# Selection on parental performance opposes selection for larger body mass in a wild population of blue tits

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## Abstract

There is abundant evidence in many taxa for positive directional selection on body size, and yet little evidence for microevolutionary change. In many species, variation in body size is partly determined by the actions of parents, so a proposed explanation for stasis is the presence of a negative genetic correlation between direct and parental effects. Consequently, selecting genes for increased body size would result in a correlated decline in parental effects, reducing body size in the following generation. We show that these arguments implicitly assume that parental care is cost free, and that including a cost alters the predicted genetic architectures needed to explain stasis. Using a large cross-fostered population of blue tits, we estimate direct selection on parental effects for body mass, and show it is negative. Negative selection is consistent with a cost to parental care, mainly acting through a reduction in current fecundity rather than survival. Under these conditions, evolutionary stasis is possible for moderately negative genetic correlations between direct and parental effects. This is in contrast to the implausibly extreme correlations needed when care is assumed to be cost free. Thus, we highlight the importance of accounting correctly for complete selection acting on traits across generations.


## Introduction

Directional selection acting on a trait causes within-generation change in the mean. Given heritable (additive) genetic variation, part of this change should be passed onto the following generation, causing evolutionary change (Lush 1943; Lande 1979; Lande and Arnold 1983). Kingsolver et al. (2001) showed that directional selection on quantitative traits is relatively common, particularly for body size, where positive directional selection predominates (Kingsolver and Pfennig 2004). However, those studies able to measure microevolutionary change often find it absent, despite estimates of selection and inheritance suggesting it should exist (e.g. Milner et al. 1999, 2000; Charmantier et al. 2004). Many hypotheses for this evolutionary stasis have been proposed (Merila et al. 2001) and investigated (e.g. Kruuk et al. 2001; Siepielski et al. 2009; Morrissey and Hadfield 2012; Kingsolver and Diamond 2011),

yet none have satisfactorily explained the patterns seen.

One hypothesis to explain the paradox of stasis is that the focal trait under selection may have antagonistic genetic correlations with other traits, also under selection. This could restrict microevolution (Lande 1979; Lande and Arnold 1983), and evidence for antagonistic genetic correlations between traits has been found in various wild populations (Gratten et al. 2008; Ohno and Miyatake 2007; Morrissey et al. 2012). In many species with extended parental care, body size is determined by both the genes of an individual, and the genes in its parent(s) that determine the amount of parental care provided to them. Under this situation, the type of genetic correlations that act antagonistically become more complex, but a solution is formalised in the Willham model (Willham 1963, 1972). Rather than explicitly considering all traits underlying parental care, the Willham model considers a composite trait, parental performance. This is a value assigned to an individual that represents the effect that individual has (through all aspects of its phenotype) on the body size of its offspring, measured in units of the offspring trait. If negative genetic correlations exist between body size and parental performance, an individual that possesses alleles for a large body would also possess alleles for lower parental performance, reducing the size of offspring they produce. This restricts net change in body size, as selection for increased body size leads to a concomitant decline in parental performance, and therefore body size in the following generation. Willham (1972) showed that when the selection gradient on body size is $\beta_b$, evolutionary change in body size has the form:

$$\Delta\bar{z}_b = (g_b + \frac{3}{2}g_{b,p} + \frac{1}{2}g_p)\beta_b \quad (1)$$

where $g_b$ is the additive genetic variance for body size, $g_p$ the additive genetic variance for parental performance (often called the maternal genetic variance) and $g_{b,p}$ is the additive genetic covariance between the two traits. Consequently, if $g_{b,p}$ is sufficiently negative, the response to selection may become zero despite selection for increased body size. However, for this to occur, the additive genetic correlation would have to be close to -1. Given that the traits underlying parental performance

are likely to be developmentally distinct from body size, and expressed in different life-stages, such an integrated genetic architecture seems unlikely. The genetic correlation is routinely estimated in the field of animal breeding (e.g. Meyer 1992; Robinson 1996) and, although negative, the mean estimate from domestic ungulates, is far from -1 (-0.167 ± 0.026 SE; Wilson and Réale 2006; Räsänen and Kruuk 2007). Estimates from non-domestic populations are far fewer, although they have been obtained for captive populations (Blomquist and Williams 2013), as well as wild animals (McAdam et al. 2002; Wilson et al. 2005) and plants (Thiede 1998; Galloway et al. 2009). Wilson et al. (2005) obtained an estimate of -0.41±0.25 SE for the genetic correlation between birth weight and parental performance in Soay sheep (*Ovis aries*), which is suggestive of a negative relationship, but one that is insufficiently strong to explain evolutionary stasis.

The Willham model (Willham 1972) was developed in an animal breeding context, where the only target of *artificial* selection is the focal trait, body size. Cheverud (1984a) highlighted that *natural* selection is unlikely to operate in such a manner, and that selection is likely to also operate on parental performance. Under these conditions evolutionary change in body size has the form:

$$\Delta\bar{z}_b = (g_b + \frac{3}{2}g_{b,p} + \frac{1}{2}g_p)\beta_b + (g_{b,p} + g_p)\frac{1}{2}\beta_p \quad (2)$$

Importantly, selection on parental performance ($\beta_p$) is likely to be negative - parents are expected to pay a cost, in terms of their own fecundity and survival, when providing care for their offspring (Smith and Fretwell 1974). Through this route, a negative genetic correlation actually facilitates the evolution of increased body size, and the conclusions drawn from the simple Willham model depend critically on the cost of parental care.

Theory generally predicts caring to be costly to the parent (Williams 1966; Trivers 1972; Stearns 1992) and this is supported by a large body of empirical work, showing costs to parents in terms of reduced immunity, increased oxidative stress and predation risk, and depletion of micronutrients (reviewed in Alonso-Alvarez and Velando 2012). These ultimately lead to a reduction in future fecundity (Török

et al. 2004), or decreased survival probabilities (Nur 1984; Dijkstra et al. 1990; Owens and Bennett 1994). In the context of body size, Rollinson and Rowe (2015) demonstrate that the majority of positive selection on body size has been measured in juveniles, and trade-off's exist between offspring body size and parental fecundity, highlighting the cost to parents of producing larger offspring, and lending support to this extension of the Willham model.

Despite the substantial evidence that $\beta_p$ should be negative, currently no attempt has been made to estimate the sign and strength of selection acting directly on parental performance, although costs to individual traits making up parts of this composite have been estimated. In part, this may reflect the statistical difficulty of relating parental performance (which is measured as an effect on offspring phenotype) to the survival and fecundity of the parents. By developing appropriate statistical methods we measure selection on parental performance using measures of body mass, survival and fecundity from a long-term cross-fostering experiment in blue tits (*Cyanistes caeruleus*). We find that selection on maternal and paternal performance is negative. Although we do not yet have the power to estimate the genetic (co)variances for parental performance, we can show that moderate negative genetic correlations between body mass and parental performance could result in evolutionary stasis.

## Methods

This study was carried out on a nest-box population of blue tits, on the Dalmeny estate, Edinburgh, UK. This population consists of 225 boxes on Craigie Hill (grid reference NT156766) and beside the Almond River (NT179758), spaced approximately 30m apart. The data used here were collected from 2010 to 2014, with additional data from 2015 and 2016 used to measure survival. Boxes were visited regularly from early April, such that in the vast majority of cases the first egg of a nest was found on the day it had been laid (see Hadfield et al. 2013a).

## Cross-foster Design

Eggs were cross-fostered between nests in this population, and although the cross-fostering design varied between years this does not affect this study. In 2010-2013, on the day the first egg was laid within a nest, the nest was assigned to a cross-fostering group. Where possible these were groups of three nests, but other group sizes were also used (from 2 to 5). Within each group, eggs were crossed on the day they were laid; the egg in nest A was moved to B, B to C, and C to A. Alternate eggs were crossed, so the first egg was cross-fostered, the second egg was not, the third was, and so on. In 2014 a different design was used, as part of a separate experiment. All nests were assigned into pairs on the day they initiated laying and each pair was assigned into one of three treatment groups. In the first treatment, eggs were crossed in the manner explained above, for the second treatment all eggs were switched between the two nests, and for the third treatment all eggs remained in their nest of origin. When interruptions in the laying sequence happened on a crossing day, the cross was postponed until all nests within a group had laid again. Cross-fostering and nest visits ceased when one of the nests within a group finished laying, or indications of incubation were found two days in a row (i.e. the female was on the nest or the eggs were warm).

During 2010-2011 a further cross-fostering experiment was implemented, wherein single eggs in 114 out of 276 clutches were cross-fostered into nests that were not part of their original group. These eggs were either the first in the laying sequence or close to the end. In half the cases these cross-fostered eggs replaced eggs that were at the opposite end of the laying sequence. These data were not excluded, as treatment had little effect on body mass or survival (Hadfield et al. 2013b). In 2012-2013 a feeding experiment was performed, in which 76 out of 247 nests were food supplemented during egg-laying. This had little effect on any pre-natal aspects of development (*Thomson et al. unpublished*), so we do not expect post-natal effects that would affect the analyses here, therefore these data were also included.

## Data Collection

Nests were checked daily for hatching from around 11 days after the last egg was laid. In the majority of cases (>98%) nests were checked within approximately 24 hours of the first chicks hatching. Chicks which had hatched during this day (day 0) were uniquely marked by clipping tufts of down on their head (and if necessary a toe-nail). Chicks that hatched on subsequent visits (day 1 or 3, no chicks hatched after this point) were also marked. Nests were visited on days 0, 1, 3, 6, 9, 12 and 15 ("nest age") when all chicks were weighed and mortality recorded. Blood samples were taken on day 3 from the medial metatarsal vein, under home office license. On day 9 chicks were ringed. Post-fledging, the identities of any unfledged (dead) chicks were recorded. As data was only used from nests that hatched, 104/113 nests from 2010, 141/160 from 2011, 116/129 from 2012, 99/104 from 2013, and 93/98 from 2014 (553/604 in total) were used in this study.

