Heredity (2017) 118, 534–541
Official journal of the Genetics Society
www.nature.com/hdy

OPEN

ORIGINAL ARTICLE

The early-life environment of a pig shapes the phenotypes of its social partners in adulthood

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Social interactions among individuals are abundant, both in natural and domestic populations, and may affect phenotypes of individuals. Recent research has demonstrated that the social effect of an individual on the phenotype of its social partners may have a genetic component, known as an indirect genetic effect (IGE). Little is known, however, of nongenetic factors underlying such social effects. Early-life environments often have large effects on phenotypes of the individuals themselves later in life. Offspring development in many mammalian species, for example, depends on interactions with the mother and siblings. In domestic pigs, individuals sharing the same juvenile environment develop similar body weight later in life. We, therefore, hypothesized that offspring originating from the same early-life environment also develop common social skills that generate early-life social effects (ELSEs) that affect the phenotypes of their social partners later in life. We, therefore, quantified IGEs and ELSEs on growth in domestic pigs. Results show that individuals from the same early-life environment express similar social effects on the growth of their social partners, and that such ELSEs shape the growth rate of social partners more than IGEs. Thus, the social skills that individuals develop in early life have a long-lasting impact on the phenotypes of social partners. Early-life and genetic social effects were independent of the corresponding direct effects of offspring on their own growth, indicating that individuals may enhance the growth of their social partners without a personal cost. Our findings also illustrate how research devoted to quantifying IGEs may miss nongenetic and potentially confounded social mechanisms which may bias the estimates of IGEs.
Heredity (2017) **118,** 534–541; doi:10.1038/hdy.2017.3; published online 22 March 2017

INTRODUCTION

In polytocous mammals, the early-life environment provided by the mother and siblings influences the development of individuals, and may have profound effects on fitness and other phenotypic traits later in life (Henry and Ulijaszek, 1996; Margulis *et al.*, 2005; Hager and Johnstone, 2006). In early life, individual development is mainly under the control of the mother. With progress through lactation, the genes of the offspring increasingly determine development, because individuals become self-sufficient for feeding, and milk production of the mother becomes limited. Siblings from precocial mammals are capable of actively competing early in life and behaving synchronically at the udder to increase their milk intake (Drake *et al.*, 2008). Individuals are expected to respond to both their own state and that of their litter mates when signaling need and soliciting resource from their mother (for example, with teat massage; Godfray and Johnstone, 2000). As a consequence, individual development is increasingly affected by social interactions among litter mates. These experiences in early life may shape individual phenotypes later in life (Stockley and Parker, 2002), including behavior and social skills (Branchi, 2009; Hudson *et al.*, 2011; Nicolas *et al.*, 2011). Though there has been a strong research focus on the interactions between mother and offspring, the consequences of the early-life environment for the development of social effects that individuals express on traits of their social partners later in life have received limited attention (but see Rice *et al.*, 2008; Ahern and Young, 2009).

We formulated the hypothesis that individuals born in the same litter develop common social skills in early life that affect their social performance later in life. We will refer to those effects as early-life social effects (ELSEs). Thus, an ELSE is the effect of an individual on the trait value of a social partner, and this effect originates from the early-life environment that the focal individual experienced. Under this hypothesis, litter mates should show similar social effects on the phenotypic traits of their social partners in adulthood. We, therefore, investigated whether adults born in the same litter and bound for separation later in life had a similar effect on the growth rate of other individuals that are part of their social group as adult.

The social effect of an individual on the phenotypic traits of its social partners may originate not only from its early-life environment, but also have a genetic component (Griffing, 1967; Moore *et al.*, 1997) that is known as an indirect genetic effect (IGE). Hence, IGEs refer to the effects of an individual's genes on the trait values of other individuals (Wolf *et al.*, 1998). IGEs can have large effects on heritable variation and response to selection (Griffing, 1967; Bijma and Wade, 2008). They can, for example, reverse the direction of response to selection, increase heritable variation to levels exceeding phenotypic variance or entirely remove heritable variation at the population level despite nonzero heritability of trait values (Kirkpatrick and Lande,

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Received 20 September 2016; revised 16 December 2016; accepted 22 December 2016; published online 22 March 2017

1989; Moore *et al.*, 1997; Wilson *et al.*, 2009; McGlothlin *et al.*, 2010; Bijma, 2011). IGEs are widespread (Frank, 2007) and have been found in both natural and domestic populations. Examples include: social survival in hens (Ellen *et al.*, 2008); social growth in drosophila (Wolf, 2003); social aggression in mice (Wilson *et al.*, 2009) and pigs (Bergsma *et al.*, 2008); and social dominance in red deer (Wilson *et al.*, 2011). Therefore, we formulate the second hypothesis that growth rate in domestic pigs is affected by IGEs.

The intensity of social interactions can depend strongly on external factors, such as the number of interacting individuals. Depending on the species and the trait of interest, individual differences in behavior may either increase (Hemelrijk and Wantia, 2005) or decrease (Sumpter *et al.*, 2008) when group size increases. Behavioral strategies change with increasing group size to ensure social success at low cost (Andersen *et al.*, 2004). From a cost–benefit perspective, social contests are expected to be less frequent when the number of interacting individuals increases (in pigs and poultry: Pagel and Dawkins, 1997; Turner and Edwards, 2004; Estevez *et al.*, 2007). This phenomenon is also observed in humans (Dunbar, 1997; Suzuki and Akiyama, 2005) and in primate communities, in which individuals space themselves to minimize aggressive interactions (Hemelrijk and Wantia, 2005). A second mechanism underlying the effects of group size may relate to a change in the ability of individuals to recognize litter mates. After weaning, domestic mammals can recognize litter mates until a certain group size, above which social interactions depend more and more on recent familiarity (in pigs: Ewbank and Meese, 1971; Turner and Edwards, 2004).

