

# Sex-specific additive genetic variances and correlations for fitness in a song sparrow (*Melospiza melodia*) population subject to natural immigration and inbreeding

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Quantifying sex-specific additive genetic variance ( $V_A$ ) in fitness, and the cross-sex genetic correlation ( $r_A$ ), is prerequisite to predicting evolutionary dynamics and the magnitude of sexual conflict. Further, quantifying  $V_A$  and  $r_A$  in underlying fitness components, and genetic consequences of immigration and resulting gene flow, is required to identify mechanisms that maintain  $V_A$  in fitness. However, these key parameters have rarely been estimated in wild populations experiencing natural environmental variation and immigration. We used comprehensive pedigree and life-history data from song sparrows (*Melospiza melodia*) to estimate  $V_A$  and  $r_A$  in sex-specific fitness and underlying fitness components, and to estimate additive genetic effects of immigrants alongside inbreeding depression. We found evidence of substantial  $V_A$  in female and male fitness, with a moderate positive cross-sex  $r_A$ . There was also substantial  $V_A$  in male but not female adult reproductive success, and moderate  $V_A$  in juvenile survival but not adult annual survival. Immigrants introduced alleles with negative additive genetic effects on local fitness, potentially reducing population mean fitness through migration load, but alleviating expression of inbreeding depression. Our results show that  $V_A$  for fitness can be maintained in the wild, and be broadly concordant between the sexes despite marked sex-specific  $V_A$  in reproductive success.

**KEY WORDS:** Cross-sex genetic correlation, genetic groups, inbreeding depression, migration load, quantitative genetic generalized linear-mixed model, sexual conflict.

The magnitude of additive genetic variance ( $V_A$ ) in fitness governs the rate of adaptive trait evolution and the expected increase in population mean fitness (Fisher 1930; Robertson 1966; Price 1970), and thereby links adaptation and population persistence (Bell 2013; Gomulkiewicz and Shaw 2013; Carlson et al. 2014; Shaw and Shaw 2014). Quantifying the magnitude of  $V_A$  in fitness, and identifying key mechanisms that maintain or constrain

such  $V_A$ , are consequently central objectives in evolutionary biology (Burt 1995; Barton and Keightley 2002; Ellegren and Sheldon 2008; Walsh and Blows 2009; Shaw and Shaw 2014; Hendry et al. 2018).

Fitness can be defined and measured in numerous ways (Brommer 2000; Metcalf and Pavard 2007; Orr 2009; Sæther and Engen 2015). In the context of Fisher's (1930) Fundamental

Theorem, absolute fitness is most straightforwardly defined as the total number of zygotes produced by a zygote (Crow and Kimura 1970; Arnold and Wade 1984; Falconer 1989, p. 336; Shaw and Shaw 2014). Such fitness emerges from a sequence of components comprising survival from conception to sexual maturity and adult lifetime reproductive success (LRS). Adult LRS itself results from a repeating sequence of reproduction and survival to the next reproductive opportunity, eventually terminated by death. Therefore, the magnitude and maintenance of  $V_A$  in fitness will ultimately depend on the magnitudes of  $V_A$  in all fitness components, and on the additive genetic correlations ( $r_A$ ) among these components.

In organisms with separate sexes, many genes that affect fitness are expressed in both sexes and can have congruent or divergent pleiotropic effects on sex-specific fitness components (Arnold and Wade 1984; Falconer 1989, p. 338; Chippindale et al. 2001). Resulting  $r_{AS}$  between the sexes, and among fitness components within each sex, can generate evolutionary sexual conflict, and multiple life-history trade-offs and multi-dimensional constraints (Lande 1980, 1982; Rose 1982; Charlesworth 1987; Chippindale et al. 2001; Kruuk et al. 2008; Bonduriansky and Chenoweth 2009; Walsh and Blows 2009; Shaw and Shaw 2014). Consequently,  $V_A$  in sex-specific fitness and fitness components, and corresponding cross-sex and within-sex  $r_{AS}$ , are key parameters shaping the total  $V_A$  for fitness that emerges and is maintained following selection (Lewontin 1974; Rose 1982; Chippindale et al. 2001; Brommer et al. 2007; Kruuk et al. 2008; Walsh and Blows 2009; Walling et al. 2014).

Further, the magnitude of standing  $V_A$  in fitness in any focal subpopulation will also depend on natural spatio-temporal variation in the form of selection and local adaptation and associated patterns of immigration and inter-deme gene flow (Merilä and Sheldon 1999; Zhang 2012; Carlson et al. 2014; Shaw and Shaw 2014). Immigration could increase  $V_A$  by introducing alleles with negative or positive additive effects on local fitness, potentially causing migration load, and impeding or facilitating adaptation and population growth (Lenormand 2002; Garant et al. 2007; Edelaar and Bolnick 2012; Carlson et al. 2014). Immigration could further change mean fitness by altering the degree of local inbreeding versus outbreeding and associated expression of inbreeding depression, heterosis, and outbreeding depression (Ingvarsson and Whitlock 2000; Tallmon et al. 2004; Frankham 2016). Such effects, and resulting effective gene flow, depend fundamentally on the genetic properties of immigrants relative to focal natives (Ingvarsson and Whitlock 2000; Tallmon et al. 2004; Edelaar and Bolnick 2012). Therefore, understanding and predicting overall evolutionary dynamics not only requires estimation of  $V_A$  in fitness and underlying fitness components in both sexes, and associated cross-sex and within-sex  $r_{AS}$  (Ellegren and Sheldon 2008; Kirkpatrick 2009; Kruuk et al. 2008, 2014; Shaw

and Shaw 2014; Walling et al. 2014), but also requires explicit estimation of multiple genetic effects resulting from immigration (Ingvarsson and Whitlock 2000; Lenormand 2002; Tallmon et al. 2004; Garant et al. 2007; Edelaar and Bolnick 2012; Carlson et al. 2014).