From day 10 onwards, parents were caught in nest boxes, or if necessary by mist net in front of the box. These adults were identified (if previously caught) or ringed, and blood samples were taken from the ulna vein. Adults were also given colour rings which identified the year in which they were first caught as adults and their sex. The presence of any adults, and the colours of the rings seen (if any) were noted at nest visits during the egg laying and nestling stage.

## Genotyping and Pedigree

Genotypes were obtained for chicks, adults and unhatched (fertile) eggs, for which there were available blood or tissue samples. DNA was extracted using DNeasy Blood and Tissue kit (Qiagen, Hilden, Germany), and genotyped at seven polymorphic microsatellite markers (Olano-Marin et al. 2010). See Hadfield et al. (2013a) for full molecular methods.

The pedigree of sampled individuals was reconstructed using a Bayesian Markov chain Monte Carlo approach, MasterBayes (Hadfield et al. 2006). Each chick had one to three potential mothers, due to the cross-fostering design. Maternity was

restricted to these females – it was assumed that mixed maternity clutches did not occur. Paternity was not restricted, but the odds that a male caught at a nest was also the sire of the chicks originally from that nest was simultaneously estimated with the pedigree. Likewise, we estimated the rate at which the probability of paternity decays with distance between the nests attended by a male and the nest from which the chicks originally came. Where one or both adults were unsampled from a nest, a non-genotyped dummy adult(s) was included in the model. The size of the unsampled male population was also estimated, representing males that gained paternity but were not associated with a specific nest. Individuals that were assigned to unsampled males with greater than 50% posterior probability were assigned into paternal sibships, using rcolony (Jones and Wang (2010); see Hadfield et al. (2013a)).

## Assignment of Social Parents

In order to assess selection on parental performance, the social parents (i.e. the care-givers) at each nest need to be known. Any individual caught at a nest was assumed to be the social male/female for that nest. In this manner we assigned social mothers to 493/553 nests and fathers to 413/553 nests. The remaining females were generally not caught because they deserted their chicks before day 10, and so were assigned dummy identities. The genetic sire with the largest proportion of paternity in a nest was assigned as the social father for the remaining 140 nests where a male was not caught. This could be either a male caught at a different nest that year, or an unsampled male with a dummy identity. In 7 cases two or more males tied, and so a (new) dummy male was assigned to be the social father. Had this method been used to assign a social father in nests where a male had been caught, the correct male would have been assigned in 92% of cases.

The number (and colour rings if possible) of adult birds present at a nest were recorded during nest visits. This allowed us to generate a score of male attendance at each nest, which provided a measure of how much care a male was likely to be giving to his offspring – males with higher nest attendance were expected to be providing more care. In order to avoid bias, we restricted this to visits made after

the first egg had been laid in a nest, as nests commencing laying later in the season were visited more times in total than those that lay early, and so would have greater sampling effort if visits prior to laying were included. We scored male attendance as a 0 if no male had ever been seen, 1 if a male had been seen but was not caught (and therefore providing no, or very little care), and 2 if the male had been caught (therefore providing care, as adults were caught when they were in the nest). There were 31, 109, and 413 nests in each of these categories. In the models for chick traits, the male attendance score for the nest-of-rearing was fitted. In the models for adult male fitness components, an individual's average male attendance score over nests in that year for which he was assigned as a social father was used.

The average rate of extra-pair paternity in this population is 18.4%, although this varies depending on the male attendance levels – 17.2% where males have been caught, 21.2% where males have been seen but not caught, and 28.9% where no male has been seen. In addition, 16.7% of males are polygamous (social male at more than one nest), and of these, 21.8% have been caught at both nests at which they are social father.

## Statistical Methods

In order to assess selection occurring through the effects of parental performance on adult survival, the effect of mass on juvenile survival, and the effect of parental performance on fecundity, we ran three models which were conditionally independent.

*Model 1*: The first model was a bivariate model with age-specific body mass and annual adult survival as response variables. The body mass model included 25915 records taken across the 7 nest ages at which weights were taken for 4345 chicks in 553 nests. Eight nests (57 chicks) were excluded because they were found more than one day after chicks had hatched, such that the nest ages could not be determined. Body mass was treated as a Gaussian response, with the body mass of individual $i$ on day $d$ of year $y$ of the form:

$$b_{iyd} = \mathbf{x}^{(b)}_{iyd}\boldsymbol{\beta}^{(b)} + \mathbf{z}^{(b)}_{iyd}\boldsymbol{u}^{(b)} + (\beta^{(b)}_{f} + d\beta^{(b)}_{f:d}) \cdot f_{ny} + (\beta^{(b)}_{c} + d\beta^{(b)}_{c:d}) \cdot c_{sy} + m_{nyd} + p_{syd} + e^{(b)}_{iyd} \quad (3)$$

where $\boldsymbol{\beta}^{(b)}$ are generic fixed effects and $\mathbf{X}^{(b)}$ their associated design matrix. These are variables that affect mass and/or fitness, but which are not the focus of the selection analysis, and include nest age (as a factor), sex, day of hatching within the nest (factor, hatched on day 0, 1 or 3), year, time of day, and overall nest hatching day (days from April 1st). Although our selection estimates remain valid if these variables are heritable and genetically correlated with body mass and/or parental performance, the equations developed for evolutionary change (see SI) would have to be modified to take this into account. Interactions between all terms and continuous nest age were included to capture trends in these effects over ontogeny (see Hadfield et al. (2013a)). We include the effects of female fecundity and male nest attendance as specific traits that form part of parental performance (see McAdam et al. 2014; Hadfield 2012, for discussion of "hybrid" approaches between classical trait-based and variance-partitioning analysis). $\beta^{(b)}_{f}$ is the main effect of female fecundity ($f_{ny}$; clutch size of nurse/social mother $n$) on body mass and $\beta^{(b)}_{f:d}$ determines how this changes with the nest age of chicks ($d$). $\beta^{(b)}_{c}$ and $\beta^{(b)}_{c:d}$ are equivalent terms for male attendance ($c_{sy}$), which is fitted in all cases as a continuous covariate; subscript $s$ denotes social father. $\mathbf{u}^{(b)}$ are generic random effects and $\mathbf{Z}^{(b)}$ their associated design matrix; these were a nest-of-origin effect, which captures prenatal maternal effects, and the genetic effect, estimated using pedigree information. The age specific random effects were assumed to be drawn from a multivariate normal distribution parameterised by a 7x7 covariance matrix (representing the seven days on which weights were taken). In both cases this covariance matrix was approximated using a lower dimensional first-order antedependence structure similar to the autoregressive structure used in Hadfield et al. (2013a), and shown to outperform alternative techniques such as random regression. The six lagged regression coefficients were allowed to vary over age, as were the seven innovation variances. (See SI for implementation details).

$m_{nyd}$ and $p_{syd}$ are a third set of random effects, hereafter referred to as parental

effects, associated with the identities of the social parents at a nest, within a year, and together capture the variance in body mass at each age due to nest-of-rearing. These were fitted as a multimembership model such that the variance of the two effects (male and female) were assumed equal. A small amount of information exists to separate nest-of-rearing effects into nurse and social father effects, as some males are social sires for more than one nest per year. This model would only be weakly identifiable, so we chose to use a multimembership model that explicitly assumes equal division of nest-of-rearing effects. The 7x7 covariance matrices for these effects were unstructured; We denote them as $\boldsymbol{V}_m = \boldsymbol{V}_p$. The covariance structure for the residual mass effects ($e^{(b)}_{iyd}$) were also unstructured.

Annual adult survival was modelled as an event history characterised by a series of 1's (years for which the individual survived) followed by a 0 (the year the individual was not observed) or a 1 (individuals surviving past 2016). The binary outcome was modelled using a threshold model (887 survival events from 552 individuals), with survival from year $y$ to year $y+1$ of the form:

$$a_{iy} = \mathrm{I}\left(\mathbf{x}^{(a)}_{iy}\boldsymbol{\beta}^{(a)} + \delta_i\left[\beta^{(a)}_f \cdot f_{iy}\right] + (1-\delta_i)\left[(\beta^{(a)}_c \cdot c_{iy}\right] + e^{(a)}_{iy} > 0\right) \tag{4}$$

where I is the indicator function. Generic fixed effects ($\boldsymbol{\beta}^{(a)}$) were sex, year and nest hatch date. We include two sex-specific fixed effects: the effect of clutch size on female survival ($\beta^{(a)}_f$), and the effect of male attendance on male survival ($\beta^{(c)}_f$). $\delta_i = 1$ when individual $i$ is female, and 0 when male. No random effects were included for adult survival, although importantly the residual adult survival effects ($e^{(a)}_{iy}$) were allowed to covary with the social parent random effect on body mass ($m_{nyd}$; see SI), giving the 8x8 covariance matrix:

$$\begin{bmatrix} \mathbf{V}_m & \mathbf{c}_{m,a} \\ \mathbf{c}^{\top}_{m,a} & 1 \end{bmatrix} \tag{5}$$

We assume the male covariance matrix is the same as the female covariance matrix above. $\mathbf{c}_{m,a}$ is a vector of covariances between age-specific nurse effects and residual nurse survival, the variance of which is fixed at one (See SI for

implementation details). The (unknown) survival outcomes of dummy parents were not included in the analysis.