In the presence of IGEs, heritable variation and response to selection depend on group size. Theoretical models suggest that a change in group size may even reverse the direction of response to selection (Griffing, 1967; Bijma and Wade, 2008). This prediction, however, depends on the relationship of the magnitude of IGEs with group size (Hadfield and Wilson, 2007; Bijma, 2010). A reduction in the intensity of social interactions with group size would reduce the magnitude of IGEs in large groups that may prevent large impacts of group size on response to selection. Hence, the dependency of IGEs on group size is a key element in dynamic models of co-evolution of trait values and group size. In this study, therefore, we also investigated a third hypothesis that both ELSEs and social genetic effects (IGEs) become smaller when group size in adulthood increases.

Separating genetic from nongenetic social effects is a challenge because the individuals that share the same early-life environment are also genetically related. However, populations of domestic pigs (*Sus scrofa*) offer a unique opportunity because it contains large families of both full- and half-siblings. The pig is an appealing species for studying social effects on trait values, because it exhibits considerable social skills and maternal care. The early-life environment created by interactions with litter mates provides a strong opportunity for piglets to develop social skills. Socialization is high when piglets establish dominance relationships by means of bullies and short fights with litter mates (Pitts *et al.*, 2000), whereas deviant play behavior patterns may lead to poor adult social skills (De Jonge *et al.* 1996). Separating genetic from nongenetic social effects on phenotypes of social partners expressed in adulthood is feasible because of extensive pedigree information, and the mixing of unacquainted individuals after weaning. The Swedish population of domestic pigs provides an ideal situation where litter mates are kept together for a longer period, offering ample opportunity for the development of social skills. Using a large data set on this population, we were able to quantify the impact of early-life experiences and genetic factors on social performance later in life, as measured by the impact of a pig on the growth rate of its group mates after mixing.

MATERIALS AND METHODS

Classical quantitative genetic model for growth

To investigate the presence and magnitude of ELSE and IGE, we analyzed growth rate from birth until end of fattening in Swedish Large White pigs (for a description of the population, see Appendix A). Mixed linear models with correlated random genetic effects, also known as 'animal models', were used to partition phenotypic variance into genetic (both direct and indirect) and environmental variance components (Henderson, 1975). This section describes sources of environmental variance and genetic variance that are important to account for in a quantitative model to study the growth rate of an individual living in group. First, we define several environmental effects that affect social interactions at the group level and influence modeling of growth traits.

Because in the adult stage, interacting individuals share the same environment, there is an obvious risk of confounding IGEs with environmental effects. To account for shared environment and for a nonheritable component of the indirect effect, the statistical model included pen effects and group effects. Pen effects account for specific characteristics of the pen (for example, lightening, location in the barn) that is relevant because the same pens were used repeatedly. Group effects account for a nongenetic covariance among interacting group mates, originating, for example, from temporary environmental effects or nonheritable behavioral interactions among group mates. Furthermore, to account for effects of early-life environment on growth rate of the focal individual, that is, the environment shared with litter mates, the statistical model included litter effects and permanent environmental effects. Litter and/or permanent mother effects are commonly included in genetic analysis of domestic pig data to avoid overestimation of genetic variances due to partial confounding of genetic effects with early-life environmental effects. Litter effects account for a nonheritable covariance among phenotypes of litter mates that may occur because they share the same early-life environment. In addition, permanent environmental effects of the mother account for nongenetic covariances among offspring of the same mother born in different litters, originating from, for example, lifelong differences in maternal ability among mothers.

Data were analyzed using restricted maximum likelihood methodology as implemented in the ASReml software package (Gilmour *et al.*, 2006). The basic model included the fixed effects of number of group mates (10 levels), sex (castrated male or female), the combination of herd, year and season as a factor, age and age^2 at weighing as covariates, and the random effects of the physical pen, the group identity, the litter of birth and the permanent environment of the mother. All effects were statistically significant ($P<0.05$). The pedigree information used for the analyses included 55 982 individuals.

The significance of random effects was tested by the change in log likelihood measured at convergence, using the χ^2 statistic and the difference in degrees of freedom between the two models, that is, the difference in the number of parameters between models. The initial model included direct genetic effects only.

$$\mathbf{y}=\mathbf{Xb}+\mathbf{Z}_D\mathbf{a}_D+\mathbf{Wc}+\mathbf{Vg}+\mathbf{Ul}+\mathbf{Tpe}+\mathbf{e}$$

(Model 1)where $\mathbf{y}$ is a vector of observations on the growth rate; $\mathbf{X}$, $\mathbf{Z}_D$,$\mathbf{W}$, $\mathbf{V}$, $\mathbf{U}$ and $\mathbf{T}$ are known incidence matrices; $\mathbf{b}$ is a vector for fixed effects; $\mathbf{a}_D$ is a vector of direct additive genetic effects of the individual producing the record, with $\mathbf{a}_D \sim N(0, \mathbf{A}\sigma^2_{A_D})$, $\mathbf{A}$ denoting the matrix of additive genetic relationships between pigs and $\sigma^2_{A_D}$ the direct additive genetic variance; $\mathbf{c}$ is a vector of random pen effects, with $\mathbf{c} \sim N(0, \mathbf{I}\sigma^2_c)$; $\mathbf{g}$ is a vector of random group effects, with $\mathbf{g} \sim N(0, \mathbf{I}\sigma^2_g)$; $\mathbf{l}$ is a vector of random litter effects, with $\mathbf{l} \sim N(0, \mathbf{I}\sigma^2_l)$; $\mathbf{pe}$ is a vector of random nongenetic permanent effects of the mother with $\mathbf{pe} \sim N(0, \mathbf{I}\sigma^2_{pe})$; and $\mathbf{e}$ is a vector of residuals, with $\mathbf{e} \sim N(0, \mathbf{I}\sigma^2_e)$.

Modeling early-life social effects and social genetic effects

This section introduces the comparison of nested models to test for ELSEs and IGEs. We formulate the hypothesis that the early-life social experience of a pig influences the growth rate of its social partners later in life. We analyze whether such nongenetic social effects can be disentangled from genetic social effects.