Fitness and its components reflect the expression of numerous developmental, physiological, morphological, and behavioral traits, and are consequently best conceptualized as highly polygenic, complex traits (e.g., Houle 1992; Barton and Keightley 2002; Flint and Mackay 2009; Hill 2012; Travisano and Shaw 2013), although loci of large effect can exist (e.g., Johnston et al. 2013; Trask et al. 2016). Hence, key  $V_{AS}$  and  $r_{AS}$  can be estimated using quantitative genetic methods derived from the infinitesimal model (Lynch and Walsh 1998). Although the phenotypic distribution of fitness is intrinsically non-Gaussian (Arnold and Wade 1984; Wagenius et al. 2010; Shaw and Etersson 2012; Bell 2013; Shaw and Shaw 2014),  $V_{AS}$  and  $r_{AS}$  can be estimated on latent scales. Here, non-Gaussian phenotypic expression is assumed to reflect underlying variation in a normally distributed latent trait to fulfill the fundamental quantitative genetic assumption of multivariate normality of the average effect of an individual's polygenic genotype (i.e., breeding value, Lynch and Walsh 1998, pp. 72–79 and Ch. 25; de Villemereuil et al. 2016).

Estimating  $V_{AS}$  and  $r_{AS}$  in latent traits in wild populations is empowered by a class of quantitative genetic generalized linear-mixed models (QGGLMMs, also known as “animal models”; Kruuk 2004; Charmantier et al. 2014). These models partition variance in observed phenotypes and, given an appropriate relatedness matrix and model, minimize biases in estimates of  $V_A$  and  $r_A$  stemming from selection (i.e., nonrandom variation in fitness, Henderson 1973; Kruuk 2004; Hadfield 2008). Such QGGLMMs can also directly estimate mean additive genetic values of immigrants relative to natives and estimate inbreeding depression, thereby elucidating key roles of immigration and resulting gene flow in shaping latent-scale and phenotypic means and variances (Reid and Keller 2010; Wolak and Keller 2014; Wolak and Reid 2017).

However, despite the recognized need and available statistical methods, few studies have rigorously estimated sex-specific  $V_{AS}$  and the cross-sex  $r_A$  in fitness in wild populations (Burt 1995; Gardner et al. 2005; Kruuk et al. 2008; Kirkpatrick 2009; Shaw and Shaw 2014; Hendry et al. 2018). Of 17 known studies that estimated  $V_A$  for sex-specific absolute fitness measured approximately from zygote to zygote, only eight considered male fitness alongside female fitness (Appendix S1). Since most such studies estimated at least one sex-specific  $V_A$  to be close to zero, only two attempted to estimate the cross-sex  $r_A$  (McFarlane et al. 2014; Zietsch et al. 2014, Appendix S1). Two further studies attempted to estimate the cross-sex  $r_A$  for fitness measured as an adult's number of adult (i.e., recruited) offspring (Brommer

et al. 2007; Foerster et al. 2007, Appendix S1). However, these cross-generation measures of fitness are harder to reconcile with evolutionary theory and risk confounding within-generation selection with evolution (Arnold and Wade 1984; Wolf and Wade 2001). Overall, existing estimates of the cross-sex  $r_A$  are very imprecise (Appendix S1). Further, few studies explicitly estimated  $V_A$  on appropriate latent scales (but see Milot et al. 2011; McFarlane et al. 2014) and these studies did not attempt to transform latent scale estimates back onto observed phenotypic scales. Such back-transformation is desirable to facilitate cross-study comparison, since latent scale estimates are highly model specific and do not necessarily have a linear relationship to the scale on which phenotypes are expressed and experience natural selection (de Villemereuil et al. 2016). Finally, no studies have yet explicitly estimated additive genetic effects of immigrants, or thereby directly assessed the role of introgressive gene flow in changing local mean breeding value and maintaining evolutionary potential.

The paucity of estimates for sex-specific  $V_{AS}$  and cross-sex  $r_{AS}$  in fitness likely reflects the substantial challenges of collecting comprehensive sex-specific fitness and relatedness data from free-living individuals. Since all conceived zygotes can rarely be counted, fitness can be pragmatically quantified as the total number of offspring produced over an individual's lifetime, where focal individuals and their offspring are censused as close to conception as feasible (typically soon after birth, hatch, or seed formation, Appendix S2.1). However, most field datasets have some degree of missing or incorrect parentage assignment, and resulting pedigree error could bias quantitative genetic analyses (Brommer et al. 2007; Firth et al. 2015; Wolak and Reid 2017). Further, challenges of tracking juveniles and of paternity assignment mean that records of survival to maturity and male reproductive success are often missing or incorrect (Kruuk et al. 2000; Brommer et al. 2007; Stinchcombe 2014). Observed fitness distributions may also exclude nonbreeders, and hence inaccurately reflect frequencies of individuals with zero fitness (Lebigre et al. 2012). Such error will likely bias key parameter estimates for fitness (e.g.,  $V_A$ , phenotypic means and variances) and hence bias standardized metrics that depend on such parameters (heritability,  $h^2$ ; evolvability,  $I_A$ ; coefficient of additive genetic variance,  $CV_A$ ; e.g., Freeman-Gallant et al. 2005). Even given comprehensive data spanning multiple generations,  $V_{AS}$  and cross-sex  $r_{AS}$  in non-Gaussian traits are notoriously difficult to estimate precisely (Shaw 1987; Poissant et al. 2010; Kruuk et al. 2014). Statistical methods that adequately quantify uncertainty should then be used to draw appropriate inference, and thereby facilitate interpretation and subsequent meta-analyses (Garcia-Gonzalez et al. 2012).