*Model 2*: Juvenile survival was also fitted as a threshold event history, although survival occurs between 9 age classes (0, 1, 3, 6, 9, 12, 15, 25, 365 days) where 1/0 in the final two age classes indicates birds that fledged but did not recruit, and a 1 in the final age class indicates recruits. In total there were 28810 survival events from 4345 chicks in 553 nests. Observations for time periods prior to hatching, or after death are omitted. Juvenile survival from day $d$ to day $d+1$ has the form:

$$j_{iyd} = \mathrm{I}\left(\mathbf{x}^{(j)}_{iyd}\boldsymbol{\beta}^{(j)} + \mathbf{z}^{(j)}_{iyd}\boldsymbol{u}^{(j)} + (\beta^{(j)}_{b} + d(1-\delta_{d25})\beta^{(j)}_{b:d} + \delta_{d25}\beta^{(j)}_{b:d25}) \cdot b_{iyd} + e^{(j)}_{iyd} > 0\right) \tag{6}$$

The generic fixed effects are the same as those for mass, but also included the nest-of-rearing clutch size and male attendance score. However, separate effects of each variable were fitted for survival from fledging to recruitment as the linear time dependency for the effect of the covariates on pre-fledging survival are unlikely to extrapolate beyond fledging. This model also includes the effect of an individual's mass on survival from day 0 to day 1 ($\beta^{(j)}_{b}$) how this increases each day up to fledging ($\beta^{(j)}_{b:d}$) and for fledging to recruitment ($\beta^{(j)}_{b:d25}$). $\delta_{d25} = 1$ when $d = 25$, and $b_{iy15}$ was used as $b_{iy25}$ was unobserved. A first order antedependence model was used to model the 8x8 covariance matrix of nest-of-rearing effects with the seven lagged regression coefficients and eight innovation variances all allowed to vary over age. Nest-of-origin was not included, as it was found to be of very small magnitude in Hadfield et al. (2013a) and the residual variance for survival ($e^{(j)}_{iyd}$) was fixed at 1 as in standard probit analysis.

*Model 3*: Annual adult fecundity was treated as a Gaussian response, since in females it is under-dispersed with respect to the Poisson (Kendall and Wittmann 2010). Female fecundity was equal to her clutch size and male fecundity was equal to all the chicks he sired in his own and other nests. In total there were 887 fecundity observations from 552 birds. The model has the form:

$$f_{iy} = \mathbf{X}_{iy}^{(f)}\beta^{(f)} + (1-\delta_i)\beta_c^{(f)} \cdot c_{iy} + e_{iy}^{(f)} \quad (7)$$

where the generic fixed effects are year and sex, and $\beta_c^{(f)}$ is the effect of male nest attendance on fecundity. The residual variance was also allowed to differ between the two sexes. The fecundity of dummy parents were not included in the analysis.

All models were fitted in MCMCglmm (Hadfield 2010) in the statistical program R (R Development Core Team 2012). The nest-of-origin and genetic random effects on body mass, and the nest-of-rearing effects on juvenile survival, had parameter expanded priors for the autoregressive structure, with a high scale (10) for the innovation variances. The autoregressive coefficients had a prior mean of 0, and variances of 10. The random-residual prior covariance (social parent effect on body mass and adult survival) followed an inverse-Wishart distribution. The degree of belief parameter was set to the dimensions of the prior matrix minus 3, and the prior scale matrix was set close to zero. Under a simple model, this prior should give posterior modes for the variances and covariances close to the REML estimators. The same prior was also used for the residual covariance matrix for mass. For those parts of the model that could be fitted with ASReml (Gilmour et al. 2009), the posterior modes from this model and the REML estimates were very similar.

## Selection gradients

We define total maternal performance *given* (not received) on day $d$ as:

$$mp_{iyd} = \delta_i \left[ (\beta_f^{(b)} + d\beta_{f:d}^{(b)}) \cdot f_{iy} + m_{iyd} \right] \quad (8)$$

and total paternal performance given as:

$$pp_{iyd} = (1-\delta_i) \left[ (\beta_c^{(b)} + d\beta_{c:d}^{(b)}) \cdot c_{iy} + p_{iyd} \right] \quad (9)$$

Where $\beta_f^{(b)}$ is the effect of female fecundity on offspring body mass, $\beta_{f:d}^{(b)}$ its change with offspring age, and $mp_{iyd}$ the female portion of the parental effect – the

effect on offspring mass attributed to the nurse. $\beta_c^{(b)}$, $\beta_{c:d}^{(b)}$ are the equivalents for the effect of male attendance, and $pp_{iyd}$ the social sire's parental performance.

*Figure 1 here*

Our aim is to obtain selection gradients for the parental performances, in addition to the selection gradients on age-specific body mass. Figure 1 shows how the parental performance traits and juvenile body mass are expected to influence individual fitness components, and ultimately total fitness (lifetime reproductive success – LRS). In order to obtain selection gradients, we first reparameterize the statistical models into the causal model shown in Figure 1 (Equations 10, 11, and 12). We then show how the fitness components, juvenile survival, adult survival and fecundity, combine to generate LRS (Equations 13 and 14). By taking the partial derivatives of LRS with respect to the traits of interest, and averaging over the distribution of traits, fixed predictors and random effects in the models, we obtain the selection gradients (Lande and Arnold 1983) (Equation 15). This procedure is repeated for each MCMC iteration in order to obtain posterior distributions.

In order to derive selection gradients for these parental performance traits it is necessary to define their causal effect on fitness, as shown in Figure 1, rather than the correlative model defined in Equation 5, which estimates the covariance between the age-specific parental effects on offspring mass and the residual for parental survival. We can consider the regression of adult survival on age specific parental effects as (for maternal effects): $\boldsymbol{\beta}_m^{(a)} = \mathbf{V}_m^{-1}\mathbf{c}_{m,a}$ (where $\mathbf{c}_{m,a}$ is a vector of covariances between the residual for nurse survival and age-specific parental effects, from the matrix in equation 5), and note that $\boldsymbol{\beta}_p^{(a)} = \boldsymbol{\beta}_m^{(a)}$ by assumption. This gives:

$$\begin{aligned} a_{iy} = &\ I\left(\mathbf{x}_{iy}^{(a)}\boldsymbol{\beta}^{(a)} + \delta_i\left[\textstyle\sum_d \beta_{m:d}^{(a)} \cdot m_{iyd}\right] + (1-\delta_i)\left[\textstyle\sum_d \beta_{p:d}^{(a)} \cdot p_{iyd}\right] + \delta_i e_{iy}^{(a|m)} + (1-\delta_i)e_{iy}^{(a|p)} > 0\right) \\ = &\ F_N\left(\mathbf{x}_{iy}^{(a)}\boldsymbol{\beta}^{(a)} + \delta_i\left[\textstyle\sum_d \beta_{m:d}^{(a)} \cdot m_{iyd}\right] + (1-\delta_i)\left[\textstyle\sum_d \beta_{p:d}^{(a)} \cdot p_{iyd}\right], 0, \sigma_e^{(a|m)}\right) \end{aligned} \tag{10}$$

where $e^{(a|m)}$ is the residual nurse survival after conditioning on parental effects, with standard deviation $\sigma_e^{(a|m)} = \sqrt{1 - \mathbf{c}'_{m,a}\boldsymbol{\beta}_m^{(a)}}$. Again, $\sigma_e^{(a|m)} = \sigma_e^{(a|p)}$. $F_N$ is the

cumulative distribution function for the normal distribution and the second line of Equation 10 expresses the threshold model in probit form. It is important to realise that adult survival is determined by the parental performance an individual expresses (rather than receives) in this model.

In the statistical model female fecundity is a predictor of mass, and consequently maternal performance. The fitness function requires the inverse of this, such that maternal performance predicts fecundity, and so we obtain this as $\beta^{(f)}_{b:d} = \left[\beta^{(b)}_{f} + d\beta^{(b)}_{f:d}\right]^{-1}$. The selection model for fecundity therefore has the form:

$$f_{iy} = \mathbf{x}^{(f)}_{iy}\boldsymbol{\beta}^{(f)} + (1-\delta_i)\beta^{(f)}_{c} \cdot c_{iy} + e^{(f)}_{iy} + \delta_i(\sum_{0}^{d} \beta^{(f)}_{b:d}) \cdot b_{iy} \tag{11}$$

These transformations represent the model described by the path diagram in Figure 1. The model for juvenile survival remains identical to that given in the statistical section, although it can be written in probit rather than threshold form:

$$j_{iyd} = F_N\left(\mathbf{x}^{(j)}_{iyd}\boldsymbol{\beta}^{(j)} + \mathbf{z}^{(j)}_{iyd}\boldsymbol{u}^{(j)} + (\beta^{(j)}_{b} + d(1-\delta_{d25})\beta^{(j)}_{b:d} + \delta_{d25}\beta^{(j)}_{b:d25}) \cdot b_{iyd} + e^{(j)}_{iyd}, 0, 1\right) \tag{12}$$

The probability of surviving from fledging to recruitment ($j_{25}$) will be underestimated by our statistical model, because many surviving fledglings may have emigrated rather than recruited locally. If there is no relationship between body mass at day 15 and dispersal, the regression slope $\beta^{(j)}_{b:d25}$ will also be downwardly biased. These two sources of bias have opposite effects on the estimated selection gradient but, surprisingly, they exactly cancel so that the issue of incorrectly scoring emigrants as dead can be ignored when calculating pre-breeding survival selection. A proof for this statement can be found in the SI, together with an in depth discussion on the issue.

