}\). Figure 1 shows that piglets reared in smaller groups have a greater ADG. The effect of parity of biological dam on ADG was not significant (\( P = 0.30 \)) and relatively small, compared with the effects of group size and parity of the group dam, which were significant (\( P < 0.001 \); Figures 1 to 3). Piglets raised by third and fourth parity group dams had greater ADG compared with piglets from other parities (Figure 3).

Table 4 shows the log-likelihood and estimates of parameters for each model. Estimates of direct heritability were \( \approx 0.07 \) for all models. Estimates of social heritability ranged from 0.0007 through 0.0009, but were not significantly different from zero (SE = 0.0005). Estimates of maternal heritability were 0.06 and significantly different from zero. Estimates of variance due to litter effects ranged from 0.05 through 0.06 of phenotypic variance. The fractions of phenotypic variance accounted for by group effects ranged from 0.09 through 0.10 and by permanent environmental effects ranged from 0.04 through 0.09. The estimate of vari-

---

**Table 3.** Mean and SD of traits and relatedness among individuals

<table>
<thead>
<tr>
<th>Item</th>
<th>Mean</th>
<th>SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>ADG, g/d</td>
<td>251</td>
<td>61.18</td>
</tr>
<tr>
<td>Birth weight, kg</td>
<td>1.49</td>
<td>0.34</td>
</tr>
<tr>
<td>Weaning weight, kg</td>
<td>8.17</td>
<td>2.03</td>
</tr>
<tr>
<td>Weaning age, d</td>
<td>26</td>
<td>3.86</td>
</tr>
<tr>
<td>Average group size, n</td>
<td>10.8</td>
<td>1.40</td>
</tr>
<tr>
<td>Mean relatedness among piglets within group (( \tau_G ))</td>
<td>0.336</td>
<td>0.12</td>
</tr>
<tr>
<td>Mean relatedness between piglets in a group and their group dam (( \tau_M ))</td>
<td>0.375</td>
<td>0.12</td>
</tr>
</tbody>
</table>

---

**Figure 1.** Estimates (and SE bars) of differences of effects of group size from a group of 7 piglets (model 3\(_{\text{maternal}}\)). Based on the conditional Wald \( F \)-statistics, group size effects were significantly different (\( P < 0.001 \)).
Maternal and social genetic effects on average daily gain

Due to permanent environmental effects decreased substantially when the group dam genetic effect was included in the model. Estimates of genetic correlations between direct and social effects were negative, but not significantly different from zero because SE were greater than the estimates (Table 4). The $T^2$ was 0.07 for model 1 basic, 0.11 for model 2 social, 0.10 for model 3 maternal, and 0.15 for model 4 social-maternal. Both social effects models (model 2 social and 4 social-maternal) were not significantly better than their equivalent nonsocial effects models (model 1 basic and 3 maternal) according to the likelihood-ratio test ($P = 0.102$ and 0.486, respectively; Table 5). Both maternal models (model 3 maternal and 4 social-maternal) performed significantly better than their equivalent nonmaternal models (model 1 basic and 2 social) according to the likelihood-ratio test ($P < 0.001$ and $P = 0.005$, respectively; Table 5). The likelihood-ratio test did not show a significant ($P = 0.486$) difference between model 3 maternal and model 4 social-maternal (Table 5), so they are equivalent. Although model 4 social-maternal is a more complete model, model 3 is preferred because of statistical computation ease.

**Figure 2.** Estimates (and SE bars) of differences of effects of parity of biological dam from the first parity (model 3 maternal). Based on the conditional Wald $F$-statistics, parity effects of biological dam were not significantly different ($P = 0.304$).

**Figure 3.** Estimates (and SE bars) of differences of effects of parity of group dam from the first parity (model 3 maternal). Based on the conditional Wald $F$-statistics, parity effects of group dam were significantly different ($P < 0.001$).