To achieve these aims, we fitted Bayesian QGGLMMs to comprehensive multigeneration fitness and pedigree data from song sparrows (*Melospiza melodia*) to estimate sex-specific  $V_{AS}$  and  $r_{AS}$  (and associated uncertainty) in fitness, and in two hierar-

chical levels of fitness components. First, we estimated  $V_A$  in sex-specific fitness and the cross-sex  $r_A$ , thereby evaluating the overall potential for evolutionary change and associated scope for intersexual conflict. Second, we estimated  $V_A$  and  $r_A$  in and among juvenile survival and sex-specific adult LRS, comprising the primary fitness components that generate the distribution of overall fitness. Third, we estimated sex-specific  $V_A$  and the cross-sex  $r_A$  in adult annual reproductive success (ARS) and estimated  $V_A$  in adult annual survival, representing the key fitness components that generate adult LRS. In all cases, we explicitly estimated additive genetic effects of immigrants relative to defined local population founders and estimated inbreeding depression, and thereby evaluated concurrent impacts of natural immigration and resulting gene flow on local additive genetic and phenotypic variation in fitness.

## Materials and Methods

### STUDY SYSTEM

Estimating  $V_A$  and  $r_A$  in fitness and fitness components in the wild is perhaps most tractable in populations with limited emigration but sufficient immigration to generate substantial variance in relatedness, and where all local residents and immigrants can be observed. A population of song sparrows inhabiting Mandarte Island, British Columbia, Canada, fulfills these criteria and has proved valuable for quantifying fitness of residents and immigrants and for pedigree-based quantitative genetic analyses (Keller 1998; Marr et al. 2002; Reid et al. 2011a, 2014a,b; Reid and Sardell 2012; Wolak and Reid 2016).

Mandarte's song sparrows typically form socially monogamous breeding pairs, starting from age one year, with a mean of  $28 \pm 11$ SD (range 11–52) breeding females per year during 1993–2015. Pairs can rear up to three broods of chicks per year (mean brood size  $2.8 \pm 1.0$ SD chicks, range 1–4). However, 28% of offspring are sired by extra-pair males (Sardell et al. 2010), creating opportunities for individual males to gain or lose substantial reproductive success compared to their socially paired female (Reid et al. 2011a, 2014b; Reid and Sardell 2012). Further, since the adult sex-ratio is often male-biased (mean proportion males during 1993–2015:  $0.60 \pm 0.09$ SD, range 0.39–0.75), some males remain socially unpaired in some years (Lebigre et al. 2012), and these males typically gain little extra-pair paternity (Sardell et al. 2010). Consequently, the population's mating system and ecology fosters different means and variances in female versus male reproductive success (Lebigre et al. 2012), creating potential for sexual conflict and trade-offs over fitness components despite social monogamy.

Since 1975, virtually all song sparrow breeding attempts on Mandarte have been closely monitored and all chicks surviving to ca. 6 days posthatch were marked with unique combinations

of metal and colored plastic bands (Smith et al. 2006). Mandarte lies within a large song sparrow meta-population and receives occasional immigrants (totaling 28 females and 16 males during 1976–2014) that were mist-netted and color-banded soon after arriving (Marr et al. 2002; Reid et al. 2006; Smith et al. 2006). Consequently, every song sparrow in the population is individually identifiable by field observation. Comprehensive surveys undertaken each April identified all surviving individuals, including unpaired males, with resighting probability  $>0.99$  (Wilson et al. 2007). Local chick survival from banding to adulthood the following April, and adult survival to subsequent years, were consequently accurately recorded (Keller 1998; Smith et al. 2006).

Each year, the socially paired parents that reared all banded offspring were identified. To determine genetic parentage, since 1993 all banded chicks and adults were blood sampled and genotyped at 160 polymorphic microsatellite loci. All chicks were assigned to genetic parents with  $>99\%$  individual-level confidence (Sardell et al. 2010; Nietlisbach et al. 2015). These analyses demonstrated zero extra-pair maternity, and effectively eliminated paternity error. Each banded individual's sex was determined from adult reproductive behavior and/or by genotyping the chromobox-helicase-DNA-binding (CHD) gene (Postma et al. 2011; Nietlisbach et al. 2015).

The local fitness of each chick banded on Mandarte since 1993 was measured as its total lifetime number of chicks banded on Mandarte. Focal chicks that died before adulthood were assigned a fitness of zero (Appendix S2). The two major fitness components, juvenile survival and adult LRS, were respectively measured as survival from banding to adulthood the following April, and as the total number of banded chicks assigned to individuals that survived to adulthood. For each adult, LRS was then further subdivided into ARS and annual survival, respectively measured as the number of banded chicks assigned to each individual in any one year, and as survival to the following April. Since adult (breeding) dispersal away from Mandarte is probably very rare, observed local adult survival likely equates to true survival (Marr et al. 2002; Smith et al. 2006). The relatively high local recruitment rate implies that juvenile (natal) dispersal is also relatively infrequent, although probably nonzero. However, surveys of immediately surrounding islands have detected few local dispersers, implying that unobserved dispersal from Mandarte is likely to be longer distance. Observed juvenile survival on Mandarte is therefore an appropriate measure of effective local survival and hence local fitness.

### QUANTITATIVE GENETIC MODELS

We built a hierarchy of three sets of QGGLMMs designed to estimate sex-specific additive genetic variances ( $V_A$ ) and covariances ( $COV_A$ ), and associated standardized statistics ( $r_A$ ,  $h^2$ ,  $I_A$ ,  $CV_A$ ), in (i) sex-specific fitness, then (ii) the two major multiplicative

components of fitness, namely juvenile survival and adult LRS, then (iii) the two components of adult LRS, namely adult ARS and annual survival. Since all traits showed non-Gaussian distributions, all QGGLMMs estimated parameters on latent scales and used appropriate error distributions.

First, we fitted a bivariate QGGLMM to estimate  $V_A$  in female and male fitness and the cross-sex  $COV_A$ , assuming Poisson distributions with log link functions. Random hatch-year effects were fitted to estimate sex-specific cohort variances in fitness and the cross-sex cohort covariance. Sex-specific residual variances, which measure random individual phenotypic deviations from expectation, were estimated assuming additive overdispersion. Residual covariance was fixed to zero, because there can be no cross-sex covariance between individual phenotypic deviations in traits with sex-limited phenotypic expression. This QGGLMM structure adequately models the observed distributions of fitness while facilitating direct biological interpretation of key parameters. Alternative modeling frameworks with more complex error distributions cannot currently be fitted to wild population relatedness structures and/or do not estimate parameters that directly relate to evolutionary quantitative genetic theory (Appendix S2.2).

