Social genetic effects (IGE) and genetic intra- and intersexual genetic correlation contribute to the total heritable variance in parental care

Julia Schroeder[1], Hannah Dugdale[2], Shinichi Nakagawa[3], Alexandra Sparks[2], and Terry Burke[4]

[1] Department of Life Science, Imperial College London, Silwood Park, Ascot, UK

[2] School of Biology, The Faculty of Biological Sciences, University of Leeds, Leeds, UK

[3] Evolution & Ecology Research Centre and School of Biological, Earth and Environmental Sciences, University of New South Wales, Sydney, Australia

[4] Department of Animal and Plant Sciences, University of Sheffield, Sheffield, UK

Corresponding author:

Julia Schroeder: julia.schroeder@gmail.com

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Abstract

The social environment can influence phenotypes through indirect genetic effects (IGEs), whereby genetic variance among interacting individuals explains some of the phenotypic variance. Empirical studies of wild populations often ignore IGEs especially among unrelated individuals, probably due to data limitations. This is problematic because IGEs can crucially affect estimates of heritable variation and subsequently influence the predicted evolutionary change. We here present a quantitative genetic analysis of biparental care in a natural bird population using a genetic pedigree. For both sexes, the conventionally calculated repeatability (15% in the female trait and 19% in the male trait) was lower than the total heritable variation including IGEs (24% in the female trait, and 25% in the male trait). These estimates of total heritable variation was also larger compared to conventionally calculated heritability (13% in both sexes), suggesting that parental care can evolve through social selection. Furthermore, we detected statistically significant genetic covariance between direct genetic effects, and between IGEs and direct genetic effects. Our work showcases how IGEs can represent substantial and important hidden heritable variance and highlights the importance of considering IGEs for theoretical models of parental care for ecology and evolution.

Introduction

All organisms behave and interact with conspecifics, and the knowledge of how interactions among phenotypes feedback into an individual's behaviour is crucial to understanding how phenotypic and genotypic variance of interactive behaviours is maintained. Furthermore, understanding feedback between social interactions and phenotypes can help us to understand how selection can act on interactive behaviours, and how they evolve.

Interactive traits by definition involve more than one individual. Interacting individuals can influence each other's phenotypes, and, depending on the trait in question, even fitness (Bijma et al. 2007). Thus, the variation of a phenotype in a population will partly depend on the social environment (Bergmüller & Taborsky 2010; Bleakley et al. 2010). This concept is similar to the idea of an extended phenotype (Dawkins 1999), except that the focus is on social interactions rather than abiotic factors influencing phenotypes (Moore et al. 1997). Variance in phenotypes is conventionally partitioned into variance due to genetic effects, and that due to environmental effects. The environmental influence on phenotypes can partially stem from the social environment (Moore et al. 1997; Bleakley et al. 2010; McGlothlin et al. 2010). Some of this variation explained by the social environment can, in turn, be determined by genetic variation in the interacting individuals. This genetic variation in the interacting individuals, that affects the phenotypes of another individual, is a *social*, or *indirect genetic effect, or IGE* (Box 1). As selection is expected to act on IGEs, IGEs should be considered when studying the how interactive traits can

Box 1: Terminology

In the existing literature the term, *indirect genetic effects* is also referred to as *associative genetic effects*, which means the same (Bijma et al. 2007) but can lead to confusion. Note that neither term does not clarify the origin of the genetic effect. The term *indirect genetic effect* is used to mean a range of things; it can even represent effects between different species, adding to the confusion (see for example (Bailey et al. 2011). In this work, the terms *social effect* refers to an effect that stems from a social environment: from one, or more, social interaction(s) with other individual(s), as opposed to indirect effects from an abiotic environment. Following this, an *social environmental effect* is one that stems from a social environment, but without an additive genetic component, while an *indirect genetic effect* (IGE) is a social genetic effect. A *maternal* genetic effect is a special form of IGE in which the interacting individual is the mother (Wilson et al. 2005).

62 evolve in a changing world. In fact, the non-consideration of IGE's is discussed as one reason for
63 the so often missing response to selection in the wild (Pujol et al. 2018).
64 IGEs can be surprisingly large (Wolf et al. 1998). Importantly, if the IGE is large, a trait still
65 has the potential for rapid evolution even when the estimate of direct heritable variance is close to
66 zero. Positive covariance between the direct genetic effect and the IGE can accelerate the evolution
67 of a trait, and negative covariance can constrain it (Moore et al. 1997; Wolf et al. 1998; Bijma
68 2011). Therefore, if IGEs are not accounted for, the additive genetic variance might be under- or
69 over-estimated (Wolf et al. 1998; Bijma 2011). Such an error can affect our judgment of the
70 potential evolutionary trajectory of the trait (Wolf et al. 1998). While the importance of IGEs is
71 appreciated in breeding programs (Muir 2005), only a few ecological and evolutionary studies have
72 quantified IGEs among unrelated individuals in natural animal populations (with the exception of
73 maternal genetic effects, which are better studied (McAdam & Boutin 2004, Wilson et al. 2005;
74 McAdam et al. 2014)). The few studies to date on wild animal populations have focused on SGEs
75 on aggressive behaviours (Wilson et al. 2011), and on IGEs on a single-sex life-history trait – the
76 timing of breeding (Brommer & Rattiste 2008; Teplitsky et al. 2010, Fisher et al. 2018). However,
77 cooperative behaviours between unrelated individuals, due to their highly social and interactive
78 nature, may harbour a larger potential for important and interesting IGEs, and would therefore seem
79 to be promising traits to study. The lack of studies of IGEs on cooperative behavioural traits may be
80 due to the limited availability of long-term observational data on behavioural traits from wild,
81 pedigreed populations (Fisher & McAdam et al. 2008). Here, we study parental care in a wild bird
82 population.
83 In species with biparental care, the amount and type of care delivered to dependent offspring
84 is often dependent on the amount and type of care delivered by the social mate. Therefore, social
85 mate effects including IGEs are likely to affect the phenotypic trait expressed by the other
86 individual of a pair. Yet, absent or reduced parental care has dire fitness consequences, and parental