Furthermore, we investigate the consequences of ignoring ELSEs for the estimation of IGEs on group mates in adulthood.

With social interactions, the trait value of an individual may be affected by genes in other individuals. This phenomenon is quantified in IGE studies. An IGE is a heritable effect of an individual on trait values of other individuals. Classically, to estimate IGEs associated with social partners in adulthood, the model for growth rate is extended with random indirect genetic effects of group mates, following the methods outlined by Muir (2005), Bijma *et al.* (2007a) and Bergsma *et al.* (2008).

$$\mathbf{y}=\mathbf{Xb}+\mathbf{Z}_D\mathbf{a}_D+\mathbf{Z}_S\mathbf{a}_S+\mathbf{Wc}+\mathbf{Vg}+\mathbf{Ul}+\mathbf{Tpe}+\mathbf{e}$$

(Model 2) where $\mathbf{Z}_s$ is a known incidence matrix linking group mates to the record of an individual, and $\mathbf{a}_s$ is a vector of IGEs. Model 2 accounted for a covariance between direct and indirect genetic effects, using the variance structure $\begin{bmatrix}\mathbf{a}_D\\ \mathbf{a}_S\end{bmatrix}\sim MVN\left(\begin{bmatrix}\mathbf{0}\\ \mathbf{0}\end{bmatrix},\begin{bmatrix}\sigma^2_{A_D} & \sigma_{A_{DS}}\\ \sigma_{A_{DS}} & \sigma^2_{A_S}\end{bmatrix}\otimes\mathbf{A}\right)$. As the incidence matrix $\mathbf{Z}_s$ includes 1 for each group mate of the individual producing the record, the $\sigma^2_{A_S}$ refers to the variance of an IGE expressed on a single recipient and $\sigma_{A_{DS}}$ to the covariance between direct and indirect genetic effects.

In contrast to IGEs, the ELSE that an individual expresses on the phenotypes of its group mates does not originate from its genes but from the early-life environment that it experienced. Thus, the ELSE component in the phenotype of the recipient of the effect originates from the early-life social effects that its group mates experienced. Thus, ELSEs are nongenetic effects, different from the above-mentioned environmental effects because of the litter and the mother. To quantify ELSE, the model was extended with random social effects due to the early-life environment of group mates.

$$\mathbf{y}=\mathbf{Xb}+\mathbf{Z}_D\mathbf{a}_D+\mathbf{Z}_S\mathbf{a}_S+\mathbf{Wc}+\mathbf{Vg}+\mathbf{Ul}+\mathbf{Tpe}+\mathbf{Qk}+\mathbf{e}$$

(Model 3) where $\mathbf{k}$ is a vector of random ELSE and $\mathbf{Q}$ is a known incidence matrix. The $\mathbf{Q}$-matrix connects the growth rate record of a focal individual to the early-life environment of each of its group mates. As early-life environments are common to individuals born in the same litter, the length of $\mathbf{k}$ equals the number of litters in the data. In the row of $\mathbf{Q}$ referring to the record of the focal individual, each of its group mates has a 1 in the column of $\mathbf{Q}$ referring to the litter of birth of that group mate. Hence, the term $\mathbf{Qk}$ tests whether individuals born in the same litter, that is, experiencing the same early-life environment, show similar social effects on the growth rate of their group mates later in life. In Model 3, direct early-life effects in $\mathbf{l}$ and ELSE in $\mathbf{k}$ were assumed independent. To investigate whether both effects are correlated, Model 4 included a covariance between $\mathbf{k}$ and $\mathbf{l}$, $\begin{bmatrix}\mathbf{k}\\ \mathbf{l}\end{bmatrix}\sim MVN\left(\begin{bmatrix}\mathbf{0}\\ \mathbf{0}\end{bmatrix},\begin{bmatrix}\sigma^2_k & \sigma_{kl}\\ \sigma_{kl} & \sigma^2_l\end{bmatrix}\otimes\mathbf{I}\right)$.

To interpret the magnitude of ELSE, we compared its variance with phenotypic variance, and the average absolute value of ELSE with phenotypic s.d. in growth rate. As an individual's ELSE is expressed once in each of its $n-1$ social partners, its total ELSE on all its partners equals $(n-1)k$, with n denoting group size. This is similar to the social component of the total breeding value of an individual (Moore *et al.*, 1997; Bijma *et al.*, 2007a; Bijma, 2011). Hence, the variance of the total ELSE expressed by an individual equals $(n-1)^2\sigma^2_k$. Moreover, the average absolute value of the total ELSE expressed by an individual equals $0.798(n-1)\sigma_k$, where 0.798 is the average absolute value of a standardized normal variable, as can be found in a table of the normal distribution. In the Results, we will present the variance and mean absolute value of the total ELSE expressed by an individual on its group mates.

The impact of group size on social effects

The relationship between group size and social effects is of biological interest, as it affects heritable variance and response to selection in populations with varying group sizes, and may lead to dynamic co-evolution of trait values and group size (see Introduction). The large variation in group size in our population allowed us to quantify the relationship between the magnitude of social effects and group size. To quantify this relationship, we estimated the dependency of social effects on group size using the method of Bijma (2010). Model 3 was, therefore, extended with a dilution factor d, on either IGEs alone (Model 5), or on both IGEs and ELSEs (Model 6), using

$$A_{S,i}(n)=\left(\frac{\overline{n}-1}{n-1}\right)^d A_{S,i}(\overline{n})$$

where $A_{S,i}(n)$ is the social effect of individual i when it is expressed in a group of n members, d is the dilution factor and $A_{S,i}(\overline{n})$ is the social effect of i when it is expressed in a group of the average size. The d measures the effect of group size on the magnitude of social effects. With no dilution, $d=0$, social effects do not depend on group size, $A_{S,i}(n)=A_{S,i}(\overline{n})$. With full dilution, $d=1$, social effects are inversely proportional to group size, $A_{S,i,}(n)=\frac{\overline{n}-1}{n-1}A_{S,i}(\overline{n})$. Dilution of social litter effects was modeled in the same way. Dilution was incorporated in the mixed model by multiplying social effects with a matrix $\mathbf{D}$:

$$\mathbf{y}=\mathbf{Xb}+\mathbf{Z}_D\mathbf{a}_D+\mathbf{Z}_S\mathbf{D}_a\mathbf{a}_S+\mathbf{Wc}+\mathbf{Vg}+\mathbf{Ul}+\mathbf{Tpe}+\mathbf{QD}_k\mathbf{k}+\mathbf{e}$$