Second, we fitted a trivariate QGGLMM to estimate  $V_A$  in juvenile survival and adult female and male LRS, and the three pairwise  $COV_{AS}$ , thereby considering the major components of overall fitness. We modeled juvenile survival as a single joint trait of both sexes with sex-specific intercepts, rather than as two sex-specific traits. This simplification facilitated multivariate analysis of juvenile survival alongside sex-specific adult LRS, and is acceptable because previously published and exploratory analyses demonstrated a positive cross-sex  $r_A$  for juvenile survival and similar magnitudes of  $V_A$  in both sexes, implying moderate shared  $V_A$  (Reid and Sardell 2012, Appendix S7). Under these conditions, modeling a single trait for both sexes does not bias estimates of  $V_A$  (Wolak et al. 2015), given the degree of uncertainty with which all parameters are estimated. Juvenile survival was modeled as a binary trait with logit link function and residual variance fixed to one. We assumed Poisson distributions for female and male LRS, with log link functions and independent residual variances (as for fitness). Random hatch-year effects were again fitted, thereby estimating cohort variances and covariances in and among the three traits.

Third, we fitted two separate QGGLMMs to estimate  $V_A$  in the two major components that generate adult LRS, namely adult ARS and annual survival. For ARS, we fitted a bivariate QGGLMM that estimated  $V_A$  in female and male ARS and the cross-sex  $COV_A$ , again assuming Poisson distributions for both traits, log link functions, and independent residual variances. Random individual effects were fitted to estimate sex-specific permanent individual variances (i.e., repeatable among-individual variation stemming from environmental and/or nonadditive genetic effects).

Random year of observation effects were also fitted to estimate among-year environmental variances and the cross-sex year covariance.

For survival, we fitted a univariate QGGLMM that estimated  $V_A$  in adult annual survival modeled as a single trait for both sexes with sex-specific intercepts (as for juvenile survival, Appendix S4). We modeled survival as a binary trait expressed by each individual adult in each year, with logit link function and residual variance fixed to one (e.g., Hadfield et al. 2013). Random year of observation and individual effects were fitted to estimate among-year environmental variance and account for overdispersion compared to the assumed geometric distribution of age-specific survival events. Initial models estimated little  $V_A$  in adult annual survival, implying that there can be no genetic covariance (or trade-off) with ARS. Exploratory trivariate QGGLMMs confirmed this view (Appendix S9). Hence, for simplicity, we present separate models for ARS and adult annual survival.

### IMMIGRANTS, INBREEDING DEPRESSION, AND FIXED EFFECTS

Standard QGGLMMs estimate  $V_A$  and  $COV_A$  for a default base population that comprises “phantom parents” of all pedigreed individuals with unknown parents (Kruuk 2004; Wolak and Reid 2017). In populations with complete local pedigree data for a focal study period but that are open to immigration, the default base population comprises phantom parents of all adults alive at the study start (hereafter “founders”) and of subsequent immigrants. To directly estimate the difference in mean additive genetic value for fitness and fitness components between the defined founders and subsequent immigrants, and account for heterogeneity that could otherwise bias  $V_A$  estimates, all QGGLMMs included trait-specific linear regressions on individual immigrant genetic group (*IGG*) coefficient. Each individual’s *IGG* coefficient quantifies the expected proportion of that individual’s autosomal genome that originated from the defined immigrant group, calculated from pedigree data (Appendix S3). The regression slope ( $\beta_{IGG}$ ), modeled as a fixed effect, estimates the difference in mean additive genetic value of the immigrant group relative to the founder group (Wolak and Reid 2017). Since immigration was infrequent, phantom parents of female and male immigrants that arrived in all years were pooled into a single genetic group (Appendix S3). This assumes that the phantom mothers of female and male immigrants have similar mean genetic values as the phantom fathers for any focal trait, and hence that alleles originating in immigrants of both sexes similarly affect the genetic values of descendants of both sexes. This mirrors the standard QGGLMM assumption that phantom mothers and fathers of founders have the same mean breeding values for any focal trait (Wolak et al. 2015).

To quantify inbreeding depression, and minimize bias in  $V_A$  estimates that can result from correlated inbreeding across

relatives, all four QGGLMMs also included trait-specific linear regressions on individual coefficient of inbreeding ( $f$ ), calculated from pedigree data (Reid and Keller 2010; Wolak and Keller 2014). Regression slopes ( $\beta_f$ ) equate to haploid inbreeding load for traits modeled with log link functions, but not with logit link functions.

Further fixed effects were restricted to those required to standardize trait observations across individuals. Since juvenile survival probability decreases with increasing seasonal hatch date (Smith et al. 2006), and hatch date reflects the parents’ breeding phenotype, models for juvenile survival included a linear regression on the first egg lay date in the nest in which each focal individual hatched. Since adult ARS and annual survival vary with age (Smith et al. 2006; Keller et al. 2008), associated models included categorical effects of age at observation (ages 1, 2, 3–5, or  $\geq 6$  years).

### PEDIGREE DATA AND MODEL IMPLEMENTATION

Comprehensive pedigree data were initially compiled by assigning all offspring banded during 1975–2014 to their observed socially paired parents. Paternal links for all chicks hatched during 1993–2014, and 37 additional chicks hatched during 1991–1992, were then corrected for extra-pair paternity based on genotypes at 160 microsatellite loci (Sardell et al. 2010; Reid et al. 2011a; Nietlisbach et al. 2015, 2017). For each QGGLMM, the pedigree was pruned to individuals with observed phenotypes and their known ancestors. The inverse numerator relatedness matrix, and individuals’ *IGG* and  $f$  coefficients, were computed using standard algorithms (Wolak and Reid 2017, Appendix S3). Immigrants were defined as unrelated to all Mandarte residents at arrival, and to subsequent immigrants (Marr et al. 2002; Reid et al. 2006).