87 care is generally expected to come at a cost to the individual delivering the care, posing a conflict
88 between biparental care parents (Clutton-Brock 1991; Royle et al. 2012). This close relation to
89 fitness implies that heritable variation in this trait may have been depleted by selection. However,
90 there is suggestive evidence that parental care has a heritable component in at least some
91 populations (Walling et al. 2008; Dor & Lotem 2010). We lack, however, empirical evidence from
92 wild populations on IGEs of parental care between the mostly unrelated members of a social pair.
93 As parental care behaviour is under strong phenotypic selection – because offspring survival
94 depends on it – the knowledge of, and if so, how, IGEs affect the phenotype is crucial to
95 understanding the evolutionary ecology of parental care behaviour (Bijma & Wade 2008;
96 McGlothlin et al. 2010).
97 Here, we study IGEs on parental care – provisioning behaviour to dependent young –
98 observed in a natural, genetically pedigreed, population of house sparrows (*Passer domesticus*).
99 House sparrows exhibit biparental care, and males are phenotypically more predictable carers than
100 females (Nakagawa et al. 2007a). Furthermore, males flexibly adjust their paternal care according
101 to the identity of their partners (Schroeder et al. 2016). Therefore, we predicted that in this species,
102 IGEs might differ by sex. We used uni– and multi–variate 'animal models' (Kruuk 2004) to estimate
103 sex-specific quantitative genetic parameters, namely repeatability, heritability, IGEs, genetic
104 correlations and total heritable variation, of parental care behaviour. We found evidence supporting
105 IGEs, genetic correlations, and social environmental effects in parental care. Our work showcases
106 the importance of accounting for social effects in the framework of evolutionary and ecological
107 studies.
108
109 **Methods**
110 ***Field data***
111 Data were collected from the house sparrow population breeding on Lundy Island, UK (51°10'N,

4°40’W). This long-term nest-box population has been closely monitored since 2000, such that we know the complete life-histories from birth for nearly every bird in the population (Schroeder et al. 2012a, 2015). All birds received a metal ring from the British Trust for Ornithology, an individual colour-ring combination and most birds received a passive-integrated transponder (PIT); these have no detectable effect on survival or subsequent reproductive success (Nicolaus et al. 2008; Schroeder et al. 2011). We used all three methods to identify parents at nest boxes. From 2004–2015, we have collected data on parental care, quantified from video observations (N = 3579 videos, Nakagawa et al. 2007b; Schroeder et al. 2012b, 2016). The majority of parental care observations were collected on days six and seven (1217 observations) and on days 11 and 12 (1543) after the chicks hatched (hatching day = 1); however, we have additional videos (819) from every day after hatching and used this full dataset for the analysis (Fig. 1). Parental care observations were measured as the number of visits of a parent to the nest box per hour. To calculate nest-box visit frequency (in the following: parental care), we calculated the ratio of nest box visits over the time period from the first time a bird entered the nest box to the end of the video (mean: 87.85 minutes, 87.67–88.04 95CI). For more methodological details see (Nakagawa et al. 2007a; Schroeder et al. 2013). In total, the dataset comprised 6873 observations of parental care (a male and a female per one video observation) by 613 pedigreed individuals (311 females and 302 males) for 1240 broods in the years 2004–2015. Among those, 140 females and 132 males changed their social partner at least once during their lifetime, these made up of 4590 observations (67%). In total, there were 837 unique parent–pair combinations. Of the 272 non-monogamous individuals, 160 had two different social partners, 67 had three, 31 had four, ten had five, and four had six different partners, providing sufficient statistical power for these analyses.