(Model 6) where $\mathbf{D}_a$ is a diagonal matrix with elements $D_{ii}=[(\overline{n}-1)/(n-1)]^d$, n denoting group size for the ith record, and $\mathbf{D}_k$ is the equivalent matrix for the ELSE. In Model 5, the $\mathbf{D}_k$ matrix was dropped. As $(\overline{n}-1)/(n-1)=1$ when $n=\overline{n}$, Models 5 and 6 yield estimates of the IGE variance and ELSE variance referring to the average group size, $\sigma^2_{A_S}(\overline{n})$ and $\sigma^2_k(\overline{n})$. The degree of dilution was estimated by varying d from 0 to 1 in steps of 0.1, and taking the maximum likelihood value as the best estimate.

Model 6 was extended with a correlation between direct and early-life social effects, giving Model 7. To evaluate whether ELSEs were significant in final model, they were omitted from Model 7, giving Model 8. To evaluate whether IGEs were significant in final model, they were omitted from Model 7, giving Model 9. In all models accounting for change in social effects because of variation in group size (Models 5–9), residual variances were allowed to vary among group sizes.

Phenotypic and heritable variation

Using a mean additive genetic relatedness between group mates of zero, the phenotypic variance was calculated as

$$\sigma^2_P=\sigma^2_{A_D}+\sigma^2_c+\sigma^2_g+\sigma^2_l+\sigma^2_{pe}+(\overline{n}-1)\sigma^2_{As}+(\overline{n}-1)\sigma^2_k+\sigma^2_e$$

In reduced models the relevant terms were omitted.

When trait values are affected by IGEs, breeding values of individuals may be summarized into a total breeding value (TBV, Bijma *et al.*, 2007a). The total genetic variance available for response to selection equals (Bijma, 2011),

$$\sigma^2_{TBV}=\sigma^2_{A_D}+2(\overline{n}-1)\sigma_{A_{DS}}+(\overline{n}-1)^2\sigma^2_{A_S}$$

Analogous to ordinary heritability, the total heritable variance was expressed relative to the phenotypic variance (Bergsma *et al.*, 2008):

$$T^2=\sigma^2_{TBV}/\sigma^2_P$$

and a comparison between T^2 and classical heritability h^2 reveals the impact of social interactions on the heritable variation that determines the potential of the population to respond to selection.

RESULTS

Early-life and genetic social effects

We found strongly significant ELSEs ($\sigma^2_k>0$, $P<0.001$, Model 7 vs Model 8, Table 1). Thus, individuals born in the same litter showed similar nongenetic effects on the growth of their group mates in adulthood, indicating that the environment that individuals experienced early in life affected their social performance later in life. ELSEs were large when compared with phenotypic differences in growth rate among individuals. The variance of the total early-life effect of an individual on the growth rate of all its group mates equaled 24% of the phenotypic variance in growth rate. Beware that this does not mean that ELSEs contributed 24% of phenotypic variance, but rather that the variance among individuals in their total ELSE on all their group mates had a magnitude equal to 24% of phenotypic variance. This total effect, however, does not surface fully in phenotypic variance because an individual's total ELSE is distributed over multiple

Table 1 Variance components (s.e.) for individual growth rate in a Swedish pig population

Model	*1*	*2*	*3*	*4*	*5*	*6*	*7*	*8*	*9*
$\sigma^2_{A_D}$	603 (68)	590 (67)	596 (67)	606 (68)	596 (67)	600 (67)	599 (67)	593 (67)	604 (68)
$\sigma^2_{A_S}$		11.5 (2)	4.0 (1.4)	3.9 (1.4)	11.9 (2.3)	8.9 (2.0)	5.6 (1.7)	17.9 (2.7)	
σ^2_P	5028 (46)	4983 (46)	4872 (45)	4850 (45)	4691 (51)	4668 (51)	4625 (51)	4771 (51)	4626 (51)
σ^2_{TBV}	603 (68)	1332 (184)	752 (142)	623 (131)	1342 (192)	1079 (170)	851 (151)	1789 (218)	604 (68)
h^2	0.12 (0.01)	0.11 (0.02)	0.12 (0.01)	0.13 (0.01)	0.13 (0.01)	0.13 (0.01)	0.13 (0.01)	0.12 (0.01)	0.13 (0.01)
$\hat{T}^2$	0.12 (0.01)	0.27 (0.04)	0.15 (0.03)	0.13 (0.03)	0.28 (0.04)	0.23 (0.04)	0.18 (0.03)	0.38 (0.05)	0.13 (0.01)
$r_{A_{DS}}$		0.07 (0.10)	−0.10 (0.15)	−0.28 (0.15)	0.06 (0.10)	−0.02 (0.11)	−0.07 (0.13)	0.12 (0.09)	
r_{kl}				0.27 (0.06)			−0.02 (0.05)		−0.03 (0.05)
σ^2_c	83 (14)	81 (13)	81 (13)	81 (13)	76 (13)	74 (13)	77 (13)	77 (13)	79 (13)
σ^2_g	502 (24)	408 (28)	276 (26)	268 (26)	258 (29)	252 (27)	210 (25)	350 (29)	218 (23)
σ^2_l	592 (34)	568 (33)	533 (33)	535 (32)	526 (32)	517 (32)	515 (32)	559 (33)	516 (32)
σ^2_k			16.2 (1.8)	14.0 (2.0)	13.1 (1.7)	16.3 (1.7)	19.8 (1.7)		22.4 (1.7)
σ^2_{pe}	164 (31)	158 (31)	154 (30)	146 (29)	151 (29)	147 (29)	146 (29)	151 (30)	148 (29)
σ^2_e	3084 (82)	3092 (43)	3080 (44)	3080 (44)	2897 (49)	2889 (50)	2890 (50)	2906 (49)	2894 (49)
$\sigma_{A_{DS}}$		6.2 (8.4)	−4.8 (7.2)	−14.6 (7.5)	5.2 (8.5)	−1.6 (8.1)	−4.1 (7.5)	12.5 (9.1)	
σ_{kl}				23.7 (5.1)			−1.9 (5.0)		−2.9 (5.0)
Log L	−203 049	−203 006	−202 309	−202 298	−202 272	−202 250	−202 230	−202 310	−202 243
N para	6	8	9	10	20	20	21	19	19
AIC	406 110	406 028	404 636	404 616	404 584	404 540	404 502	404 658	404 524