For each model, phenotypic data were restricted to cohorts for which all or virtually all individuals had complete fitness or fitness component data, known sex, and genetically verified parents (Appendix S2). Observations of immigrants’ own phenotypes were excluded because they might reflect ecological effects associated with dispersal or subsequent settlement (Marr et al. 2002), and because immigrants’ pedigree  $f$  values are undefined relative to the Mandarte pedigree base population (Reid et al. 2006). However, immigrants that produced  $\geq 1$  banded offspring were explicitly included in the pedigree to enable estimation of relatedness among descendants and genetic group effects.

To facilitate estimation for non-Gaussian traits, and associated uncertainty, all models were implemented in a Bayesian framework, using a Markov chain Monte Carlo (MCMC) algorithm to sample posterior distributions. We used diffuse normal prior distributions for all fixed effects (mean = 0, variance =  $10^{10}$ ), and multivariate parameter expanded priors for covariance matrices that gave uniform marginal prior distributions

on the correlation. Parameter expanded priors were used for other variance components, giving scaled noncentral F-distributions with numerator and denominator degrees of freedom of one (Gelman 2006; Hadfield 2010) and scale parameter of 10 for binary traits or 1000 for Poisson traits (Appendix S4).

We retained 5000 samples of each marginal posterior distribution, with MCMC burn-in and thinning interval set to yield absolute autocorrelation values  $<0.1$  and satisfy convergence criteria (Appendix S4). Inference from such posterior distributions should be drawn from defined summary statistics, not directly from the full sample distribution (King et al. 2009, p. 85). Posterior distributions can show skew, kurtosis, or multiple peaks, including when parameters are near their boundary (e.g., variance near zero). Inferences drawn from posterior modes versus means may then differ. Consequently, we report the marginal posterior mean, mode, and 95% highest posterior density credible intervals (95%CI) and, for key metrics, also depict full marginal posterior distributions alongside prior distributions to further facilitate interpretation (Appendix S4). The 95%CI is especially pertinent when posterior distributions are non-Gaussian and/or uncertainty is large, and directly identifies the parameter values that can and cannot be excluded with 95% confidence given the data, prior, and model (King et al. 2009, pp. 86–88).

All QGGLMMs assumed Poisson or binary distributions and therefore estimated (co)variances on latent scales. Posterior distributions of latent-scale heritability ( $h^2_{\text{latent}}$ ) and  $r_A$  were computed from all samples of the marginal posterior distributions of underlying components following standard formulae (Appendix S4). Further, to facilitate future comparative studies and evolutionary inferences, we attempted to back-transform posterior distributions of latent-scale variances to the observed phenotypic scale and calculate observed-scale posterior distributions of standardized summary statistics ( $h^2_{\text{observed}}$ ,  $I_{A\text{-observed}}$ ,  $CV_{A\text{-observed}}$ , Appendices S4, S5). However, we could not recover reliable observed-scale variance component posteriors from our bivariate QGGLMM of female and male fitness due to the substantial overdispersion (Appendix S2).  $I_{A\text{-observed}}$  was not calculated for juvenile or adult survival because mean standardized variances are not meaningful for binary traits where the mean phenotype is bounded by 0 and 1 (Houle 1992).

Analyses were conducted in R (v3.2.3, R Core Team 2015) using the MCMCglmm (v2.22.1, Hadfield 2010), nadiv (v2.14.3.2, Wolak 2012), and QGglmm (v0.6.0, de Villemereuil et al. 2016) packages. Additional univariate QGGLMMs for sex-specific fitness, and univariate and bivariate QGGLMMs for combinations of juvenile survival and adult LRS, and trivariate models for adult ARS and annual survival (Appendix S9), gave quantitatively similar variance component estimates as the main QGGLMMs presented. Key (co)variance component estimates are robust to reasonable alternative

priors (Appendix S6), and remained similar when additional parental and common environmental effects were modeled (Appendix S5). Additional details of model specifications, results, and descriptive figures, are in Appendices S4 and S5. Data and R code for all analyses are available from GitHub: [https://github.com/matthewwolak/Wolak\\_etal\\_SongSparrow-FitnessQG](https://github.com/matthewwolak/Wolak_etal_SongSparrow-FitnessQG) and the Dryad Digital Repository: <https://doi.org/10.5061/dryad.p7p1jb3> (Wolak et al. 2018).