### Table 4. Log-likelihoods (logL) and estimates of parameters1 for each model with SE given in subscripts

<table>
<thead>
<tr>
<th>Model</th>
<th>logL</th>
<th>$h^2_D$</th>
<th>$h^2_S$</th>
<th>$h^2_M$</th>
<th>$l^2$</th>
<th>$g^2$</th>
<th>$p^2$</th>
<th>$g_D$^2</th>
<th>$g_M$^2</th>
<th>$g_S$^2</th>
<th>$p_D$^2</th>
<th>$p_M$^2</th>
<th>$p_S$^2</th>
<th>$T^2$</th>
<th>$r_{SM}$</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 basic</td>
<td>-602</td>
<td>-0.066</td>
<td>0.069</td>
<td>0.050</td>
<td>-0.074</td>
<td>0.052</td>
<td>0.053</td>
<td>0.089</td>
<td>0.041</td>
<td>-0.086</td>
<td>0.094</td>
<td>0.053</td>
<td>0.095</td>
<td>0.054</td>
<td>0.01</td>
</tr>
<tr>
<td>2 social</td>
<td>-600</td>
<td>0.071</td>
<td>0.098</td>
<td>0.058</td>
<td>0.055</td>
<td>0.067</td>
<td>0.058</td>
<td>0.095</td>
<td>0.041</td>
<td>-0.086</td>
<td>0.094</td>
<td>0.053</td>
<td>0.095</td>
<td>0.054</td>
<td>0.01</td>
</tr>
<tr>
<td>3 maternal</td>
<td>-595</td>
<td>0.071</td>
<td>0.098</td>
<td>0.058</td>
<td>0.055</td>
<td>0.067</td>
<td>0.058</td>
<td>0.095</td>
<td>0.041</td>
<td>-0.086</td>
<td>0.094</td>
<td>0.053</td>
<td>0.095</td>
<td>0.054</td>
<td>0.01</td>
</tr>
<tr>
<td>4 social-maternal</td>
<td>-593</td>
<td>0.071</td>
<td>0.098</td>
<td>0.058</td>
<td>0.055</td>
<td>0.067</td>
<td>0.058</td>
<td>0.095</td>
<td>0.041</td>
<td>-0.086</td>
<td>0.094</td>
<td>0.053</td>
<td>0.095</td>
<td>0.054</td>
<td>0.01</td>
</tr>
</tbody>
</table>

1 Direct heritability: $h^2_D = \sigma_D^2 / \sigma^2$; social heritability: $h^2_S = \sigma_S^2 / \sigma^2$; maternal heritability: $h^2_M = \sigma_M^2 / \sigma^2$; litter effect: $l^2 = \sigma_L^2 / \sigma^2$; group effect: $g^2 = \sigma_{g}^2 / \sigma^2$; total heritable effect: $T^2 = \sigma_{T}^2 / \sigma^2$; genetic correlation between direct effect and social effect: $r_{ps}$; genetic correlation between direct and maternal effect: $r_{pm}$; genetic correlation between social and maternal effect: $r_{sm}$; genetic correlation between direct and group effect: $r_{pc}$; genetic correlation between social and group effect: $r_{sc}$; genetic correlation between maternal and group effect: $r_{mc}$; permanent environmental effect: $pe$; estimated genetic variation: $\sigma^2$; residual variance: $\sigma_e^2$; genetic correlation between direct and social effect: $rgDS$; genetic correlation between direct and maternal effect: $rgDM$; genetic correlation between social and maternal effect: $rgSM$. 

---

**Figure 2.** Estimates (and SE bars) of differences of effects of parity of biological dam from the first parity (model 3 maternal). Based on the conditional Wald $F$-statistics, parity effects of biological dam were not significantly different ($P = 0.304$).

**Figure 3.** Estimates (and SE bars) of differences of effects of parity of group dam from the first parity (model 3 maternal). Based on the conditional Wald $F$-statistics, parity effects of group dam were significantly different ($P < 0.001$).
DISCUSSION

The social interaction and maternal models were successfully combined. Estimates of variance components and likelihoods satisfied the convergence criteria for ASReml. The results showed no significant evidence of a heritable social effect for ADG of piglets and that the generally used maternal model (model 3maternal) performed equally well as the social-maternal model (model 4social-maternal).

The nongenetic terms in the models (litter, group, and permanent environment effects) were entered one by one into the models during the analysis to determine their effects on the estimates of the genetic variance components. The model including all 3 nongenetic terms had a significantly better likelihood than the models without one of the nongenetic effects. Excluding litter effects mainly increased estimates of variance due to direct genetic effects, but also due to social genetic effects. Excluding group effects increased the estimates of variance due to maternal genetic and social genetic effects. Excluding permanent environmental effects increased estimates of variance due to maternal genetic effects. Excluding social genetic effects from the full model (model 4social-maternal vs. model 3maternal) increased estimates of variance due to litter, group, and permanent environmental effects. Estimates of genetic variance due to direct and maternal genetic effects did not change. Excluding social genetic effects from the nonmaternal model (model 2social vs. model 1basic) increased estimates of variance due to direct genetic, group, and permanent environmental effects and slightly increased estimates of variance due to litter effects. As with other studies (Arango et al., 2005; Van Vleck and Cassidy, 2005; Bergsma et al., 2008; Chen et al., 2008; Hsu et al., 2010), the estimate of group variance increased when social effects were ignored and versa. An increase in litter variance in models without social effects was also reported by Bergsma et al. (2008), although the litter variances actually decreased in models without social effects in the studies of Arango et al. (2005) and Chen et al. (2008). In present study, there was some confounding between litter and group effects because not all litters were affected by cross-fostering. Excluding social genetic effects from the model resulted in increases in variance components due to group and litter effects.