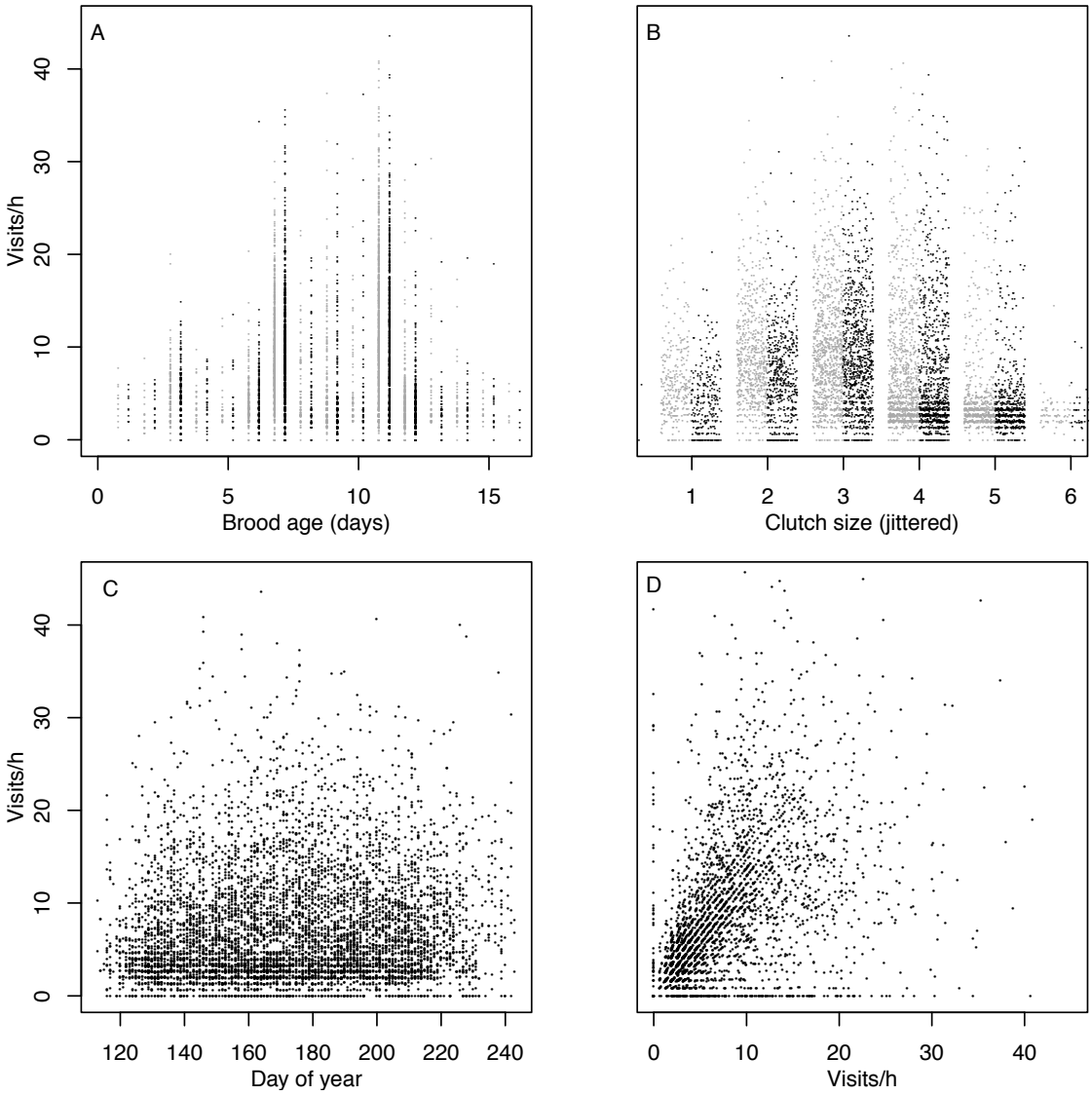

Figure 1: The relationships between parental care (on the x-axis in A, B and C) in Lundy island house sparrows between 2004 and 2015, and: (A) brood age (in days), (B) brood size (grey dots = females, black dots = males), and (C) day of the year (1st May = 121). D shows the parental care of each female house sparrow (y-axis) and their male social partner (x-axis).

Genetic pedigree

We used up to 15 microsatellite markers and methods (Dawson et al. 2012) detailed in the supplement to construct a full pedigree, spanning 1989–2015 and containing 8546 individuals (Table S1). Our genetic pedigree is near complete for the years 2000–2015 (Schroeder et al. 2015).

We pruned our genetic pedigree to include only individuals that are informative for parental care. We considered individuals as informative if they themselves were either phenotyped or were related to two or more phenotyped individuals. This pruned pedigree contained 1018 individuals.

147 These individuals in the pruned pedigree are more than those we have phenotypic data for (613)
148 because the ancestors of phenotyped individuals are kept in the dataset. These individuals, while not
149 phenotyped, are informative in a quantitative genetic analysis by their genetic relatedness, –
150 because by decent, they connect at least two phenotyped individuals. We also used the genotypic
151 data, and subsequent parentage analysis, to assess the immigration rate to Lundy, which was 0.5%,
152 or five individuals, of all breeding birds between 2000 and 2015 (Schroeder et al. 2015).
153
154 ***Statistics***
155 We used standard exploratory data analysis and graphs to test for violations from the assumptions of
156 regression analyses (Zuur et al. 2010). We present results from Gaussian REML models, but we also
157 tested for the robustness of these results using Poisson PQL models, which led to the same
158 conclusions qualitatively. We ran a general linear model to confirm the covariates and factors that
159 we know to be biologically relevant based on previous findings (Nakagawa et al. 2007a; 2007b;
160 Schroeder et al. 2012b; 2013, 2016, see supplement for more details). We then proceeded to run
161 animal models using the so-decided fixed effect structure. For the animal models, we used all
162 observations, including observations of birds that were socially monogamous over their entire
163 lifetime. Even though the latter observations do not add power to the estimation of the social
164 environmental effects, they increase the power for estimating the indirect and direct genetic effects.
165 All models were run in R 3.3.3 (R Development Core Team 2017). We used the function
166 ASReml from the package ASReml-R version 3.00 for variance partitioning (Gilmour et al. 2009).
167 For the animal models, we calculated p-values for variance components via likelihood ratio tests,
168 using -2 times the difference in log likelihoods. This test statistic was then compared to a 50:50 c^2
169 distribution of $\frac{1}{2}\ c^2(q-1) + \frac{1}{2}\ c^2(q)$, where q is the difference in the number of random effects in
170 the compared models (Vischer 2006), because significance testing of variance components with log-
171 likelihood ratio tests may be overly conservative (Wilson et al. 2010).

172

173 ***Univariate animal models***

174 House sparrow males are more predictable care-givers than females (Nakagawa et al.
175 2007a), and react flexibly to the identity of their female partner (Schroeder et al. 2016). We
176 therefore first ran animal models separately for each sex. The model procedure was the same for
177 both sexes, with respectively the male and the female trait as response variable. We modelled the
178 individual identity of the caregiver as a random effect: the individual direct effect (V_d), where d
179 stands for *direct effects*. We then iteratively added random effects, and tested their significance
180 using likelihood ratio tests. We first partitioned V_d into the variance due to direct *additive genetic*
181 *effects* (V_{Ad}), and *direct permanent environment* effects (V_{PEd}). We did this by including the identity
182 of the focal individual as two separate random effects, one of which we linked to a pedigree-based
183 relatedness matrix to estimate V_{Ad}. We then added a random effect of the identity of the social
184 partner (V_s) to test for the presence of any *social effects* – we indicate any social indirect effects
185 with an index s. We then, if the social effect (V_s) was detected, partitioned it into the IGE (V_{As}),
186 which is the variance of the social effect explained by additive genetic effects. We also fitted a
187 *permanent social environment effect* (V_{PEs}). This was done in a similar way as we partitioned V_d:
188 we included the identity of the social partner as separate two random effects, one of which was
189 linked to the pedigree-based relatedness matrix to estimate the IGE. We then modelled the
190 covariance between the direct genetic effect by the caregiver, and the IGE by the social mate on the
191 trait of the caregiver ($COV_{Ad,As}$). This covariance is estimable because the genes that males carry (or
192 the polygenic combination) and that code for the IGE on the female trait in the male individuals are
193 inherited to daughters, too. We then tested if this intersexual genetic covariance $-COV_{Ad,As}-$ was
194 significantly different from zero by testing a model where the covariance could take on any value,
195 against a model in which the covariance was fixed to zero. We used these univariate models to
196 calculate sex-specific repeatability (R_f and R_m), heritability (h^2_f, h^2_m), and sex-specific total

heritable variation (t^2_f, t^2_m, for how to calculate those see below).

Note that from here on throughout this work, we use sex-specific notation that reflects the sex of the focal individual in which the trait was measured. Hence, V_{Adf} refers to the direct genetic effect in the female trait (i.e. the additive genetic effect of the female on parental care provided by herself), and V_{Asf} refers to the IGE in the female trait (i.e. the IGE induced by the male partner on the parental care provided by the female). Similarly, V_{Adm} refers to the direct genetic effect in the male trait, and V_{Asm} refers to the IGE in the male trait.