## Results

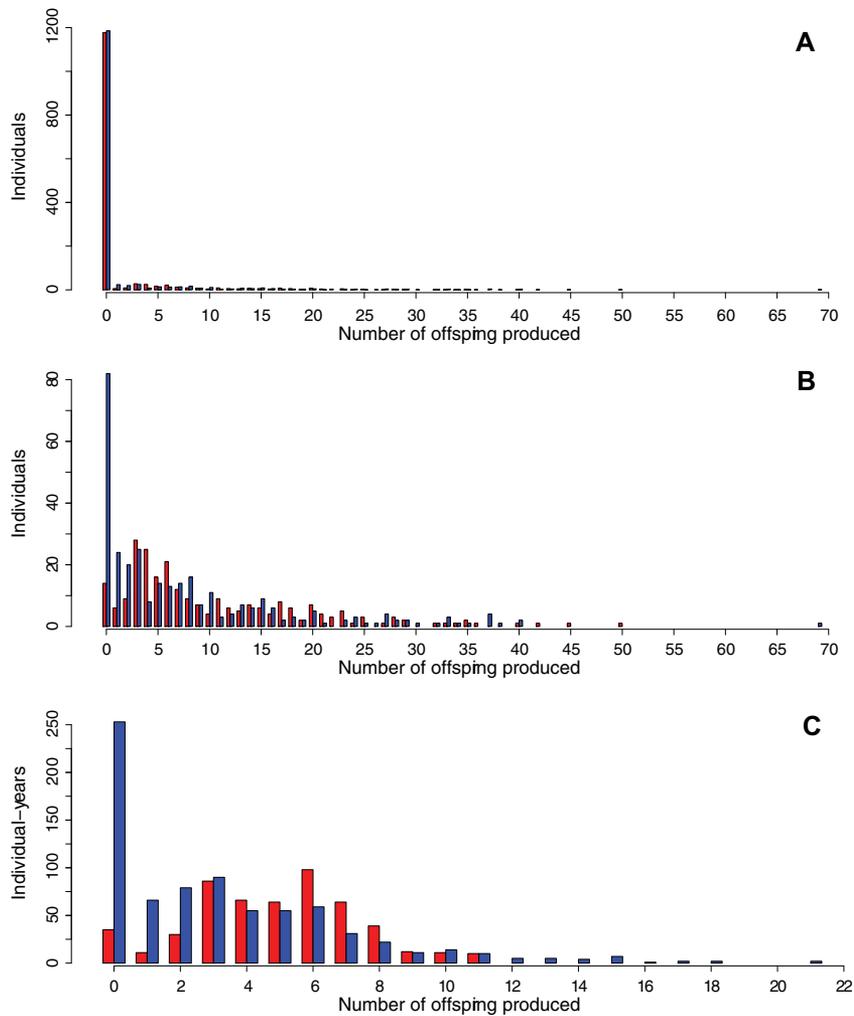
### FITNESS

Across 1406 female and 1415 male chicks banded on Mandarte during 1993–2012, 1177 (83.7%) and 1185 (83.7%) respectively had zero fitness. Consequently, fitness distributions were strongly right-skewed, with maxima of 50 and 69 banded offspring for females and males, respectively (Fig. 1A). Raw mean sex-specific fitness was 1.78 and 1.70, respectively, with substantial phenotypic variances (females 29.8, males 31.7).

In the bivariate QGGLMM, the posterior distributions for latent-scale  $V_A$  in female and male fitness showed clear peaks that were substantially shifted away from zero and from the prior distributions, indicating substantial  $V_A$  for sex-specific fitness (Fig. 2A,B). The posterior modes were similar in both sexes, and the lower 95%CI limits did not converge toward zero (Table 1). There was nonzero cohort variance and substantial residual variance in both sexes, reflecting the overdispersed phenotypic distributions (Table 1, Fig. 1). Consequently, there was relatively small but nonzero heritability of fitness in both sexes; posterior modes and means for  $h^2_{\text{latent}}$  were 0.08–0.09, with lower 95%CI limits that did not converge to zero (Table 1, Fig. S2).

The posterior mode for the cross-sex  $COV_A$  in fitness was positive, generating a posterior mode for the cross-sex  $r_A$  of intermediate magnitude between zero and one (Table 1, Fig. 2C). The 95%CI for  $r_A$  was wide and included zero. However, 88% of the posterior density exceeded zero, representing substantial divergence from the uniform prior density, yet the upper 95%CI limit did not converge toward one (Table 1, Fig. 2C). This implies that fitness variation most probably has some, but not all, of the same additive genetic basis in females and males.

In total, 26 immigrants that arrived on Mandarte during 1976–2012 made nonzero expected genetic contribution to the 2821 Mandarte-hatched individuals whose fitness was observed (Appendix S3). Across these 2821 individuals, mean *IGG* coefficient was  $0.52 \pm 0.13SD$  (range 0.14–0.86). Approximately half the focal individuals' genomes are therefore expected to have originated from immigrants on average, implying that immigration could contribute substantially to standing  $V_A$  within the Mandarte breeding population. The posterior modes for the regressions of sex-specific fitness on *IGG*, which quantify mean immigrant

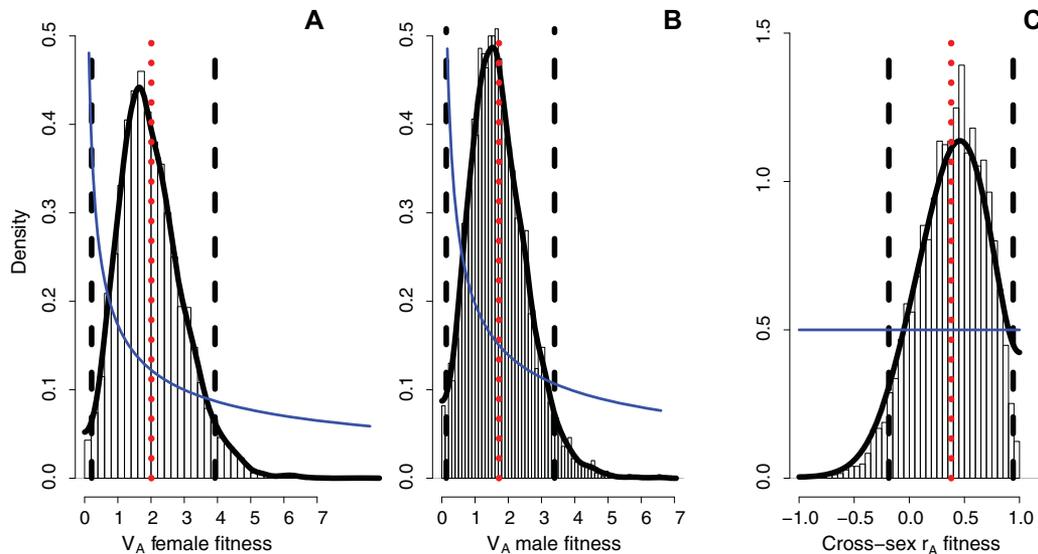


**Figure 1.** Phenotypic distributions of (A) fitness, (B) adult lifetime reproductive success, and (C) adult annual reproductive success measured as the number of banded chicks attributed to each focal individual. Red and blue denote females and males, respectively.

**Table 1.** Marginal posterior means, modes (in square brackets), and 95% credible intervals (in parentheses) for latent-scale estimates from the bivariate model for female and male fitness.