Bijma et al. (2007a) showed that it is not possible to estimate social effects when groups are composed entirely of full sibs. Cheng et al. (2009) confirmed this; their estimates of variance components with Gibb sampling methods were biased when the simulated groups were composed of full sibs. With no cross-fostering, all groups would have been composed of full sibs. Thus, the greater amount of cross-fostering applied in this study made it possible not only to separate variation due to common environmental effects among litter mates due to the biological dam from those due to the foster dam, but also to estimate the variance due to social effects. In this study, a cross-fostering percentage of 28.8% led to an average relatedness among group mates of 0.336. This is quite large compared with previous studies regarding social genetic effects, the average relatedness within groups in the study of Bergsma et al. (2008) and Arango et al. (2005) were 0.18 and 0.10, respectively. Hsu et al. (2010) used a population with an average relationship among pen mates of 0.30 and obtained similar estimates of social heritability as our study. Less cross-fostering will lead to a greater relatedness within a group, which makes it more difficult to estimate variance due to social effects. What the ideal level of relatedness within groups is, and thus the ideal amount of cross-fostering, was not addressed in this study. One thing that is clear from Bijma et al. (2007a) is that social effects cannot be estimated when all groups are composed of full sibs nor when all groups are composed of half sibs; therefore, cross-fostering is necessary to estimate social effects in the suckling period.

The cross-fostering status (yes/no) of the piglets was included in a preanalysis of fixed effect, and was significant. Cross-fostered piglets grew on average 12.8 g/d less than piglets that were not cross-fostered (model 3maternal). However, it is debatable whether to include cross-fostering status into the model or not. On the experimental farm, cross-fostering was done to increase the chances of small piglets and to minimize birth weight variation in groups. Cross-fostering was, therefore, not a random process, but mainly animals with either the lightest or the heaviest birth weights of the litter were placed in groups with piglets of similar birth weight. So, correcting for cross-fostering status would actually be a correction for birth weight. The fact that cross-fostered piglets grew less was probably due to their own ability to grow, as indicated by their low birth weight, and not so much a result of the fact that they were cross-fostered. Therefore, such a correction was not made in this study.

Generally groups are standardized according to birth weight of piglets when cross-fostering is applied. This results in reduced variation in birth weight within a group, which is often considered to benefit survival of light BW piglets. It might also have an impact on the social interactions between group mates; therefore, a

<table>
<thead>
<tr>
<th>Models compared</th>
<th>LRT</th>
<th>df</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>soc vs. basic</td>
<td>4.56</td>
<td>2</td>
<td>0.102</td>
</tr>
<tr>
<td>mat vs. basic</td>
<td>14.84</td>
<td>2</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>soc-mat vs. soc</td>
<td>12.72</td>
<td>3</td>
<td>0.005</td>
</tr>
<tr>
<td>soc-mat vs. mat</td>
<td>2.44</td>
<td>3</td>
<td>0.486</td>
</tr>
</tbody>
</table>

Table 5. Likelihood-ratio tests of comparable models

1soc = social; mat = maternal.
2LRT: $\chi^2$ test statistic for the likelihood-ratio test $= -2(\text{LogL}_{\text{reduced model}} - \text{LogL}_{\text{full model}})$.
3Degrees of freedom for the $\chi^2$ test statistic.
completely random cross-fostering strategy might be more appropriate, but that would lead to less commercial practice. In this study, mainly the smallest and largest piglets in a litter were transferred. To reach the desired cross-fostering percentage (>25%), some additional randomly chosen piglets were cross-fostered. On average the CV of birth weight within groups after cross-fostering (CVw = 0.15) was less than, but quite similar to, the CV of birth weight within litters before cross-fostering (CVl = 0.17). The CV of birth weight in present study was similar to the CV of birth weight in nonmanipulated litters reported by Milligan et al. (2001). In the study of Milligan et al. (2001) reduced variation in birth weight did not lead to a change in missed nursings, teat consistency scores, or sibling aggression. Milligan et al. (2001) reported no significant difference in BW gain between piglets reared in groups with low birth weight variation (CV ~0.08) and high BW variation (CV ~0.25). Although Milligan et al. (2001) removed very small and unhealthy piglets, litters with more than 12 piglets, and litters raised by sick sows, their study showed no evidence that standardizing groups has an impact on competition in groups and growth.