Equations 10, 11, and 12 above relate the parameters of interest to each of the three fitness components that comprise total fitness. However, we are interested in obtaining estimates of the total selection on body mass and parental performance; We do this by deriving the expected life-time reproductive success (LRS). This can

be considered as the sum of annual fitnesses: the product of age specific fecundity
374 ($f_t$) and survival to that age ($\prod_{y=1}^{t} s_y$):

$$W = \sum_{t=1}^{\infty} f_t \prod_{y=1}^{t} s_y \tag{13}$$

In our model survival to the first breeding attempt is $s_1 = \prod_d j_d$, where $d$
376 is taken over the measurement days 0, 1, 3, 6, 9, 12, 15 and 25. We assume annual survival thereafter is constant with age ($s_t = a \; \forall \; t > 1$), as is fecundity.
378 Consequently, Equation 13 is a geometric series and can be simplified:

$$W = \frac{f \prod_d j_d}{1 - a} \tag{14}$$

We partition the variables that vary over individuals into traits of interest
380 $\boldsymbol{\theta} = [b, mp, pp]^{'}$ and the remaining fixed effect predictors and random effects not of inherent interest $\boldsymbol{\eta}$. The selection gradients on the traits of interest are given by
382 (Lande and Arnold 1983):

$$\boldsymbol{\beta}_\theta = \frac{E[\partial W(\theta, \eta)/\partial \theta]}{E[W(\theta, \eta)]} = \frac{\int_\theta \int_\eta (\partial W(\theta, \eta)/\partial \theta) p(\theta, \eta) d\theta d\eta}{\int_\theta \int_\eta W(\theta, \eta) p(\theta, \eta) d\theta d\eta} \tag{15}$$

The partial derivative represents the change in fitness (per unit change in the
384 trait) that would be acheived if an individual's trait value was increased by a small amount. The partial derivative can be calculated analytically for this model. When
386 there is a non-linear relationship between the trait and fitness (as here) increasing each individual's trait value by the same small amount will cause different changes
388 in their fitness. As a consequence, we need to take the average change in fitness (represented by an expectation term) across individuals. This cannot be done
390 analytically, so we essentially simulated records for 10,000 individuals, calculated the change in their fitness had we increased their trait value (body mass or parental
392 performance) by a small amount, and took the average. This was done for every stored MCMC iteration, in order to obtain a posterior distribution for $\theta$. An
394 individual's variables were sampled from the posterior predictive distribution of the

model with the generic fixed predictors ($\mathbf{X}\beta$) sampled from a normal distribution with mean and variance equal to the empirical mean and variance of the generic fixed predictors in the actual population. This assumes the generic fixed predictors for each model are distributed independently of the random effects and the other fixed predictors. Male attendance, like the generic fixed predictors, was also not explicitly modelled in the analyses and so was sampled from a multinomial with cell probabilities equal to those in the actual population. Selection gradients were also obtained for the individual components of parental performance (such as male attendance) shown in Equations 8 and 9.

## Results

**Body mass**: The fixed effects for body mass are summarised in Table 1. Broadly, these showed the same patterns as those found in Hadfield et al. (2013a). Clutch size and male attendance were not included in Hadfield et al. (2013a) however, and both were found to have significant effects, particularly after day 0. An increased clutch size led to decreased body mass of -0.004 g/egg ([-0.010 - 0.001], P=0.110) at day 0, and -0.110 g/egg ([-0.156 - -0.062], P<0.001) at day 15, suggesting females provided less care to each individual offspring when they have a larger number to care for. Although male attendance alone had no significant effect on mass at day 0 (0.004 g/attendance-unit [-0.014 - 0.022] P=0.650), there was a strong positive interaction with day, such that there was an increase in body mass of 0.936 g/attendance-unit ([0.786 - 1.121], P<0.001) at day 15 (i.e. a juvenile in a nest with a male feeding is predicted to be almost 1g heavier than if a male has been seen but never caught).

*Table 1 here*

The proportion of variation in body mass explained by the different random effects included in the model is shown in Figure 2. This shows that at day 0 the social parents (as a pair) accounted for 32% (27.3 - 36.5) of the variance in mass, which is likely to reflect differences in the true time since hatching (between 0 and

23 hours), and whether a female has spent that time brooding additional eggs or feeding those that have hatched. After day 6 they were the main driver of body mass, and accounted for 70.9% (66.7 - 75) of the variance at day 15.

*Figure 2 here*

**Juvenile Survival**: The fixed effects for juvenile survival are summarised in Table 2. The effect of mass on survival was positive and significant, indicating an increased survival probability with increased mass – survival improved by 2.332 probits/g ([1.975 - 2.729], P<0.001) at day 0. However, there was a negative interaction with day, such that the extent to which increased mass improved survival declined across ontogeny, such that survival was increased by 0.821 probits/g ([0.725 - 0.947], P<0.001) by the mass at day 15 prior to fledging, and 0.194 probits/g ([0.074 - 0.298], P<0.001) after fledging (survival to recruitment).

*Table 2 here*

The between-nest variances for age-specific survival generally show decreasing between-nest variance across ontogeny, particularly for survival between fledging and recruitment. The full covariance matrix is presented in the SI.

**Adult Survival**: The fixed effects for adult survival are summarised in Table 3. None of the fixed effect predictors for adult survival were significant, including those that form part of maternal and paternal performance – female survival declined non-significantly with clutch size (change of -0.024 probits/egg [-0.077 - 0.059] P=0.746), and increased nest attendance caused a non-significant decline in survival in males (-0.151 probits/attendance unit [-0.604 - 0.438] P=0.653). The probit regression coefficients of adult survival on the parental effects (m/p; Equation 10) are also shown in Table 3 and have large credible intervals. This probably reflects the strong correlations between the predictors: the age-specific parental performances. However, the residual standard deviation $\sigma_e^{(a|m)}$ is close to 1 (0.977 [0.943 - 0.995]) meaning that the variation in adult survival explained by the parental performances across all ages is quite modest.

*Table 3 here*

**Adult Fecundity**: The fixed effects, and residual variances, of adult fecundity are shown in table 4. Both the intercepts and residual variances were different for males and females. Males had higher fecundity than females when they did not attend their nest (male attendance score of zero), but increased male attendance (i.e. care) decreased fecundity by -7.531 eggs/attendance unit ([-8.751 - -5.999], P<0.001).

*Table 4 here*

## Selection Gradients

Selection gradients on parental performance are in units of body mass, and therefore directly comparable with the selection gradients on body mass. However, because the mean and variance in body mass increases over ontogeny, we mean-standardise the selection gradients to make them comparable across ages (Houle 1992). As a consequence the selection gradients are the effect on relative lifetime fitness of doubling one's own body mass or, in the case of the parental performance, doubling your contribution to your offspring's body mass. We assume that the relative contribution of each parental performance trait (maternal/paternal effects and male-attendance/female-fecundity) to a proportional increase in parental performance is equal to their relative contribution to the variation in parental performance. For males, the relative weighting on day $d$ for paternal effects is $w_{p:d} = \sigma^2_{p:d}/(\sigma^2_{p:d} + (\beta_c + \beta_{c:d})^2\sigma^2_c)$ and $1 - w_{p:d}$ for male attendance, where $\sigma^2_{p:d}$ is the age-specific variance in paternal effects and $\sigma^2_c$ is the variance in male attendance. For females, the relative weighting on day $d$ for maternal effects is $w_{m:d} = \sigma^2_{m:d}/(\sigma^2_{m:d} + (\beta_f + \beta_{f:d})^2\sigma^2_f)$ and $1 - w_{m:d}$ for female fecundity, where $\sigma^2_{m:d}$ is the age-specific variance in maternal effects and $\sigma^2_f$ is the variance in female fecundity. This weighting is used because doubling parental performance by doubling the parental effects is not possible because parental effects have zero mean by definition.

Age-specific selection gradients are shown in Figure 3, together with age-constant selection gradients (i.e. the change in fitness caused by doubling mass at *all* ages). As expected, there was significant positive selection on body mass (age-constant selection 9.291 [4.640 - 11.994] P<0.001). The majority of this selection, however, occurs after nine days, with weak selection on mass early in ontogeny, e.g. selection is 0.008 ([0.003 - 0.016], P<0.001) at day 0. The magnitude of selection is greatest at day 15 (4.043 [1.686 - 5.496] P<0.001) – the mass at this age affected both the probability of an individual surviving to fledge, and the probability of fledged individuals recruiting to breed.

The selection gradients on *total* parental performance overlap zero overall (Figure 3, black points, age constant selection is -10.505 [-14.541 - -3.356] P=0.023 in males and -3.464 [-9.075 - -0.040] P=0.023 in females), although there is some variation with nest age. Looking at the components of parental performance, the parental effects (acting through adult survival) overlap zero at all ages (Figure 3, light grey points, age-constant selection is -0.355 [-3.632 - 1.458] P=0.459 in males and -0.437 [-5.646 - 2.149] P=0.370 in females) giving little evidence for a survival cost to the parental effects. The selection gradients on parental performance via male attendance and female fecundity were both negative with age-constant selection gradients of -9.303 ([-14.077 - -2.228], P=0.031) and -2.635 ([-5.025 - -0.827], P<0.001) respectively. In general the selection on parental performance via male attendance and female fecundity changed little with offspring age.