	Additive genetic matrix		Cohort matrix		$V_R$	$h^2_{\text{latent}}$	$\beta_f$	$\beta_{IGG}$
	Female fitness	Male fitness	Female fitness	Male fitness				
Female fitness	$V_A = \mathbf{2.01}$ [1.56] (0.21, 3.93)	$COV_A = 0.62$ [0.42] (-0.43, 1.82)	$\mathbf{3.12}$ [2.46] (0.90, 5.98)	$COV = 1.46$ [1.16] (0.12, 2.97)	16.58 [15.29] (12.99, 21.06)	0.09 [0.08] (0.02, 0.18)	-21.41 [-19.91] (-32.53, -11.48)	-6.27 [-5.79] (-11.36, -2.00)
Male fitness	$r_A = 0.38$ [0.45] (-0.19, 0.94)	$VA = \mathbf{1.72}$ [1.70] (0.13, 3.39)	$r = 0.67$ [0.79] (0.27, 0.98)	$\mathbf{1.54}$ [0.97] (0.38, 3.17)	15.61 [15.60] (11.86, 19.31)	0.09 [0.08] (0.01, 0.17)	-27.86 [-25.53] (-39.47, -16.50)	-5.47 [-5.19] (-9.91, -0.71)

Within the additive genetic and cohort matrices, sex-specific variances are shown along the diagonal (bold) with cross-sex covariances (COV) and correlations ( $r$ , italics) above and below the diagonal, respectively. Sex-specific residual variances ( $V_R$ ), latent scale heritabilities ( $h^2_{\text{latent}}$ ), and slopes of regressions on individual coefficient of inbreeding ( $\beta_f$ ) and immigrant genetic group coefficient ( $\beta_{IGG}$ ) are also shown.



**Figure 2.** Marginal posterior MCMC samples (bars), kernel density estimation (solid black line), posterior mean (red dotted line), 95% credible interval limits (black dashed lines), and prior (solid blue line) for the additive genetic variances ( $V_A$ ) in (A) female fitness, (B) male fitness, and (C) the cross-sex additive genetic correlation ( $r_A$ ) in song sparrows. In A and B, the priors are depicted over the range of each posterior distribution, but extend to substantial positive values.

genetic group effects, were negative in both sexes with 95% CIs that did not overlap zero (Table 1). Additive effects of alleles carried by immigrants therefore decreased fitness, relative to additive effects of alleles in the defined founder population, in both sexes.

Across the 2821 individuals, mean  $f$  was  $0.074 \pm 0.052$  (range 0.000–0.347, 7.4% zeroes). Substantial variation in  $f$  was directly attributable to immigration: 91% of individuals with  $f = 0$  had one immigrant parent. However, since immigrants' descendants commonly inbred in future generations, the model covariates  $f$  and  $IGG$  were only moderately correlated across individuals (Pearson correlation coefficients: females  $r = -0.25$ , males  $r = -0.30$ ). The posterior modes for the regressions of sex-specific fitness on  $f$  were negative with 95% CIs that did not overlap zero, demonstrating very strong inbreeding depression in fitness in both sexes (Table 1).

#### JUVENILE SURVIVAL AND ADULT LIFETIME REPRODUCTIVE SUCCESS

Of 1542 female and 1562 male chicks banded during 1993–2014, 254 (16.5%) females and 331 (21.2%) males survived on Mandarte to the following April. Adult LRS was measured for 243 adult females and 312 adult males hatched during 1993–2012, with sex-specific means of 10.3 (median 7, variance 85.1, 5.8% zeroes) and 7.7 (median 4, variance 97.6, 26.3% zeroes) banded offspring, respectively (Fig. 1B).

In the trivariate QGGLMM, the posterior distribution for  $V_A$  in juvenile survival showed a clear peak, and hence posterior mean, that departed from zero and from the prior distribution.

However, the lower 95% CI limit converged toward zero, and there was a second peak of posterior density near zero that mirrored the prior distribution (Table 2, Fig. 3A). Since there was substantial cohort variance (Table 2), the posterior means for  $h^2_{\text{latent}}$  and  $h^2_{\text{observed}}$  were small, but again showed clear peaks away from zero (Fig. S3). Although the lower 95% CI limits converged toward zero, approximately 93% and 82% of posterior samples for  $h^2_{\text{latent}}$  and  $h^2_{\text{observed}}$ , respectively, exceeded a minimal value of 0.01 (Table 2, Fig. S3).