Bivariate animal models

The direct and social effects apparently differed between models for the sexes. We tested whether these differences could be considered statistically significant, for which they needed to be estimated in the same model. Therefore, we constructed a bivariate animal model, where male and female parental care that were observed at the same occasion were modelled as a bivariate response (Teplitsky et al. 2010). To test the statistical significance of the sex specificity, we compared models in which the parameter estimates of each respective variance component for both traits were conditioned to be equal (i.e. $V_{Adf} = = V_{Adm}$, and $V_{Asf} = = V_{Asm}$) with models in which the estimates were allowed to take on any values. We modelled heterogeneous residual variances because the univariate models suggested these to be different, and we modelled residual covariance where that did not prevent model convergence.

The data for these bivariate models testing sex-specificity were coded such that each observation, of the male or the female parental care, was on separate lines, with columns for the identity of the focal individual, its parental care, its sex, the identity of its social partner, and the trait measured in the social partner (entered as 'NA' as this cannot be recorded - one cannot record male provisioning rate in females, or vice versa) along with columns for the fixed effects. This data structure allows testing the statistical significance of sex-specificity.

We then estimated the covariances between the direct and social genetic effects, and the covariances within individuals in the non-genetic components. For the estimation of all covariances we re-coded the data again, such that the observations for the female trait and the male trait, which were made at the same time on the same brood, were on the same line, with separate columns for female identity and male identity. This model however cannot formally test for sex-specificity, but since we have shown the sex specificity before, we assumed sex specificity in this model. We validated the model by comparing the variance components between overlapping models of this data structure with the one testing for sex-specificity, and the parameter estimates were quantitatively similar (two to three decimal places). We assessed the covariances between all genetic components. The covariance structure for the permanent environment effects (V_{PEdf} and V_{PEdm}), and the social environmental effects (V_{PEsf} and V_{PEsm}) is constrained compared to that of the genetic effects, because not all combinations exist on the phenotypic level. Only the covariance between an individual's permanent direct individual effect (e.g. V_{PEdf} for females in the female trait), and its permanent social individual effect on the trait expression in a partner (e.g. V_{PEsm}) can be estimated ($COV_{(PEDf,PESm)}$ and $COV_{(PEdm,PEsf)}$) because it occurs in the same observation and take place at the same time. However, as the estimate of V_{peSf} was zero or very close to zero in all models, $COV_{(PEdm,PEsf)}$ was not estimable and fixed to 0. We calculated genetic correlations as $r_g = \frac{COV_g}{\sqrt{V_{g1}+V_{g2}}}$.

We used the best of these bivariate models with all statistically significant covariances as detailed above as the final model to calculate the total heritable variation.

Quantitative genetic parameters

We calculated repeatability (R) as

(1) $R = \frac{V_{Ad}+V_{PEd}}{V_P}$,

and heritability (h^2) as

(2) $h^2 = \frac{V_{Ad}}{V_P}$

where V_P is the total phenotypic variance in parental care.

Our study treats female and male parental care as different traits, each only expressed in one sex, causing some difficulty in summing the genetic effect within individuals, because a female social effect is an effect on a male trait, and vice versa. However, females pass on their genes to their sons, and males to daughters. If PC is the sum of the parental care that a brood experienced, from both the female and the male parent, we can sum these effects (following Bijma 2011) and PC is (Bijma, pers. comm.):

(3) $PC = A_{df} + A_{sf} + A_{dm} + A_{sm} + e$

$A_{df} + A_{sf}$ describe the genetic contribution to the female trait, where A_{df} is the direct genetic component of the female and A_{sf} is the social genetic effect. We estimated the latter effect from the identity of the female's social partner – because these males pass their genes on to their daughters, this can also be estimated among females. $A_{dm} + A_{sm}$ describes the genetic contribution to the male trait, where A_{dm} is the direct genetic component of the male and A_{sm} is the social genetic effect of the partner on the male trait. Again, this can also be estimated among males, because the female partner also passes on her genes to her sons. The term e represents residual effects. Therefore, the genetic mean trait value of total parental care contains terms for both sexes, and applies to individuals irrespective of sex because they receive genes from both their parents. Therefore, the total breeding value (A_T, Bijma 2011) for individual j, be it male or female, is

(4) $A_{T,j} = A_{df,j} + A_{sf,j} + A_{dm,j} + A_{sm,j}$

When we consider female and male provisioning to be different traits, however, we define the total breeding values for these traits separately as

(5) $A_{Tf,j} = A_{df,j} + A_{sf,j}$

(6) $A_{Tm,j} = A_{dm,j} + A_{sm,j}$

These are breeding values that refer to each specific trait. We calculated the population-wide parameter, the total heritable variation on which selection can act (here exemplary on female care), V_{Atf} , within a single-sex trait as

(7) $V_{Atf} = V_{Adf} + 2COV_{(Adf,Asf)} + V_{Asf}$

and the respective quantitative genetic parameter for the proportion of total heritable variation for the female trait t_f^2 , where t^2 is analogous to h^2, as

(8) $t_f^2 = \frac{V_{Atf}}{V_{Pf}}$

Where V_{Pf} is the total phenotypic variance in the female trait. We then calculated male t^2_m similarly. For this, we used the parameter estimates of the univariate models.

We then estimated the proportion of total heritable variance in the parental care that the brood receives, t^2, from the final bivariate model:

(9) $t^2 = \frac{V_{At}}{V_P}$.

where

(10) $V_{At} = V_{Adf} + 2COV_{(Adf,Asf)} + V_{Asf}$

$+2COV_{(Adf,Adm)} + 2COV_{(Asf,Adm)} + V_{Adm}$

$+2COV_{(Adf,Asm)} + 2COV_{(Asf,Asm)} + 2COV_{(Asf,Adm)} + V_{Asm}$

$= V_{Atf} + 2COV_{(Atf,Atm)} + V_{Atf}$

We aimed to contrast the estimates of total heritable variation with the ones for heritability and repeatability from models not including social effects. Therefore, we estimated heritability of the female and male trait (h^2_f and h^2_m respectively) from the univariate models containing the direct genetic effect, but no social effect. We then estimated t^2_f and t^2_m from the univariate sex-specific models that included social effects and a genetic covariance. We calculated the overall total

heritable variation t^2 from the best supported bi-variate model including genetic and phenotypic covariances.

Results

Phenotypic data

On average, sparrows visited their broods 7.31 times per hour (95% CI: 0.72–51.32). Parental care increased in both sexes with the age of the brood and decreased after chicks reached an age of 11 days (Fig. 1A). Parental care increased with the number of chicks in a brood and plateaued at a brood size of three (Fig. 1B). Time of day and day of season (Fig. 1C) were not associated with nest box visit frequency. Male and female nest box visit frequencies, measured at the same nestbox and at the same time, were positively correlated with each other (Fig. 1D, correlation coefficient: 0.46, 95% CI: 0.43–0.49, $P < 0.0001$).