The significance of effects was tested by the change in log likelihood (Log L) at convergence between successive models. The average Information criterion (AIC) was calculated for each model using Log L and number of parameters (*N* para). Model 1 includes random pen effects σ^2_c, adult social group (nongenetic) effects σ^2_g, permanent environmental effects of the mother σ^2_{pe}, direct litter effects σ^2_l and direct genetic effects $\sigma^2_{A_D}$. Model 2 tests for social genetic effects (IGEs), $\sigma^2_{A_S}$, while accounting for the correlation between direct and social genetic effects $r_{A_{DS}}$ (covariance $\sigma_{A_{DS}}$). Model 3 tests for early-life social effects (ELSEs) σ^2_k. Model 4 tests for the correlation between direct and social early-life effects r_{kl} (covariance σ_{kl}). Models 5 and 6 arise from Model 3, and test the effect of group size on social variances (see also Figure 2). Model 5 includes a dilution factor of 1 on IGEs ($d_a = 1$). In addition, Model 6 includes a dilution factor of 1 on ELSE ($d_k = 1$). Model 7 tests for r_{kl} in Model 6. Estimated social variances from Models 5–8 refer to the average group size ($\overline{n} = 8.5$). Model 8 vs Model 7 tests for ELSE when IGEs with $d_a = 1$ are included in the model. Model 9 vs Model 7 tests for IGEs when ELSE with $d_k = 1$ are included in the model. For models without dilution, σ^2_{TBV} depends on group size. Results of σ^2_{TBV} for such models are presented using the average group size. With full dilution, $d = 1$, phenotypic variance becomes independent of group size. This follows from the Equations for $A_{S,i,(n)}$ and the Equation for σ^2_{TBV}. The ratio $h^2 = \sigma^2_{A_D}/\sigma^2_P$ is the classical heritability. The ratio $\hat{T}^2 = \sigma^2_{TBV}/\sigma^2_P$ expresses total heritable variance relative to phenotypic variance. Model 7 proved superior. Beware that estimates for social effects refer to the effect on a single group mate; the total effect on all group mates is on average a factor 7.5 greater, so that the variance of the total effect is on average a factor $7.5^2 = 56.25$ greater.

individuals. The average absolute value of an individual's total ELSE equaled 26.6 g per day, and this is 39% of the phenotypic s.d. in growth rate.

Moreover, the social effect of an individual on the growth rate of its group mates contained a heritable component (IGE) that contributed 30% of the total heritable variation available for response to selection (σ^2_{TBV}, Bijma, 2011) in growth rate ($P < 0.001$, Model 7 vs Model 9, Table 1; Model 7: $\mathbf{y} = \mathbf{Xb} + \mathbf{Z}_D\mathbf{a}_D + \mathbf{Z}_S\mathbf{D}_a\mathbf{a}_S + \mathbf{Wc} + \mathbf{Vg} + \mathbf{Ul} + \mathbf{Tpe} + \mathbf{QD}_k\mathbf{k} + \mathbf{e}$ with $\begin{bmatrix}\mathbf{k}\\ \mathbf{l}\end{bmatrix} \sim MVN\left(\begin{bmatrix}\mathbf{0}\\ \mathbf{0}\end{bmatrix}, \begin{bmatrix}\sigma^2_k & \sigma_{kl}\\ \sigma_{kl} & \sigma^2_l\end{bmatrix} \otimes \mathbf{I}\right)$). Thus, social skills depended not only on the environment experienced early in life, but were also partly genetically determined, and contributed significantly to the potential of growth rate to respond to selection in this population.

Social effects, due to both genetic factors and early-life experiences, were independent of the ordinary direct effect of an individual on its own growth rate, as indicated by the near-zero correlations between direct effects and social effects in Table 1 ($r_{A_{DS}}$ and r_{kl}). In other words, the effects of early-life environment and genetic factors on the growth rate of the individual itself were independent of the social effect that the individual expressed on the growth of its group mates. This result demonstrates that providing a positive social effect on the growth rate of a group mate had no cost for the individual itself.

Group size dependency

The magnitude of the social effect that an individual expressed on a single group mate depended on group size (Table 1, Models 5 and 6 vs Model 3). Accounting for the dependency of social effects on group size clearly increased the goodness of fit of the statistical model (Figure 1), and allowed us to better capture the social effects, both those due to ELSEs and IGEs (Table 1). In large groups, both IGEs and ELSEs on an individual group mate decreased, with $\hat{d}_a = 1$ and $\hat{d}_k = 1$. Thus, the expression of the social skills, acquired either early in life or determined genetically, depended on group size. As a consequence, the social genetic variance $(\sigma^2_{A_S})$ decreased proportionally to group size (Figure 2), so that total heritable variance (σ^2_{TBV}) in growth rate was independent of group size.