*Figure 3 here*

## Response to Selection

In the absence of parental performance effects we can use the Lande (1979) Equation to predict evolutionary change in age-specific mass $\mathbf{\Delta \bar{b}} = \mathbf{G}\beta_{\mathbf{b}}$. Using $\mathbf{G}$, the genetic (co)variance matrix of age-specific mass, obtained in model 1, the 95% credible intervals for the expected response to selection on mass early in ontogeny overlap zero (e.g. for day 0 the expected response was -0.000 [-0.003 - 0.004]). After

day six the expected responses to selection become significantly positive (0.018 [0.003 - 0.039] P<0.001), and by day 15 an increase in mass of 0.054 grams per year ([0.017 - 0.093], P<0.001), i.e. 0.50% of mean day 15 mass, would be expected.

In the presence of parental performance effects the expected response in age-specific mass requires quantifying selection on each trait contributing to parental performance, and the genetic (co)variances between these traits and (juvenile) body mass (Kirkpatrick and Lande 1989; Hadfield 2012). In the SI we show that if selection on parental performance traits is proportional to their parental effect (i.e. the cost to the parent is proportional to their effect on the offspring's phenotype) then the evolutionary dynamics of body mass can be captured by a simple single-trait parental performance model as used by Cheverud (1984b). Assuming this, and extending Cheverud's (1984b) model to allow for maternal/paternal-specific effects and extra-pair paternity (see SI), we can determine the genetic architectures of body mass and parental performance that result in the evolutionary stasis of body mass. For uniparental care (or when genetic variance in parental performance is only present in one sex) stasis is achieved when the genetic correlation between parental performance and body mass $r^*_{b,p}$ is

$$r^*_{b,p} = -\frac{1 + \frac{1}{2}\tilde{g}_p(1+\tilde{\beta}_p) - \frac{p}{2}\tilde{g}_p}{\sqrt{\tilde{g}_p}(\frac{3}{2} - \frac{p}{2} + \frac{1}{2}\tilde{\beta}_p)} \tag{16}$$

where $\tilde{\beta}_p$ is the selection on paternal/maternal performance relative to selection on mass ($\tilde{\beta}_p = \beta_p/\beta_b$), $\tilde{g}_p$ is the genetic variance in parental performance relative to the genetic variance for mass ($\tilde{g}_p = g_p/g_b$) and $p$ is the probability that an individual is extra-pair. In maternal-care scenarios $p = 0$ and the result can be derived directly from Equation 2 (see Cheverud (1984b) also).

With biparental care, and when the trait is expressed in both sexes with identical genetic architecture (i.e. the genetic correlation between maternal and paternal performance is one), the equilibrium genetic correlation is:

$$r^*_{b,p} = -\frac{1 + 2\tilde{g}_p(1+\tilde{\beta}_p) - p\tilde{g}_p}{2\sqrt{\tilde{g}_p}(\frac{3}{2} - \frac{p}{4} + \frac{1}{2}\tilde{\beta}_p)} \tag{17}$$

where $\tilde{\beta}_p$ is now the sex-averaged strength of selection on parental performance compared to that on body mass. With the same definition of $\tilde{\beta}_p$ the equilibrium genetic correlation between body mass and parental performance, but when parental performance in the two sexes is not genetically correlated, is:

$$r^*_{b,p} = -\frac{1 + \tilde{g}_p(1 + \tilde{\beta}_p) - \frac{p}{2}\tilde{g}_p}{2\sqrt{\tilde{g}_p}(\frac{3}{2} - \frac{p}{4} + \frac{1}{2}\tilde{\beta}_p)} \tag{18}$$

Using the extra-pair paternity rate in our poulation ($p$ =0.18) and our estimates of $\tilde{\beta}_p$ we can find the genetic correlation for which an equilibrium is reached ($r^*_{b,p}$) for a given value of $\tilde{g}_p$. As we are unable to estimate $\tilde{g}_p$ from our data, we determine $r^*_{b,p}$ over a range of values of $\tilde{g}_p$. The posterior distribution of $r^*_{b,p}$ is presented in Figure 4. In the case of males the total selection gradient for parental performance compared to the selection gradient on body mass is -1.195 [-2.356 - -0.303]), and so reduces the need for an extreme negative genetic covariance compared to when there is an absence of selection on the trait. The relative selection on female parental performance is -0.516 [-1.208 - -0.004]), so on average does not lift the constraint on stasis as much as male performance. However, the posterior distribution of the difference is -0.679 [-1.753 - 0.134] and overlaps zero. The combined effect of selection on sex-specific parental performances depends on the degree to which their genetic basis is shared, but in general the equilibrium genetic correlation lies between that required when selection operates on both sexes simultaneously.

*Figure 4 here*

## Discussion

Here we provide the first estimates of selection upon parental performance, a trait defined through its impact on the phenotype of offspring but caused by multiple unmeasured traits in the individual (such as nest-site selection and food provisioning rate; Willham 1963, 1972). As expected (Hadfield 2012), these results show that increasing the mass of an offspring is costly to an individual, when fitness is measured as the lifetime total number of zygotes an individual produces

over its lifetime (Arnold 1985). The cost of care is driven chiefly through reduced fecundity, rather than survival, and the magnitude of this cost seems to be borne differently by the two sexes. Males that show higher levels of attendance (and therefore care) at those nests for which they are the social father pay a current fecundity cost, through reduced rates of polygamy and extra-pair mating, as has been predicted (Werren et al. 1980; Westneat et al. 1990; Houston and McNamara 2002) and for which there is some previous evidence (Magrath and Elgar 1997; Schwagmeyer et al. 2012). Increased maternal performance also reduced current fecundity, supporting the prediction of a trade-off between investment per offspring and offspring number (Smith and Fretwell 1974), as has previously been found (e.g. Badyaev and Ghalambor 2001; Nakagawa et al. 2007). Indeed, Rollinson and Rowe (2015) demonstrate this trade-off at both phenotypic and genetic levels, with estimates of correlations between size and number being predominantly negative.

We set out to demonstrate how stasis in body mass in the face of positive directional selection might be explained in species with extended parental care and determinate growth. In the absence of selection acting against parental performance, the Willham model (Willham 1963, 1972) suggests that genetic correlations may contribute to stasis by constraining evolutionary change, although for stasis to be caused by genetic correlations alone, they must be close to -1. However, we show that the selection against parental performance that we observe lessens the need for extreme and implausible genetic architectures, although a negative genetic correlation would still be needed. The degree to which this is reduced depends upon the relative magnitudes of the direct and parental genetic effects on body mass. Although we were not able to estimate these, evidence for heritable variation in parental performance (Wilson and Réale 2006; Räsänen and Kruuk 2007) and general parental care traits has been found (MacColl and Hatchwell 2003; Walling et al. 2008; Dor and Lotem 2010), although see Wetzel et al. (2015). Estimates of the direct-parental genetic correlation in domestic populations (Wilson and Réale 2006; Räsänen and Kruuk 2007) are generally negative, although not strongly so. Thus, it is likely that a negative genetic correlation between parental performance and body mass in blue tits might also be found, but whether these would be sufficient

to explain stasis in body mass is not currently known. To our knowledge, the only estimates of the genetic parameters from a wild population are those for growth rate in squirrels (McAdam et al. 2002) and birth weight in Soay sheep (Wilson et al. 2005). In the latter case, $\tilde{g}_p = 1.42$ and $r_{b,p} = -0.41$. Thus to achieve stasis, $\tilde{\beta}_p \approx$-2.1 would be required, although there is considerable uncertainty about this value given the genetic parameter estimates have very low precision.

Our study employs correlational data to estimate selection, which may underestimate the costs of care compared to experimental studies that manipulate parental care. Differences in individual quality (Reznick 1985; van Noordwijk and de Jong 1986; Lessells and McNamara 2012) mean some individuals are likely to bear fewer costs through increased performance than others, which correlational studies struggle to account for without defining and measuring the quality of individuals. However, manipulating parental performance effectively is challenging; Of the various methods to measure the costs of care (Reznick 1985; Alonso-Alvarez and Velando 2012), clutch size manipulations have been the most commonly used (e.g. Nur 1988; Yoccoz et al. 2002; De Heij et al. 2006; Parejo and Danchin 2006; Santos and Nakagawa 2012). However, although such studies attempt to manipulate *total* parental investment of the focal individual, the parents may respond by only marginally increasing total investment and reducing the investment *per offspring*. Nevertheless, selection on parental performance could still be estimated from such data by considering the change in parental fitness in units of the total brood weight change caused by the treatment.

Our results imply that fecundity selection against parental performance differs between the sexes – the mean selection estimate is stronger in males. This might be expected, as males have higher variance in fecundity, and thus by increasing care they suffer a higher fitness cost (Trivers 1972; Smith 1977). However, the method with which males are assigned as social fathers to nests at which they were not caught may cause us to overestimate the strength of selection – we assign males based on majority paternity, so consequently these uncaught social fathers will have a higher fecundity, and lower male attendance scores. In order to assess the magnitude of this

problem we reran the model using only males that had ≥50% and ≥75% paternity at all their social nests. For nests with known social fathers (because they were caught) the correct male would have been assigned in 92% cases if maximum paternity had been used, but this increases to 98% and 100% when restricted to nests where a male secured ≥50% and ≥75% paternity, respectively. The model results remained qualitatively and quantitatively similar (see SI), but catching males on territories prior to breeding, or identification using unique colour rings or PIT tags (as in Kidd et al. 2015), would aid in assigning these uncaught social males to validate this relationship. Similarly, individuals failing in a breeding attempt prior to hatching remain unknown. Some of these individuals may then go on to lay a second clutch, at which they are caught. Thus, we underestimate the fecundity of those individuals (e.g. a female who lays a replacement clutch after abandoning her first may lay twice as many eggs as are counted). If individuals are not missing at random, estimates of selection may be biased (Hadfield 2008), and Kidd et al. (2015) found female great tits (*Parus major*) were less likely to be caught (due to early nest failure) if they were immigrants to the population, and when in poor quality habitats.