The female trait

Generally, a lot of phenotypic variance (26.96±0.69 SE) was left unexplained. However, while the amount of variance explained by direct genetic effects was small (5.00±0.73 SE, Table 1, females, model 2), it was larger than the permanent environment effect and it improved the model fit over a model without additive genetic variance (Table 1, females, model 2 *vs.* 1).

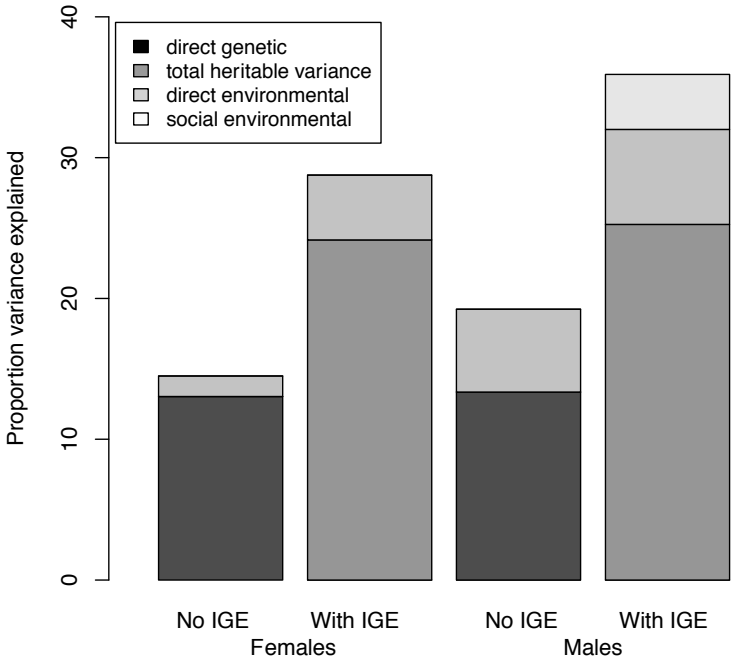

Figure 2: The proportion of variance explained in female and male parental care. The total height of the bars of models with No IGE represents the conventionally calculated repeatability. The bars with the IGE considered were calculated from models 6 in Table 2, the models without IGE from models 2 in Table 2.

Adding the social effect improved the fit of the model, but also lead to a decrease in direct additive genetic variance (Table 1, females, model 3 *vs.* model 2). However, the social effect was due to a social genetic effect (2.75±0.68 SE), which when included significantly improved the model fit (Table 1, females, model 4 *vs.* 3). Excitingly, the genetic covariance between the additive genetic variance and the indirect genetic variance component in female parental care was positive and improved the fit of the model (Table 1, females, model 6 *vs*. 5), leading to a high genetic correlation (0.65±0.41). The repeatability from the model that does not account for social effects (Table 1, model 2) was 15%, this would be considered the upper limit of the heritable variation (Lynch & Walsh 1998). The heritability calculated from this model ignoring social effects (Table 1, model 2), was 13% (Fig. 2). The total heritable variation was considerably larger when taking the IGE and the genetic covariance into account (model 6, female trait). Heritable variation explains 24% of all variation in female parental care (Fig. 2), which is larger than the traditionally calculated

repeatability.

The male trait

In male parental care, there was significant additive genetic variance (4.65±1.57 SE Table 1, males, model 2), compared to the residual variance which was similar to the female trait (28.12±0.72 SE). The social individual effect similarly improved the model fit (Table 1, males, model 3 *vs.* model 2). However, a permanent environment effect remained and most of the variance was drawn from the residual variance and did not affect the additive genetic estimate (Table 1, males, model 3 vs. model 1). A statistically significant social genetic, and social permanent environment effect were detected (Table 1, males, model 4 vs model 3). The genetic covariance between the direct and the social genetic was positive and marginally significantly improved the model fit, with a genetic correlation of 0.55 ± 0.49 SE. The repeatability of a model without social effects (Table 1, males, model 2) was R_m = 19%, and the heritability calculated from this model ignoring the IGE was 13% (Fig. 2). The total heritable variation in the male trait was considerably larger when taking the IGE and the genetic covariation into account (Table 1, males, model 6). Heritable variation explains 25% of all variation in male parental care (Fig. 2).

Bivariate animal model

We used a bivariate animal model to formally assess the sex-specificity of effects, which can only be done in the presence of both the male and the female trait. The results of the bivariate animal model reflect the patterns found in the univariate, sex-specific analyses (Table 2). All sex-specific covariance estimates significantly improved the model fit (Table 2), and thus we decided to go forward with a model that was completely sex-specific.

We then tested for covariances in a bivariate model in which all genetic, phenotypic and residual covariances could be estimated. We built the model by first introducing the covariances that we knew were important from the univariate models ($COV_{aDf,aSf}$ and $COV_{aDm,aSm}$, Table 3,

models 2 and 3). Both covariances were confirmed in the bivariate model. We then modelled the between-sex genetic covariance of the direct effect ($COV_{aDf,aDm}$), which was confirmed and positive (1.46±0.76SE; Table 3, model 4). We then tested for genetic covariance between the IGEs ($COV_{aSf,aSm}$), which was not distinguishable from zero (Table 3, model 5), and we therefore in the following models fixed this component to zero. We then tested for the intra-sexual genetic covariances ($COV_{aDf,aSm}$ and $COV_{aDm,aSf}$). A model that estimated the former did not converge (Table 3, model 6), however, the covariance between the direct additive genetic effect of a male on its parental care, and that of a male on its partners parental care, was different from zero and positive (Table 3, model 7). The permanent environment covariance between the direct effect in the female trait and the indirect effect of the female on the male trait was positive ($COV_{peDf,peSm}$, Table 3, model 8). As expected, a model estimating the covariance between the male direct permanent environment and the female indirect environment $COV_{peDm,peSf}$ effect did not converge, likely because the latter was estimated near zero in nearly all models.

Finally, we calculated the total heritable variation in bi-parental care from this final bivariate model (Table 3, model 8, variance components in Table 4). This total heritable variance was $t^2 = 0.19$.

Discussion

We confirmed the presence of IGEs in biparental care interactions in a wild passerine bird. IGEs were found in both the female and the male trait, but only the social environmental effect was sex-specific. In fact, the heritable variation was similar in both traits.