The results show no significant evidence that ADG of piglets is affected by heritable social effects. For breeding purposes, this means that there is no reason to take heritable social effects into account if selection is for ADG of piglets. Under other conditions (e.g., group size, fostering strategy, pen dimensions), the results might be different. Therefore, these results cannot be used directly to predict social effects in other situations. In this study, the generally used maternal model performed equally well as the social-maternal model. These results suggest that the social-maternal model could be used for other species and traits with maternal effects related to behavior, if sufficient cross-fostering is applied.

LITERATURE CITED


**APPENDIX**

**Phenotypic Variance**

In scalar notation, model 4 is

\[
y_{ijklm} = \text{fixed} + A_{Di} + \sum_{i \neq j}^{n-1} A_{S,j} + A_{M,k} + I_i + g_m + p_k + e_{ijklm},
\]

where \(i\) denotes the individual producing the record, \(j\) its group mates, \(k\) its group dam, \(l\) its birth litter, and \(m\) its group. The nongenetic terms are mutually independent and independent of the genetic terms, whereas the genetic terms may be correlated due to genetic relatedness. Therefore, phenotypic variance equals

\[
\sigma^2_p = \text{Var}(y) = \sigma^2_{A_D} + \sum_{i \neq j}^{n-1} \sigma^2_{A_{S,j}} + \sigma^2_{A_M} + 2\text{Cov}(A_{Di}, \sum_{i \neq j}^{n-1} A_{S,j}) + 2\text{Cov}(A_{Di}, A_{M,k}) + 2\text{Cov}(\sum_{i \neq j}^{n-1} A_{S,j}, A_{M,k}) + \sigma^2_I + \sigma^2_g + \sigma^2_p + \sigma^2_e
\]

with \(\bar{r}_G\) denoting mean relatedness among group mates,

\[
\text{Var}\left(\sum_{i \neq j}^{n-1} A_{S,j}\right) = (n-1)\sigma^2_A + (n-1)(n-2)\bar{r}_G^2 \sigma^2_A,
\]

and

\[
\text{Cov}\left(A_{Di}, \sum_{i \neq j}^{n-1} A_{S,j}\right) = (n-1)\bar{r}_G \sigma_{A_{Di}A_{S,j}}.
\]

With \(\bar{r}_M\) denoting mean relatedness between individuals and their group dam, \(\text{Cov}(A_{Di}, A_{M,k}) = \bar{r}_M \sigma_{A_{Di}A_{M,k}}\), and \(\text{Cov}(\sum_{i \neq j}^{n-1} A_{S,j}, A_{M,k}) = (n-1)\bar{r}_M \sigma_{A_{SM}}\). Combining terms yields the expression for phenotypic variance for model 4 given in the main text. For models 1 through 3, the appropriate terms are omitted.

**Nongenetic Covariances Among Group Mates**

This section clarifies which biological components contribute to the covariance among group mates. The social and maternal effects affecting the phenotype of an individual of a heritable and a nonheritable component. Therefore, the following biological model underlying the observed phenotypes is assumed to be

\[
P_{ijkl} = A_{Di} + E_{Di} + \sum_{i \neq j}^{n-1} (A_{S,j} + E_{S,j}) + A_{M,k} + E_{M,k} + E_{C,l},
\]

where \(P_i\) is the phenotype of individual \(i\), depending on several genetic \((A)\) and environmental \((E)\) effects. The direct effect \((D)\) is caused by the individual \(i\) itself, the sum of social effects \((S)\) is caused by its \((n-1)\) group mates \(j\), the maternal effect \((M)\) is caused by its group dam \(k\) and the common prenatal environmental effect \((C)\) of the biological dam in common to all individuals in litter \(l\). This model shows that 3 components contribute to nongenetic covariance among group mates. First, group mates have the same group dam, contributing a covariance, \(\sigma^2_{A_{Di}A_{M,k}}\). Second, each pair of group mates has \((n-2)\) other group mates in common, contributing a covariance, \((n-2)\sigma^2_{E_{S,j}}\). Finally, the direct effect of an individual affects its own phenotype, whereas its social effect affects the phenotypes of its group mates. Because the direct and social effect of an individual may be correlated, this contributes a covariance, \(2\sigma^2_{E_{Di}}\) (Bijma et al., 2007b). In total, the nongenetic covariance among group mates equals \(2\sigma^2_{E_{Di}} + (n-2)\sigma^2_{E_{S,j}} + \sigma^2_{E_{M,k}}\), which is likely to be positive unless \(n\) is small.
This article cites 21 articles, 14 of which you can access for free at:
http://jas.fass.org/content/88/9/2883#BIBL