DISCUSSION

The social skills of individuals are extremely difficult to measure in a quantitative way because they are a complex combination of different types of interactions. The importance and effect of each interaction cannot be assessed easily. However, an individual's social skills may have an impact on measurable phenotypes of its social partners. Such impacts may be estimable using statistical models that include social effects, similar to the estimation of maternal effects on phenotypes of offspring (Falconer, 1989). Social effects originating from the early-life environment, referred to as ELSEs here, had a sizable effect on lifetime growth in domestic pigs. This implies that individuals who share the same early-life environment develop similar social skills, the effects of which can be observed in the phenotypes of their social partners later in life.

Our findings show that differences in early-life environment may affect phenotypes of other individuals that interact with the focal individual. The influence of an individual's early-life experience on its social performance has been investigated in mammalian species by disruption or deprivation of contact between the offspring and the mother. Such interventions may result in rather extreme social effects, not reflecting the naturally occurring range of social effects. Apart from humans (Hartup, 1983; Fantuzzo *et al.*, 1988) and Rhesus

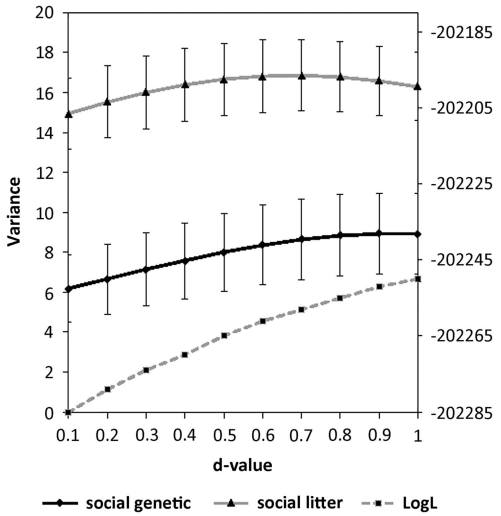

Figure 1 Variances of social genetic effects (IGEs) and of early-life social effects (ELSE) as a function of the dilution factor (d), with standard errors (±s.e.). Herein, the dilution factors for IGEs (d_a) and ELSE (d_k) varied from 0.1 to 1, but had the same value, $d_a = d_k = d$. Log-likelihood values (Log L) are given on the secondary y axis.

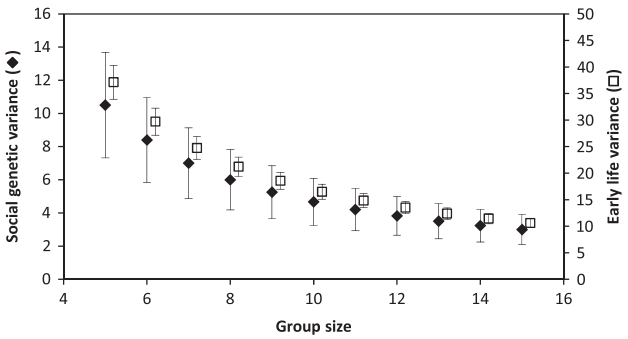

Figure 2 Variances of social genetic effects (IGEs) and of early-life social effects (ELSE), as a function of group size, with standard errors (±s.e.) and for $d_a = d_k = 1$ (Model (7) in Table 1).

monkeys (Harlow and Suomi, 1971), for which it is clear that peer interactions enhance the development of sophisticated social responses and social skills, the long-term social effects of early-life social interactions among litter mates are largely unknown (but see Branchi, 2009 in mice and Macri and Wurbel, 2006 in rats). In pigs, a species with large cognitive abilities, early-life social experiences affect how adult animals react to novel situations. Social skills are expected to have long-lasting benefits, for instance, enabling pigs to solve dominance conflicts more rapidly when they occur in adult life at meeting with unfamiliar pigs (D'Eath, 2005).

ELSEs and IGEs can be distinguished because related individuals have similar IGEs, but only litter mates have the same ELSE. Compare, for example, full-sib litter mates with half sibs that are from different litters. Because of both IGE and ELSE, the covariance between social partners of two full-sib litter mates equals $\frac{1}{2}\sigma^2_{A_S} + \sigma^2_{ELSE}$, whereas the covariance between social partners of two half sibs equals $\frac{1}{4}\sigma^2_{A_S}$. Hence, information to separate IGEs from ELSEs comes, for example, from the covariance between social partners of full sibs vs that between social partners of half sibs. This is similar to the common method of breeders to distinguish between (direct) additive genetic variance and the common-litter (direct) variance in data containing a mix of full and half sibs families. The mixing of individuals from different litters with a reasonable number of litter mates in each group was the key to disentangle ELSEs from IGEs. Note that omitting ELSEs from the model substantially inflated the estimated IGEs (Model 2 vs Model 3 and Model 8 vs Model 7, Table 1). If ELSEs are ignored, their variance is partly included into that of the estimated IGEs because the two sources of variation are partly confounded. ELSEs were large and hence they shaped the growth rate of social partners more than IGEs. Thus, ELSEs are not only of biological interest in their own right, but may also need to be considered to obtain unbiased estimates of IGEs.

The null genetic correlations obtained between direct and social effects on both IGEs and ELSEs show respectively: (1) that pigs display a genetic capacity to influence the growth of group mates independent of their own genetic capacity to grow and (2) that pigs expressing social skills benefitting the growth of group mates do not necessarily originate from litter environments favoring their own growth. This result suggests that early-life environments enhancing the physical development of individuals themselves are different from the environments enhancing the development of social skills. The null correlations indicate that positive ELSEs and IGEs do not come at a cost to the individual, at least in terms of growth rate. Maybe 'helpful' pigs for the growth of others are merely pigs not disturbing other pigs. Alternatively, the absence of a negative relationship between direct and social effects may reflect the abundance of feed in our population. If independence of direct and social effects extends to natural populations, it creates the opportunity for the evolution of helping behavior for growth without an accompanying cost for the personal growth rate of an individual.