Direct genetic effects were small, yet the estimate of total heritable variation in female and male parental care was similar at 24 and 25% respectively. We found environmental social effects in the male trait, but the variance estimate for the female trait was near-zero. We found surprisingly robust evidence for positive genetic correlations between the direct male and female effect (inter-sexual

direct genetic correlation), between the direct female and indirect female effect, the same in males (inter-sexual direct-indirect genetic correlation), and between the direct male effect and the female indirect effect (intra-sexual genetic correlation). It is possible that these positive genetic correlations may reinforce our main conclusion: that a cooperative trait like parental care can evolve through selection pressures on a partner.

We found evidence supporting the idea that IGEs on parental care in wild populations exist, and that there are genetic correlations between and within the sexes. Optimization theory in behavioural ecology predicts indeed that the sexes will differ in the amount of care that they provide. Even more, this may be a potential solution to sexual conflict, where usually females are expected to manipulate the amount of care provided by a male (Alonzo et al. 2010; Lessells & McNamara 2012, Schroeder et al. 2016). Most behavioural ecology models assume some sort of flexible, short-term negotiation or a sealed-bid model (Houston & Davies 1985; McNamara et al. 1999; Houston et al. 2005). Our data suggest that a combination of individual quality and mate behavioural compatibility may be important and that this link is, at least partly, genetic. This, in addition to a sealed bid model where male care is phenotypically flexible, is likely governing house sparrow parental care (Schwagmeyer et al. 2002). Males being phenotypically flexible, yet predictable among pairs, may allow females to choose good carers (Nakagawa et al. 2007a), but also allows male to flexibly adjust to partner changes (Schroeder at al. 2016). This mechanism may have depleted the heritable variation which may be evidenced by the large amount of residual variation.

Our results suggest that it is possible that females and males may adjust their care in relation to the genetic quality rather than the phenotype of care of their mate, and so perhaps compensate for poor-quality partners. In addition, we find support for our previous finding that males phenotypically adjust their paternal care to the female they are mated with (Schroeder et al. 2016). Most theoretical models explaining the evolution of parental care treat female and male care behaviour as the same trait (Royle et al. 2012). Relaxing this assumption in theoretical models may

enable novel insights.

The finding that parental care is partly determined by the genetics of the social partner is especially interesting in a trait with mutual consequences for the interacting partners, since not only will an individual pass on its genes to its offspring, but the genes of the mating partner will also be passed on to the offspring. IGEs in social traits between mating partners may therefore be responsible for the maintenance of genetic variance in traits that affect their mutual fitness (Miller & Moore 2007; Kotiaho et al. 2008). This might be one reason for why we fail to find a clear response to selection in wild populations (Pujol et al. 2018). Clearly, we need to develop testable hypotheses, including IGEs also in the context of fitness to fully understand how parental care can evolve (Kotiaho et al. 2008). As we have evidence for heritable variation in fitness in the same population (Schroeder et al. 2012a), some of the recently developed theory (Fisher & McAdam 2018) may be testable in this study system.

Finally, it is often suggested that repeatability presents an upper limit to narrow-sense heritability (Lynch & Walsh 1998). While this relationship is often true, this rule of thumb has to be treated with care in the presence of strong IGEs. Our results show that accounting for IGEs can uncover a wealth of quantitative genetic architecture and explain missing heritability that may otherwise remain undetected. In conclusion, IGEs and genetic correlations between unrelated individuals exist in wild populations and may contribute to the maintenance of variance and evolution of life-history traits.

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Table 1: Univariate animal model of female and male house sparrow parental care visits to their chicks. Variance components with a star* were constrained and fixed to zero. Random terms refer to the indices of variance components. P-values indicate whether the first model number in the LRT column provided a better fit to the data versus the second model number.

Model	Random terms	V_{aD} SE	V_{aS} SE	$COV_{(aD,aS)}$ SE	V_{peD} SE	V_{peS} SE	V_R SE	LogL	LRT df =1	P
	females									
1	V_{peDf}				**5.00** **0.73**		**28.20** **0.71**	-7612		
2	$V_{aDf} + V_{peDf}$	**4.30** **1.19**			0.48 0.77		**28.22** **0.71**	-7591	2 vs. 1	<0.001
3	$V_{aDf} + V_{peDf} + V_{peSf}$	**3.89** **1.21**			0.21 0.84	**2.36** **0.56**	**27.02** **0.69**	-7568	3 vs. 2	<0.001
4	$V_{aDf} + V_{aSf} + V_{peSf} + V_{peDf}$	**2.81** **1.10**	**2.76** **0.68**		0.90 0.84	0.00 0.00	**26.96** **0.69**	-7561	4 vs. 3	<0.001
5	$V_{aDf} + V_{aSf} + COV_{aDf,aSf} + V_{peSf} + V_{peDf}$	**2.81** **1.10**	**2.76** **0.68**	0.00*	0.90 0.84	0.00 0.00	**26.96** **0.69**	-7561		
6	$V_{aDf} + V_{aSf} + COV_{aDf,aSf} + V_{peSf} + V_{peDf}$	**2.10** **0.98**	**2.75** **0.68**	**1.56** **0.71**	1.52 0.84	0.00 0.00	**26.70** **0.69**	-7559	6 vs 5	0.02
	males									
1	V_{peDm}				**6.75** **0.91**		**28.09** **0.72**	-7428		
2	$V_{aDm} + V_{peDm}$	**4.65** **1.57**			2.05 1.13		**28.12** **0.72**	-7416	1 vs. 2	<0.001
3	$V_{aDm} + V_{peDm} + V_{peSm}$	**4.35** **1.58**			1.36 1.17	**3.80** **0.77**	**26.41** **0.69**	-7382	2 vs. 3	<0.001
4	$V_{aDm} + V_{aSm} + V_{peSm} + V_{peDm}$	**2.98** **1.39**	**2.93** **1.33**		2.26 1.18	**2.93** **1.33**	**26.41** **0.69**	-7378	4 vs 3	0.003
5	$V_{aDm} + V_{aSm} + COV_{aDm,aSm} + V_{peSm} + V_{peDm}$	**2.98** **1.39**	**2.93** **1.33**	0.00*	2.26 1.18	1.19 1.09	**26.41** **0.69**	-7378		
6	$V_{aDm} + V_{aSm} + COV_{aDm,aSm} + V_{peSm} + V_{peDm}$	**2.96** **1.33**	**2.72** **1.25**	**1.56** **0.86**	**2.35** **1.14**	1.36 1.04	**26.40** **0.69**	-7376	6 vs. 5	0.053

Table 2: Variance component estimates, standard errors (SE), and likelihood ratio tests (LRT) for bivariate models of male and female house sparrow parental care visits to chicks, testing for sex specificity of variance components. LRT = likelihood ratio test, where the numbers refer to the model number.