We do not estimate sex-specific effects of parental performance on survival – we assume that changes in performance affect survival in each sex identically. Although parents may provide different *total* amounts of care, the survival cost per unit care may not differ. For example, Santos and Nakagawa (2012) found sex differences in survival costs when clutch size was manipulated – males suffered reduced survival when clutch sizes were increased, but females suffered no such cost. This result is likely to be due to males altering their care levels in response to treatment, and thus suffering a survival cost, rather than the treatment altering the cost per unit care between the sexes. Additionally, we assume that the males and females contribute an equal amount to the parental performance for each nest, as a model including separate effects would only be weakly identifiable, but it is likely that parental performance is not equally divided. In blue tits the female alone builds the nest, incubates eggs and broods chicks (Cramp and Perrins 1993), and although both parents feed offspring, evidence of differences exist between feeding rate and duration, and nest sanitation behaviours (Banbura et al. 2001). Some aspects of

parental performance may also be attributable to the combination of the parents, rather than being divisible between the two – Ihle et al. (2015) found individuals pairs resulting from free mate choice had lower offspring mortality than those in forced pairs, implying behavioural compatibility may affect combined parental performance.

In conclusion, our results show that, when appropriate measures of fitness are used, there is selection against parental performance for body mass. This acts antagonistically to selection upon body mass, and goes some way towards explaining stasis in this trait. These are the first estimates of selection upon parental performance, and highlight the need to estimate these parameters when predicting how traits influenced by other individuals may respond to selection. Although an exact analysis requires that we measure selection (and $\mathbf{G}$) on the individual traits that constitute parental performance (Kirkpatrick and Lande 1989), we have shown that measuring selection on total parental performance is empirically tractable and, we believe, sufficiently accurate to get a better understanding of how body mass evolves. Due to limitations of the data, we did not estimate genetic parameters of parental performance, and thus any future studies that are able to fully estimate both selection and genetics will be able to generate more complete predictions as to the way in which stasis is maintained.

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Table 1: Posterior mean and 95% credible intervals for the fixed effects from a Gaussian model of body mass. pMCMC is twice the posterior probability that the estimate is negative or positive (whichever probability is smallest). pWald is the P-value from a Wald test that jointly tests the main effect and the interaction with day.

| | mean | l-95% | u-95% | pMCMC | pWald |
|---|---|---|---|---|---|
| Day 0 | 0.605 | 0.466 | 0.730 | **<0.001** | |
| Day 1 | 1.114 | 0.969 | 1.270 | **<0.001** | |
| Day 3 | 2.563 | 2.315 | 2.816 | **<0.001** | |
| Day 6 | 5.579 | 5.150 | 6.022 | **<0.001** | |
| Day 9 | 8.516 | 7.912 | 9.197 | **<0.001** | |
| Day 12 | 10.105 | 9.226 | 10.917 | **<0.001** | |
| Day 15 | 10.711 | 9.629 | 11.729 | **<0.001** | |
| Sex (F) | -0.003 | -0.009 | 0.004 | 0.425 | |
| Sex (F) : Day | -0.022 | -0.025 | -0.019 | **<0.001** | **<0.001** |
| Hatch Day (0-1) | -0.180 | -0.192 | -0.169 | **<0.001** | |
| Hatch Day (0-1) : Day | -0.028 | -0.033 | -0.023 | **<0.001** | |
| Hatch Day (1-3) | -0.711 | -0.769 | -0.657 | **<0.001** | |
| Hatch Day (1-3) : Day | -0.043 | -0.055 | -0.031 | **<0.001** | **<0.001** |
| Year.2011 | -0.031 | -0.064 | 0.003 | 0.073 | |
| Year.2011 : Day | -0.035 | -0.054 | -0.018 | **<0.001** | |
| Year.2012 | -0.031 | -0.063 | 0.001 | 0.054 | |
| Year.2012 : Day | -0.029 | -0.046 | -0.011 | **0.002** | |
| Year.2013 | -0.087 | -0.127 | -0.043 | **<0.001** | |
| Year.2013 : Day | -0.028 | -0.051 | -0.005 | **0.022** | |
| Year.2014 | 0.020 | -0.013 | 0.054 | 0.251 | |
| Year.2014 : Day | -0.015 | -0.035 | 0.003 | 0.113 | **<0.001** |
| Clutch Size | -0.004 | -0.010 | 0.001 | 0.11 | |
| Clutch Size : Day | -0.007 | -0.010 | -0.004 | **<0.001** | **<0.001** |
| Nest Hatch Date | 0.003 | 0.001 | 0.005 | **<0.001** | |
| Nest Hatch Date : Day | -0.002 | -0.003 | -0.002 | **<0.001** | **<0.001** |
| Male Attendance | 0.004 | -0.014 | 0.022 | 0.65 | |
| Male Attendance : Day | 0.062 | 0.052 | 0.074 | **<0.001** | **<0.001** |
| Time | 0.554 | 0.485 | 0.629 | **<0.001** | |
| Time : Day | 0.141 | 0.118 | 0.165 | **<0.001** | **<0.001** |

Table 2: Posterior mean and 95% credible intervals for the fixed effects from a threshold model of juvenile survival. pMCMC is twice the posterior probability that the estimate is negative or positive (whichever probability is smallest). pWald is the P-value from a Wald test that jointly tests the main effect and the interaction with day and pre/post fledging.

| | mean | l-95% | u-95% | pMCMC | pWald |
|---|---|---|---|---|---|
| Day 0 | 8.497 | 2.364 | 15.268 | **0.004** | |
| Day 1 | 6.187 | 0.676 | 12.311 | **0.017** | |
| Day 3 | 0.408 | -3.752 | 4.366 | 0.871 | |
| Day 6 | 0.152 | -2.958 | 3.386 | 0.948 | |
| Day 9 | -3.934 | -6.539 | -1.327 | **0.004** | |
| Day 12 | -3.585 | -6.133 | -0.886 | **0.007** | |
| Day 15 | -0.937 | -4.047 | 2.605 | 0.583 | |
| Day 25 (Postfledging) | -2.292 | -4.547 | 0.036 | **0.047** | |
| Sex (F) | 0.087 | -0.217 | 0.399 | 0.582 | |
| Sex (F) (Prefledging) : Day | 0.015 | -0.012 | 0.043 | 0.293 | |
| Sex (F) (Postfledging) | -0.239 | -0.583 | 0.130 | 0.185 | **<0.001** |
| Hatch Day (0-1) | 0.133 | -0.253 | 0.544 | 0.533 | |
| Hatch Day (0-1) (Prefledging) : Day | -0.040 | -0.070 | -0.003 | **0.026** | |
| Hatch Day (0-1) (Postfledging) | -0.294 | -0.732 | 0.164 | 0.196 | |
| Hatch Day (1-3) | 0.318 | -0.565 | 1.176 | 0.467 | |
| Hatch Day (1-3) (Prefledging) : Day | -0.052 | -0.128 | 0.020 | 0.154 | |
| Hatch Day (1-3) (Postfledging) | -0.632 | -1.640 | 0.412 | 0.222 | **0.001** |
| Year.2011 | -0.711 | -2.122 | 0.831 | 0.352 | |
| Year.2011 (Prefledging) : Day | -0.093 | -0.211 | 0.040 | 0.155 | |
| Year.2011 (Postfledging) | -0.165 | -1.661 | 1.395 | 0.813 | |
| Year.2012 | -2.195 | -3.620 | -0.714 | **0.003** | |
| Year.2012 (Prefledging) : Day | 0.101 | -0.016 | 0.210 | 0.067 | |
| Year.2012 (Postfledging) | 1.470 | -0.214 | 2.808 | **0.048** | |
| Year.2013 | -1.153 | -2.752 | 0.403 | 0.146 | |
| Year.2013 (Prefledging) : Day | 0.083 | -0.044 | 0.204 | 0.190 | |
| Year.2013 (Postfledging) | 1.235 | -0.260 | 3.075 | 0.136 | |
| Year.2014 | -0.170 | -1.704 | 1.308 | 0.830 | |
| Year.2014 (Prefledging) : Day | 0.018 | -0.098 | 0.141 | 0.768 | |
| Year.2014 (Postfledging) | -0.167 | -1.718 | 1.311 | 0.804 | **<0.001** |
| Clutch Size | -0.351 | -0.620 | -0.119 | **0.004** | |
| Clutch Size (Prefledging) : Day | 0.019 | -0.001 | 0.040 | 0.068 | |
| Clutch Size (Postfledging) | 0.338 | 0.078 | 0.591 | **0.013** | **0.018** |
| Nest Hatch Date | 0.008 | -0.054 | 0.080 | 0.800 | |
| Nest Hatch Date (Prefledging) : Day | -0.008 | -0.014 | -0.002 | **0.014** | |
| Nest Hatch Date (Postfledging) | -0.044 | -0.107 | 0.031 | 0.230 | **<0.001** |
| Male Attendance | 3.156 | 2.342 | 4.081 | **<0.001** | |
| Male Attendance (Prefledging) : Day | -0.153 | -0.224 | -0.083 | **<0.001** | |
| Male Attendance (Postfledging) | -2.751 | -3.739 | -1.793 | **<0.001** | **<0.001** |
| Weight | 2.347 | 1.975 | 2.729 | **<0.001** | |
| Weight (Prefledging) : Day | -0.101 | -0.131 | -0.075 | **<0.001** | |
| Weight (Postfledging) | -2.157 | -2.549 | -1.743 | **<0.001** | **<0.001** |