Both IGEs and ELSEs decreased proportionally to the number of group mates of an individual. As a consequence, an individual's total social effect summed over all its group mates was independent of group size. This result suggests that the social effect of an individual on a single group mate decreases in large groups because the total effect is distributed over more recipients, a phenomenon known as dilution (Bijma, 2010).

Models of IGEs predict that variation in group size may reverse the direction of response to selection (Griffing, 1967; Moore *et al.*, 1997; McGlothin *et al.*, 2010). The direction of response to selection is determined by the sign of the covariance between an individual's phenotypic trait value and its total breeding value A_T (Griffing, 1967; Bijma and Wade, 2008) that equals

$$Cov(P, A_T) = \sigma^2_{A_D} + (n-1)\sigma_{A_{DS}}(n)$$

when group members are unrelated, where $\sigma^2_{A_D}$ is the direct genetic variance and $\sigma_{A_{DS}}(n)$ the direct–indirect genetic covariance that may depend on group size (n). When $\sigma_{A_{DS}}(n) < 0$, a change in group size may change the sign of the covariance, thus reversing the direction of response to selection. Our results, however, showed that IGEs were inversely proportional to the number of group mates, so that $\sigma_{A_{DS}}(n) = \sigma_{A_{DS}}(\overline{n})/(n-1)$, with $\overline{n}$ denoting the average group size. Consequently, the covariance between an individual's trait value and its total breeding value was independent of group size, $Cov(P, A_T) = \sigma^2_{A_D} + \sigma_{A_{DS}}(\overline{n})$. In this population, therefore, the decrease of IGEs with group size would prevent a change in the direction of response to selection because of a change in group size, irrespective of the genetic correlation between direct and indirect genetic effects.

In mammals, mothers shape the development of their offspring (Mousseau and Fox, 1998; Meaney, 2001). We, therefore, also investigated the presence of maternal genetic effects on the growth rate, and the relationship between maternal effects on the growth of

the offspring itself and the IGE of those offspring on the growth of their group mates. Results showed small but statistically significant heritable maternal effects (Appendix B). Thus, although maternal effects usually diminish with time elapsed from birth (Wilson and Réale, 2006), the maternal genetic contribution to growth was still observed in the pigs in adulthood. The data did not allow to simultaneously fit both ELSEs and maternal genetic effects. Thus, maternal genetic effects were omitted from the final model. We compared Akaike information criterion of models containing either maternal genetic effects or ELSEs, and found a considerably better fit for the model with ELSEs (Akaike information criterion = 404 502 in Table 1 vs 404 712 in Appendix B Table 1). Using a reduced model, we found a positive genetic correlation between maternal effects on offspring growth and offspring social performance ($r_{DS} = 0.47$). Thus, mothers with positive maternal genetic effects beget offspring that, later in life, tend to have positive IGEs on the growth of their group mates. The extent to which the social skills underlying the growth of group mates depend on maternal care is not known in pigs, but several studies in mice and humans give clear indication of the importance of maternal care for adult performance (Meaney, 2001; Fries *et al.*, 2005; Champagne, 2008).

An interesting question is whether our results extend to natural populations. The ELSEs are probably more important in natural populations than in domestic populations, because behavioral interactions in the wild are more important, and litter mates probably stay together for a much longer period of time. To detect ELSEs and separate them from IGE, information on genetic relatedness is required, either from pedigree or molecular markers. Furthermore, social groups should differ between early life and adulthood.

A considerable proportion of the heritable variation in pig growth depended on social interactions, meaning that the response to selection in this trait depends on genetic relatedness among group mates (Griffing, 1967; Cheverud, 2003; Bijma *et al.*, 2007a). This suggests that breeders can use artificial kin selection to genetically improve the growth rate in pigs (Wade *et al.*, 2010), and this may offer a promising route to simultaneously improve productivity and welfare in domestic animals. Moreover, our findings suggest that social effects on group mates and maternal effects on offspring are co-inherited, which further enhances opportunities for sustainable genetic improvement in domestic pigs.

Our analysis provides evidence that the growth in domestic pigs, bound to live at high density, is affected by several genetic and nongenetic social factors that are expressed later in life but originate in part from the early-life environment. ELSEs were identified as a new source of environmental influence on the performance of growing animals in a social context. Large ELSEs were identified, meaning that an individual can strongly influence the phenotypes of its social partners by means of its social skills acquired in early life. Moreover, accounting for ELSEs is required in IGEs studies to avoid bias in the estimated genetic parameters for indirect effects. Further work in this area could focus on the identification of the causal pathways and behavioral processes that underlie our findings.

DATA ACCESSIBILITY

Data available from the Dryad Digital Repository: http://dx.doi.org/10.5061/dryad.48963.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

ACKNOWLEDGEMENTS

The data used for this study were kindly provided by Nordic Genetics, Sweden.

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APPENDIX A

This appendix describes the study population. We analyzed the lifetime growth rate of pigs originating from a Yorkshire breeding line kept at 10 breeding herds in Sweden. Data were provided by Nordic Genetics, the Swedish company for pig genetic evaluation (S-242 92, Hörby, Sweden). Under Swedish farming conditions, mothers are raised with their litter in loose-housing lactation pens until ~5 weeks of age. Afterwards, mothers are removed from the lactation pen, whereas piglets remain with their litter mates until ~10 weeks of age. In this study, interest was in the ELSE originating from the first 10 weeks of an individual's life. Litters are mostly formed of full-sibs until this age. Next, they are moved by the farmers to the fattening facility of the herd, where they are mixed with siblings and unfamiliar pigs to form groups of pigs that will remain together until the end of the fattening period. A total of 5 to 15 pigs of same gender were mingled in each pen to form the groups of pigs. As common in pig production, the penning strategy applied by the farmer was to limit variation in body weight among pen mates, and to maximize the number of litters involved per pen so as to avoid confounding of litter with fattening pen. During the study period, animals were fed *ad libitum* and had permanent access to water. Body weight of the pigs was recorded at the end of the fattening period, at an average weight of ~100 kg. The growth rate per day, known as average daily gain (ADG) in pig breeding, was derived: ADG = weight/(date of weighing − date of birth). Information was available on 43 332 pigs, born from 6461 litters, 4005 mothers and 424 fathers. Because of mortality, some records were missing, but missing records accounted for <5% of observations.