Model	V_{aDf} SE	V_{aDm} SE	V_{aSf} SE	V_{aSf} SE	V_{peDf} SE	V_{peDm} SE	V_{peSf} SE	V_{peSm} SE	V_{Rf} SE	V_{Rm} SE	LRT	P
1		**2.25** **0.72**		**1.67** **0.65**		**2.17** **0.62**		**1.52** **0.57**	**26.74** **0.68**	**26.65** **0.69**		
2		**2.21** **0.71**		**1.72** **0.66**	**1.38** **0.70**	**3.12** **0.89**		**1.46** **0.57**	**26.87** **0.69**	**26.53** **0.69**	2 vs. 1	0.04
3	**2.26** **1.03**	**2.66** **1.31**		**2.18** **0.71**	**1.22** **0.87**	**2.60** **1.16**		**1.26** **0.58**	**26.87** **0.69**	**26.52** **0.69**	3 vs. 2	0.005
4	**2.31** **1.01**	**2.66** **1.33**		**2.27** **0.72**	**1.23** **0.85**	**2.52** **1.18**	**0.39** **0.64**	**2.17** **0.83**	**26.99** **0.69**	**26.36** **0.69**	4 vs. 3	0.03
5	2.81 2.96	**2.98** **1.39**	**2.76** **0.66**	**2.93** **1.33**	0.89 0.84	2.26 1.18	0.00 0.00	1.19 1.09	**26.96** **0.69**	**26.41** **0.69**	5 vs. 4	0.04

Table 3: Model selection with covariances and corresponding standard errors. A starred value means it was constrained to 0, and a u represents a covariance that was allowed to take on any value in a model that did not converge. The reported p result from likelihood-ratio tests (comparisons indicated in the LRT column), testing with one degree of freedom against a model in which the covariance in question was fixed to zero and thus not estimated.

	(V_{aDf}, V_{aSf})	(V_{aDm}, V_{aSm})	(V_{aDf}, V_{adm})	(V_{aSf}, V_{aSm})	(V_{aDf}, V_{aSm})	(V_{aDm}, V_{aSf})	(V_{peDf}, V_{peSm})	(V_{rf}, V_{rm})	LRT	p
1	0.00*	0.00*	0.00*	0.00*	0.00*	0.00*	0.00*	**11.37** **0.52**		
2	1.22 0.67	0.00*	0.00*	0.00*	0.00*	0.00*	0.00*	**11.37** **0.52**	2 vs 1	0.043
3	1.25 0.67	1.64 0.86	0.00*	0.00*	0.00*	0.00*	0.00*	**11.35** **0.52**	3 vs 2	0.02
4	1.03 0.64	1.12 0.83	1.46 0.76	0.00*	0.00*	0.00*	0.00*	**11.39** **0.52**	4 vs 3	0.03
5	1.03 0.65	1.11 0.82	1.46 0.76	0.006 0.00	0.00*	0.00*	0.00*	**11.39** **0.52**	5 vs 4	0.99
6	u	u	u	0.00*	u	0.00*	0.00*	u		Not converging
7	**1.51** **0.66**	0.91 0.78	1.84 0.78	0.00*	0.00*	**1.46** **0.60**	0.00*	**11.23** **0.69**	7 vs 4	0.006
8	**1.53** **0.64**	0.96 0.77	1.84 0.76	0.00*	0.00*	**1.39** **0.61**	0.85 0.48	**11.07** **26.30**	8 vs 7	0.032

Table 4: Final model (Table 3 Model 8) variance/covariance matrices. On the diagonal are the variances, on the upper off-diagonal the covariances, on the lower off-diagonal the correlations. This is the model from which we calculate quantitative genetic parameter estimates. NAs refer to impossible covariances.

	aDf	aDm	aSf	aSm			peDf	peDm	peSf	peSm			Rf	Rm
aDf	**1.72** **0.86**	**1.84** **0.76**	**1.53** **0.64**	0.00*		peDf	**1.67** **0.81**	NA	NA	0.85 0.48		Rf	**26.96** **0.69**	**11.07** **0.52**
aDm	0.83 0.21	**3.14** **1.29**	**1.39** **0.61**	0.96 0.77		peDm	-	**2.85** **1.09**	0.00*	NA		Rm		**26.30** **0.68**
aSf	0.75 0.31	0.77 0.29	**2.45** **0.94**	0.00*		peSf	-	-	0.31 0.72	NA				
aSm	-	0.54 0.50	-	2.33 1.17		peSm	0.47 0.55	-	-	1.56 1.04				

Supplementary material

Contents:

Supplementary methods

Genetic pedigree construction

We genotyped individuals that were DNA-sampled (N = 8546) for up to 15 microsatellite loci (Dawson et al. 2012). A summary of the pedigree is available in Table S1 (Morrissey & Wilson 2009). With these data we aimed to determine the genetic ancestry of every genotyped bird, and extended the pedigree detailed in (Schroeder et al. 2012, 2015) to include all years from 1989–2015, and fill in gaps where needed. The pedigree is near-complete for all observed breeding attempts for the years 2000–2015, but we also used sparser data from birds (N = 609) caught before 2000 (Griffith et al. 1999). The dataset pre-2000 was incomplete because DNA samples were only taken in two years, however these data were also used in the parentage analysis, for example some of these birds bred in 2000 and later.

Genotyping quality control

We exemplary analysed the genotyping quality of the data collected in 2012, using samples from 682 samples (437 birds) collected in winter 2011/12 and summer 2012. Thirty-nine of these samples were from birds that were sampled and genotyped previously, and three of those mis-matched their existing genotypes from older, and previously genotyped samples at more than one loci. We assigned these mixed-up samples to natal or cross-fostered siblings of the alleged sampled individual, using identity analysis in CERVUS 3.0 (Marshall et al. 1998; Kalinowski et al. 2007).