Table 3: Posterior mean and 95% credible intervals for the fixed effects from a threshold model of adult survival. m/p are the age-specific maternal/paternal effects expressed by the individual. pMCMC is twice the posterior probability that the estimate is negative or positive (whichever probability is smallest).

| | mean | l-95% | u-95% | pMCMC |
|---|---|---|---|---|
| Sex (F) | 0.306 | -0.938 | 1.479 | 0.626 |
| Sex (M) | 0.191 | -0.792 | 1.217 | 0.727 |
| Year.2011 | -0.020 | -0.282 | 0.250 | 0.883 |
| Year.2012 | -0.112 | -0.387 | 0.144 | 0.412 |
| Year.2013 | 0.076 | -0.229 | 0.400 | 0.625 |
| Year.2014 | -0.254 | -0.542 | 0.021 | 0.079 |
| Nest Hatch Date | -0.006 | -0.021 | 0.010 | 0.482 |
| Clutch Size (F) | -0.011 | -0.077 | 0.059 | 0.746 |
| Male Attendance (M) | -0.124 | -0.604 | 0.438 | 0.653 |
| m/p Day 0 | 3.089 | -0.047 | 6.038 | 0.051 |
| m/p Day 1 | -4.834 | -9.592 | 0.027 | 0.050 |
| m/p Day 3 | 1.002 | -0.916 | 3.221 | 0.338 |
| m/p Day 6 | 0.262 | -0.825 | 1.285 | 0.643 |
| m/p Day 9 | -0.424 | -1.184 | 0.272 | 0.280 |
| m/p Day 12 | 0.168 | -0.316 | 0.644 | 0.495 |
| m/p Day 15 | -0.053 | -0.363 | 0.278 | 0.740 |

Table 4: Posterior mean and 95% credible intervals for the fixed effects and residual variances from a Gaussian model of adult fecundity. pMCMC is twice the posterior probability that the estimate is negative or positive (whichever probability is smallest).

| | mean | l-95% | u-95% | pMCMC |
|---|---|---|---|---|
| Sex (F) | 10.030 | 9.703 | 10.328 | **<0.001** |
| Sex (M) | 25.076 | 22.380 | 27.750 | **<0.001** |
| Year.2011 | -0.612 | -1.040 | -0.239 | **0.004** |
| Year.2012 | -1.197 | -1.605 | -0.777 | **<0.001** |
| Year.2013 | -1.970 | -2.417 | -1.537 | **<0.001** |
| Year.2014 | -0.540 | -1.010 | -0.121 | **0.016** |
| Male Attendance | -7.399 | -8.751 | -5.999 | **<0.001** |
| **Residual Variances** | | | | |
| Sex (F) | 2.705 | 2.379 | 3.056 | |
| Sex (M) | 12.338 | 10.705 | 14.115 | |

**Figure 1**: Path diagram showing the connections between the traits and fitness components considered within the model. Solid lines represent those connections that are included in the models and involve an individual affecting themselves (e.g. paternal effect an individual provides affects their *own* survival), whereas dashed lines represent parental effects on their offspring (e.g. paternal effect an individual provides affects their *offsprings'* body mass). Dotted lines represent possible connections that are not included in the model, as we are not considering the effects of the two parents on each other. Male attendance is assumed to affect their own fecundity (not that of the female).

**Figure 2**: The proportion of variance in body mass across ontogeny (nest age in days) explained by multiple factors included in the model. Lines are the posterior means, with shaded ribbons representing the 95% credible intervals. The social parent effects (maternal and paternal effects combined) are those that make up part of the parental performance.

**Figure 3**: The selection gradients for body mass of juveniles (panel c), maternal performance (a) and paternal performance (b). Selection on maternal performance is in black, and selection on its constituent parts are in light grey (via the maternal effect) and dark grey (via female fecundity). Selection on paternal performance is in black, and selection on its constituent parts are in light grey (via the paternal effect) and dark grey (via male attendance). The summed selection gradients are shown (as total), as well as how selection changes across all ages of ontogeny at which nests are visited. Bars show the 95% credible intervals. Note that the scale of the y-axes varies between the different fitness components.

**Figure 4**: Parameter space in which there is evolutionary stasis in body size. Black lines show the genetic correlation that would be needed to explain stasis for different strengths of selection on parental performance (relative to selection on body mass) and different amounts of genetic variance in parental

performance (relative to genetic variance in body mass). Grey shading represents
708 the density of the posterior estimates of selection from the model. The top
panel is when selection and/or genetic variance is limited to paternal (left) or
710 maternal (right) performance (Equation 16). The difference in the countours is
because males are less related to the offspring they care for due to an extra-pair
712 paternity rate of 0.18. The bottom panel is when selection happens on paternal
and maternal performance, assuming the genetic variances for paternal and
714 maternal performance are equal. The genetic correlation between performance
in the two sexes is assumed to be one (left: Equation 17) or zero (right: Equation 18).

716

| Letter | Definition |
|---|---|
| Fitness | |
| a | adult survival |
| f | fecundity |
| j | juvenile survival |
| w | fitness (total) |
| Traits | |
| b | body mass |
| c | male attendance |
| m | maternal effect |
| p | paternal effect |
| mp | maternal performance |
| pp | paternal performance |

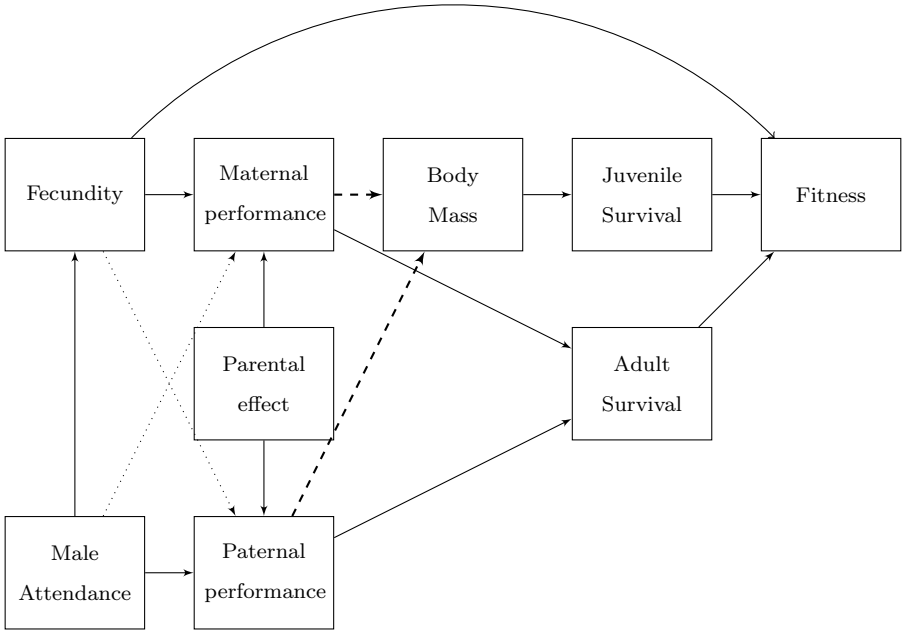


Fig. 1.—

718

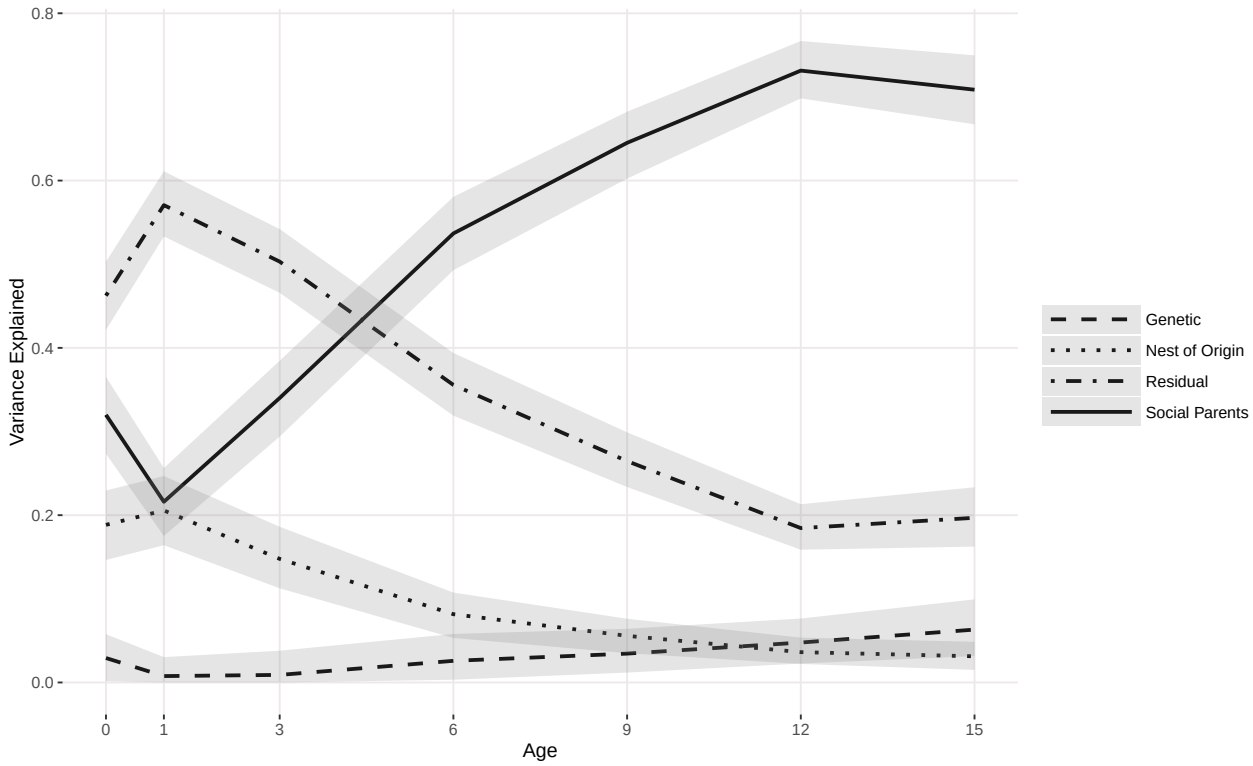
0.8
0.6
0.4
0.2
0.0
Variance Explained
0
1
3
6
9
12
15
Age
Genetic
Nest of Origin
Residual
Social Parents