APPENDIX B

This appendix describes the models and results used for the estimation of maternal genetic effects. Indeed, to investigate whether the growth rate was affected by maternal genetic effects, which might affect estimates for both IGEs and ELSEs, maternal genetic effects were included in the model with direct genetic effects (Model 1), using

$$\mathbf{y}=\mathbf{Xb}+\mathbf{Z}_D\mathbf{a}_D+\mathbf{Z}_M\mathbf{a}_M+\mathbf{Wc}+\mathbf{Vg}+\mathbf{Ul}+\mathbf{Tpe}+\mathbf{e}$$

(Model 10)and in the model with both direct and social genetic effects (Model 2), using

$$\mathbf{y}=\mathbf{Xb}+\mathbf{Z}_D\mathbf{a}_D+\mathbf{Z}_S\mathbf{a}_S+\mathbf{Z}_M\mathbf{a}_M+\mathbf{Wc}+\mathbf{Vg}+\mathbf{Ul}+\mathbf{Tpe}+\mathbf{e}$$

(Model 11)where $\mathbf{a}_M$ is a vector of random maternal genetic effects and $\mathbf{Z}_M$ the corresponding incidence matrix linking observations on pigs to the maternal-effect breeding value of their mother. The covariance structure of the additive genetic effects was then:

$$\begin{bmatrix}\mathbf{a}_D\\ \mathbf{a}_S\\ \mathbf{a}_M\end{bmatrix}\sim MVN\left(\begin{bmatrix}0\\0\\0\end{bmatrix},\begin{bmatrix}\sigma^2_{A_D} & \sigma_{A_{DS}} & \sigma_{A_{DM}}\\ \sigma_{A_{DS}} & \sigma^2_{A_S} & \sigma_{A_{SM}}\\ \sigma_{A_{DM}} & \sigma_{A_{SM}} & \sigma^2_{A_M}\end{bmatrix}\otimes\mathbf{A}\right)$$

where $\sigma_{A_{SM}}$ describes the covariance between maternal and social genetic effects. A model with IGEs, ELSEs and Maternal effects was attempted, but did not converge. Moreover, using an additive genetic relatedness between piglets and their mother of ½, phenotypic variance for Models 10 and 11 was calculated as

$$\sigma^2_P=\sigma^2_{A_D}+\sigma^2_c+\sigma^2_g+\sigma^2_l+\sigma^2_{pe}+(\overline{n}-1)\sigma^2_{A_S}+\sigma_{A_{DM}}+\sigma^2_{A_M}+\sigma^2_e$$

For Model 11, total heritable variance was

$$\sigma^2_{TBV}=\sigma^2_{A_D}+2(\overline{n}-1)\sigma_{A_{DS}}+(\overline{n}-1)^2\sigma^2_{A_S}+2\sigma_{A_{DM}}+2(\overline{n}-1)\sigma_{A_{SM}}+\sigma^2_{A_M}$$

For the reduced model, the appropriate terms were omitted.

Appendix B Table 1. Variance components (s.e.) for individual growth rate with maternal effects

Model	*10*	*11*
$\sigma^2_{A_D}$	585 (81)	562 (33)
σ^2_{As}		11.7 (2.1)
$\sigma^2_{A_M}$	104 (39)	104 (38)
σ^2_P	5034 (46)	4975 (46)
σ^2_{TBV}	587 (73)	1401 (192)
h^2	0.12 (0.02)	0.11 (0.02)
$\hat{T}^2$	0.12 (0.02)	0.28 (0.04)
$r_{A_{DS}}$		−0.08 (0.12)
$r_{A_{DM}}$	−0.21 (0.16)	−0.17 (0.16)
$r_{A_{SM}}$		0.47 (0.18)
σ^2_c	83 (14)	81 (13)
σ^2_g	501 (23)	414 (27)
σ^2_l	585 (33)	562 (33)
σ^2_{pe}	135 (35)	114 (34)
σ^2_e	3092 (48)	3091 (49)
$\sigma_{A_{DS}}$		−6.3 (9.7)
$\sigma_{A_{DM}}$	−51 (45)	−41 (44)
$\sigma_{A_{SM}}$		16 (7)
Log L	−203 042	−202 347
N para	8	11
AIC	406 100	404 712

The model for analyses of the growth rate yielded estimates of variances for random pen effects σ^2_c, social group (nongenetic) effects, σ^2_g, direct litter effects σ^2_l, random nongenetic permanent effects σ^2_{pe}, direct additive genetic effects $\sigma^2_{A_D}$, social genetic effects, σ^2_{As}, and maternal genetic effects $\sigma^2_{A_M}$, and of the correlation between direct and social genetic effects $r_{A_{DS}}$, direct and maternal genetic effects $r_{A_{DM}}$ and maternal and social genetic effects $r_{A_{SM}}$, with corresponding covariances $\sigma_{A_{DS}}$, $\sigma_{A_{DM}}$ and $\sigma_{A_{SM}}$ respectively. The ratio $h^2 = \sigma^2_{A_D}/\sigma^2_P$ is the classical heritability. The ratio $\hat{T}^2 = \sigma^2_{TBV}/\sigma^2_P$ expresses total heritable variance relative to phenotypic variance. Based on the difference in log likelihood at convergence between both models, Model 11 proved superior over Model 10 ($P<0.0001$), but Model 7 (Table 1) was superior over Model 11.