Of these 437 individuals, 245 were sampled twice in this 2012 dataset only. Of those 245 repeated genotypes, four did not match each other. Identity analysis confirmed the natal or cross-fostered sibling to which these samples belonged. Identity analysis was then run on the genotyped samples and did not detect more mixed up samples. We used similar analyses on our complete dataset to detect and correct mix-ups, field errors, and lab errors as best as possible. When in doubt, we decided to err on the side of caution and only included unambiguous data. We used individuals that were repeatedly genotyped (284 birds) for a quality control analysis of genotyping error, using the software Pedant 1.0 (Bonin et al., 2004). The rates of genotyping error and dropout rates are presented in Table S1.

Parentage assignment

We used CERVUS 3.0 (Marshall et al. 1998; Kalinowski et al. 2007) to assign the genetic fathers to offspring, and to confirm the genetic mothers. In the parentage simulations, we set the genotyping error rate to 0.01. We set the percentage of genotyped mothers and fathers to 95%, which is a conservative assumption given that we have high resighting probabilities (mean annual resighting probability between 94% and 96% (Schroeder et al., 2011, Simons et al., 2015). We first confirmed that the social (observed) mother was the genetic mother, allowing maximal two mismatched loci. We then ran a parentage analysis where we constrained the genetic mothers to the confirmed mother, and for birds where this was not known, we allowed the mother to take on any identity of alive female adults. We assigned fathers from the population of live adult males. We defined a bird as adult and alive if it was born in a previous year and was seen up to 12 months before April of the year that was analysed. This means that we allowed all first-year birds to be assigned as parents, even if they have not been seen since fledging.

We then took an iterative approach to checking parentage assignments. If more than one parent-pair could be assigned with zero or one mismatch, we examined the resighting history of all

potential genetic parents. In these cases, we assigned parentage according to the following decision rules: 1) If any assigned parent-pair was also the observed social pair with up to one mismatch, we assigned those; 2) If none of the assigned fathers was the known social father, we checked the assigned fathers' resighting histories, and assigned the male that was actually observed to be alive during the focal breeding season. If more than one was known to be alive, we did not assign a father unless rule 3 applied. 3) We checked whether an assigned pair was a known social pair in the year before or after and assigned known social pairs. We always took information of siblings into account, - in case of a tie between two potential extra-pair fathers, we assigned an extra-pair male if it was the extra-pair sire for another sibling in the nest. If none of the above information was available, and more two or more mismatches occurred, we conservatively did not assign parentage.

Of the individuals from 2000-2011 that could not be assigned with fewer than 2 mismatches 77% were rotten eggs, dead embryos, or otherwise compromised samples. In those, DNA may have been severely degraded or present in too low concentration such that less than 6 loci amplified. Of all birds that were sampled and survived to 12 days, on average, from 2000 – 2011, 1.2% annually (range: 0%–4.4%) were not assigned at both parents, mostly due to genotyping errors and/or a lack of DNA/too small sample volume (Hsu et al. 2015, Supplemental Table S2d).

Fixed effects model structure:

We ran a general linear model to determine the fixed effects to include in our animal models. The biologically relevant variables that we considered, based on previous findings (Nakagawa et al. 2007a; 2007b; Schroeder et al. 2012; 2013), were: the age of the chicks when parental care was measured (brood age, in days), the number of chicks present in a brood when parental care was measured (brood size) because both may affect how often parents provide offspring with food (Fig 1). We added the quadratic effects of brood age and brood size, because birds cannot infinitely increase the frequencies of their provisioning visits even if demands increase. We also tested for

effects of the day of the year and the time of day as provisioning activity may vary with those factors. Social fathers may adjust the level of their parental care according to how certain they are of their genetic paternity (Akcay and Roughgarden 2007, Griffin et al. 2013, Schroeder et al. 2016). Since females may also compensate for retaliating males, we added the frequency EPO in the brood as a fixed covariate in our models, and, when investigating male and female care together, an interaction between EPO and sex (Schroeder et al. 2016). For broods where no data on EPO were available, we used the median value. We added a two-level factor (noisy/quiet) for the location of each nest box, because location artificially affects provisioning rate (Schroeder et al. 2012). Parental age has little to no effect on parental care, therefore, we did not add this covariate (Schroeder et al. 2013).

After exploratory analysis, we retained the following fixed effects in our animal models (Table S2): the age of the brood, brood size, the squared effect of both, EPO, location, foster status, sex, the interaction of brood size and sex, and an interaction of sex with EPO.

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Supplementary tables

Table S1: Summary of the full pedigree of the Lundy island house sparrows (1995–2015), and a pruned pedigree in which only informative individuals with respect to provisioning frequency were retained. The pedigree was assembled with genotypic information from up to 15 polymorphic microsatellite markers, see methods for more details.

Quantity	Full pedigree	Pruned pedigree
Records	8.546	1018
Maternities	6.954	971
Paternities	7.086	971
Full sibs	33.334	2144
Maternal sibs	88.765	4279
Maternal half-sibs	55.431	2135
Paternal sibs	101.243	3858
Paternal half-sibs	67.909	1714
Maternal grandmother	6.174	911
Maternal grandfathers	6.381	901
Paternal grandmothers	6.048	892
Paternal grandfathers	6.088	858
Maximum pedigree depth	16	16
Founders	1.413	33
Mean maternal sibship size	13.8	4.26
Mean paternal sibship size	13.1	4.13
Non-zero F	4350	828

F > 0.125	472	29
Mean pairwise relatedness	0.04	0.09

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143 **Table S2:** Parameter estimates (*b*) and 95% credible interval (CI) of the fixed effects in the general
144 linear model explaining provisioning frequency per hour in Lundy island house sparrows. Foster
145 status, location and sex were two-level factors. Extra-pair offspring (EPO) was the proportion of
146 chicks in the clutch that was sired by a male other than the social male.

Fixed effect	*b*	*95% CI*
Intercept	-4.73	-6.00– -3.41
Sex (male)	-2.19	-3.03– -1.43
Brood age	2.04	1.78– 2.29
Brood age x Brood age	-0.11	-0.13– -0.10
Brood size	4.50	3.98– 4.97
Brood size x Sex	0.51	0.29– 0.71
Brood size x Brood size	-0.83	-0.90– -0.75
Location (noisy)	-0.82	-1.13– -0.50
EPO	-0.08	-0.81– 0.66
EPO x Sex	-1.51	-2.55– -0.47
Foster status (fostered)	-0.41	-0.69– -0.13

Random	*Variance*	*95% CI*
Residual	31.95	31.87–34.02

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