Fig. 2.—

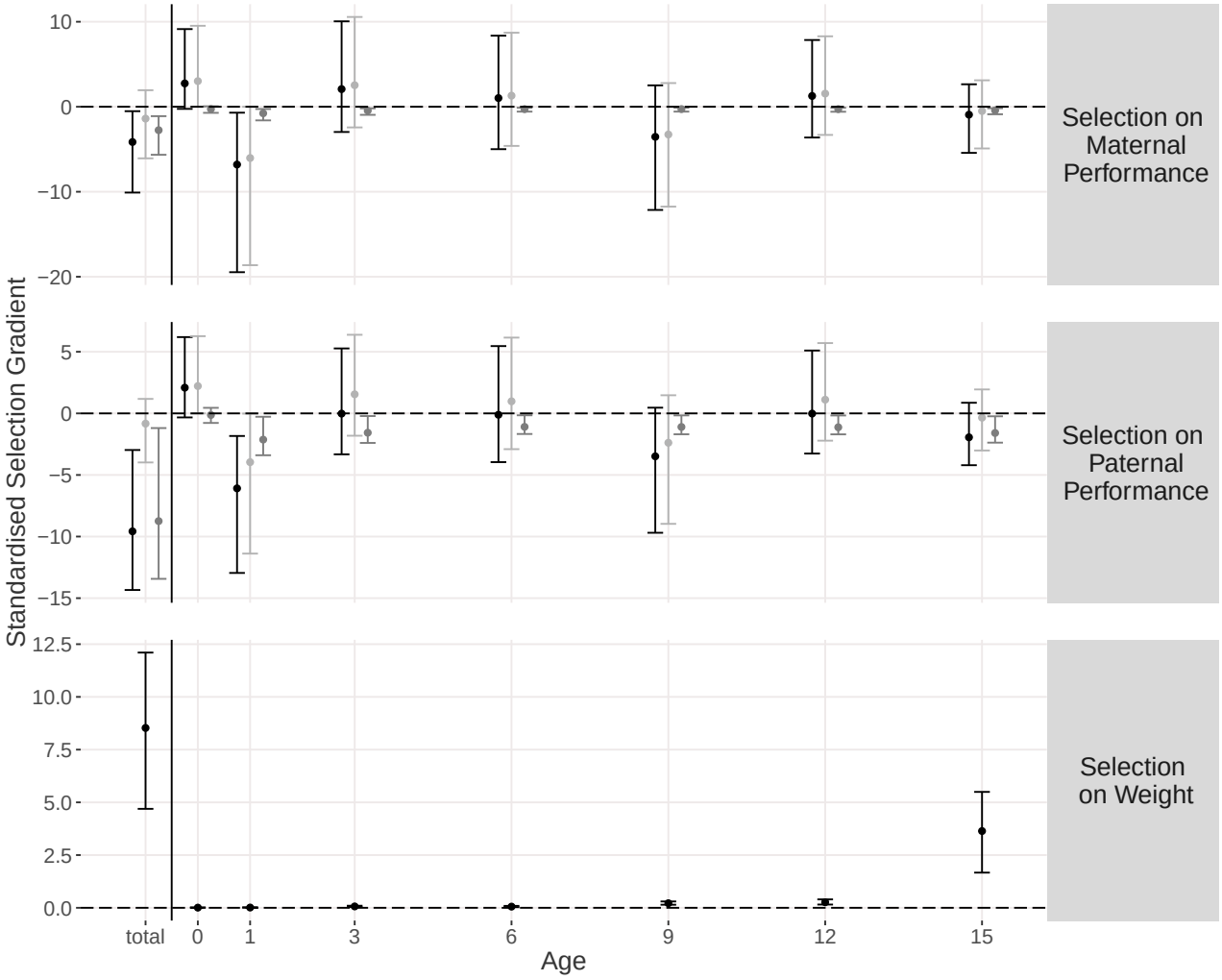
Standardised Selection Gradient
10
0
−10
−20
5
0
−5
−10
−15
12.5
10.0
7.5
5.0
2.5
0.0
total
0
1
3
6
9
12
15
Age
Selection on Maternal Performance
Selection on Paternal Performance
Selection on Weight

Fig. 3.—

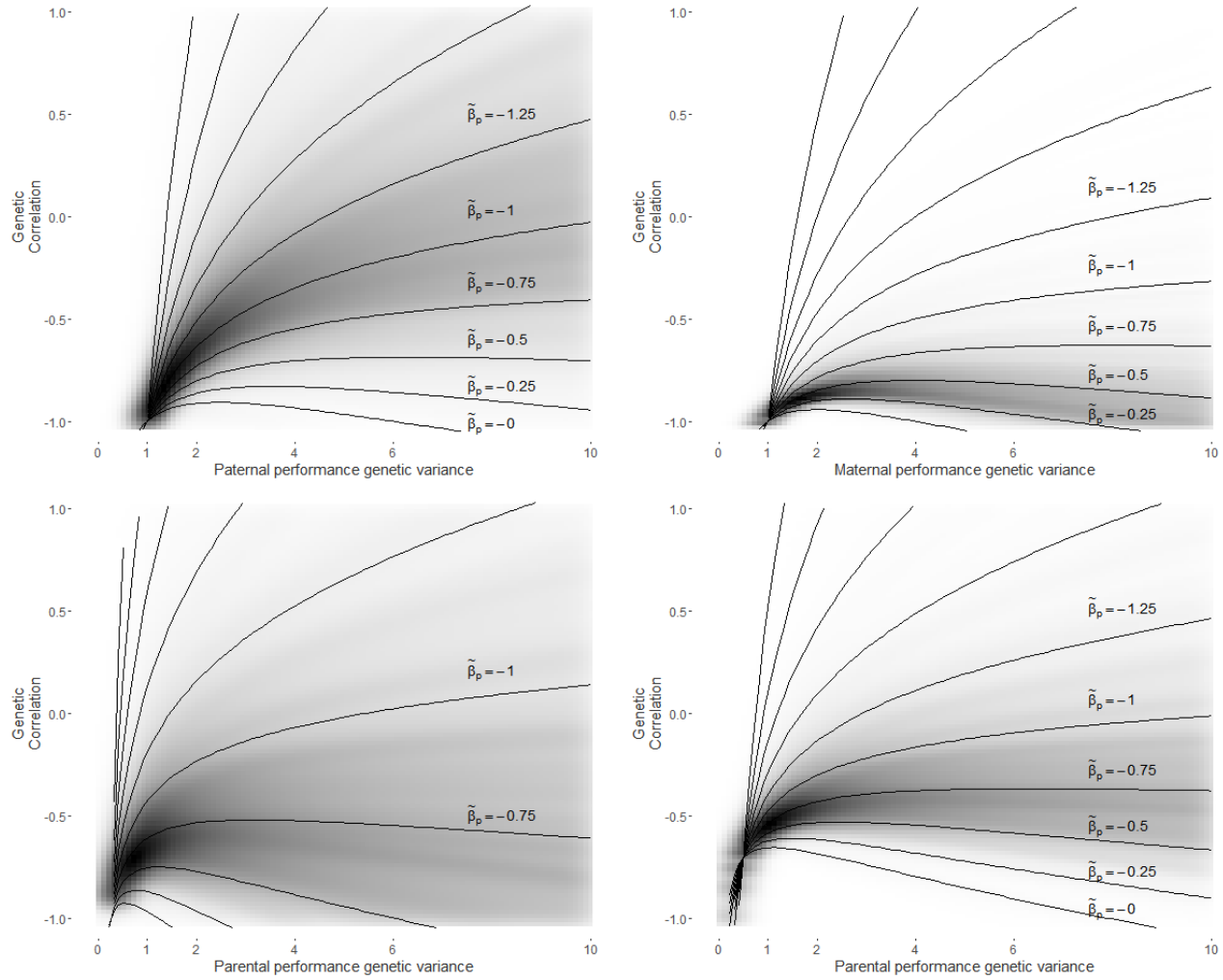
Genetic Correlation
1.0
0.5
0.0
-0.5
-1.0
0
1
2
4
6
10
Paternal performance genetic variance
Maternal performance genetic variance
Parental performance genetic variance
$\tilde{\beta}_p = -1.25$
$\tilde{\beta}_p = -1$
$\tilde{\beta}_p = -0.75$
$\tilde{\beta}_p = -0.5$
$\tilde{\beta}_p = -0.25$
$\tilde{\beta}_p = -0$

Fig. 4.—

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