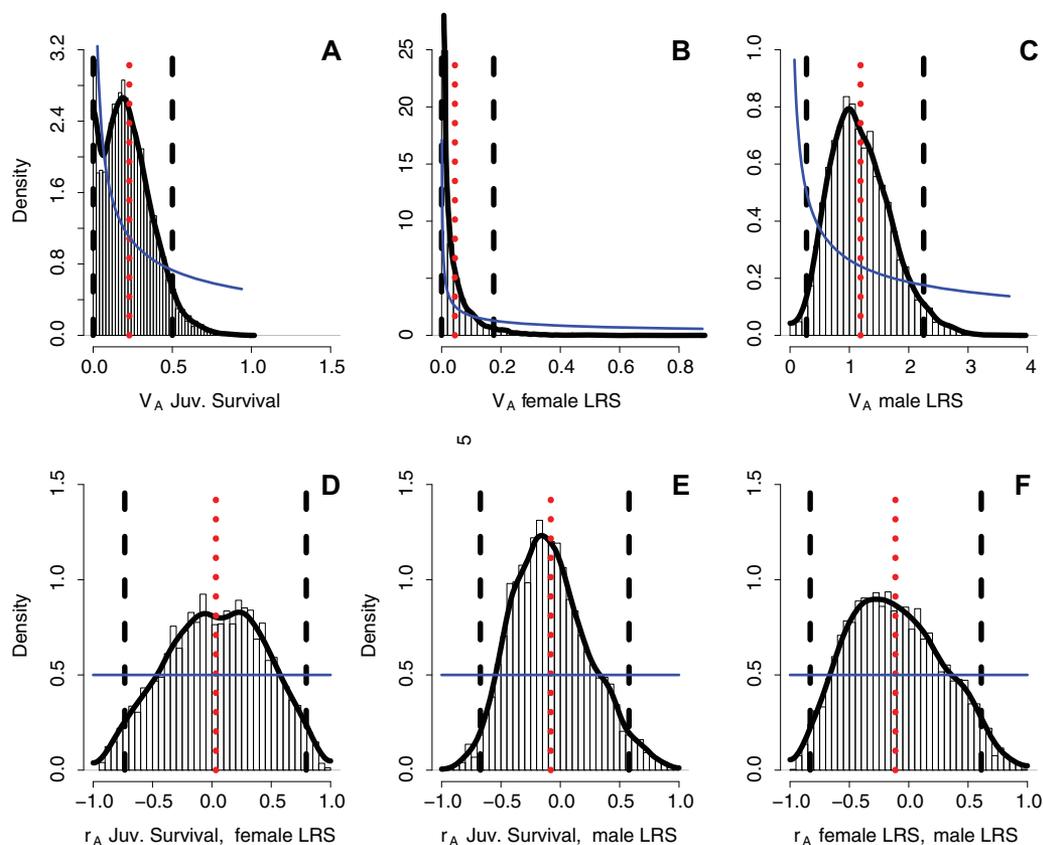
The posterior mode for  $V_A$  in adult female LRS was very small (Table 2). The posterior mean was slightly greater due to the right-skewed posterior distribution (Table 2, Fig. 3B). However, there was substantial posterior density close to zero compared to the prior distribution, and the lower 95% CI limit converged towards zero (Fig. 3B, Table 2). Consequently, the posterior modes (and means) of  $h^2_{\text{latent}}$ ,  $h^2_{\text{observed}}$  and  $I_{A\text{-observed}}$  for female LRS were small, with lower 95% CI limits that converged towards zero (Table 2, Figs. S4, S5).

In marked contrast, the posterior mode and mean for  $V_A$  in adult male LRS were substantial and the lower 95% CI limit considerably exceeded zero (Table 2, Fig. 3C). Consequently, although there were also moderate cohort and residual variances, the posterior mode and mean for  $h^2_{\text{latent}}$  for male LRS were substantial (Table 2, Fig. S4). These values were smaller for  $h^2_{\text{observed}}$ , reflecting the nonlinear transformation induced by the mean-variance relationship of the Poisson distribution, but the lower 95% CI limit still did not converge toward zero (Table 2, Fig. S4). The posterior mode for  $I_{A\text{-observed}}$  for male LRS was also moderate (Table 2, Fig. S5). Overall, there was substantially more

**Table 2.** Marginal posterior means, modes (in square brackets), and 95% credible intervals (in parentheses) for latent- and observed-scale estimates from the trivariate model for juvenile survival and adult female and male lifetime reproductive success (LRS).

Additive genetic matrix		Cohort matrix										
	Juvenile survival	Female LRS	Male LRS	Juvenile survival	Female LRS	Male LRS	$V_R$	$h^2_{latent}$	$h^2_{observed}$	$I_{A-observed}$	$\beta_f$	$\beta_{GG}$
Juvenile survival	$V_A = 0.23$ [0.002] (<0.001, 0.50)	$COV_A = 0.001$ [<0.001] (-0.08, 0.08)	$COV_A = -0.06$ [0.01] (-0.41, 0.24)	0.73 [0.71] (0.27, 1.28)	$COV = -0.02$ [<0.001] (-0.17, 0.08)	$COV = -0.11$ [<0.001] (-0.43, 0.21)	1 (fixed)	0.09 [0.001] (<0.001, 0.18)	0.03 [<0.001] (<0.001, 0.05)	NA	-9.31 [<0.001] (-12.85, -5.61)	-2.58 [<0.001] (-4.26, -0.85)
Female LRS	$r_A = 0.03$ [<0.001] (-0.73, 0.80)	$V_A = 0.05$ [0.001] (<0.001, 0.18)	$COV_A = -0.04$ [<0.001] (-0.26, 0.13)	$r = -0.17$ [<0.001] (-0.48, 0.63)	<b>0.03</b> [<0.001] (<0.001, 0.11)	$COV = 0.01$ [<0.001] (-0.07, 0.10)	0.72 [0.71] (0.54, 0.93)	0.05 [0.001] (<0.001, 0.21)	0.03 [<0.001] (<0.001, 0.13)	0.05 [0.001] (<0.001, 0.18)	-1.89 [<0.001] (-5.34, 1.43)	0.32 [0.40] (-0.85, 1.69)
Male LRS	$r_A = -0.08$ [<0.001] (-0.67, 0.58)	$r_A = -0.11$ [<0.001] (-0.83, 0.61)	$V_A = 1.19$ [1.00] (0.28, 2.25)	$r = -0.23$ [<0.001] (-0.72, 0.33)	$r = 0.11$ [0.06] (-0.61, 0.83)	<b>0.35</b> [0.26] (0.02, 0.79)	1.12 [0.99] (0.49, 1.80)	0.44 [0.38] (0.15, 0.74)	0.09 [0.09] (0.03, 0.15)	1.19 [0.99] (0.24, 2.20)	-9.00 [<0.001] (-15.04, -3.15)	-0.41 [<0.001] (-3.35, 2.44)

Within the additive genetic and cohort matrices, variances are shown along the diagonal (bold) with covariances (COV) and correlations ( $r$ , italics) above and below the diagonal respectively. Residual variances ( $V_R$ ), latent-scale heritabilities ( $h^2_{latent}$ ), observed-scale heritabilities ( $h^2_{observed}$ ), observed-scale evolvabilities ( $I_{A-observed}$ ), and slopes of regressions on individual coefficient of inbreeding ( $\beta_f$ ) and immigrant genetic group coefficient ( $\beta_{GG}$ ) are also shown.  $I_{A-observed}$  is not applicable (NA) for juvenile survival. Posterior modes and lower 95%CI limits that converged towards zero are reported as <0.001.



**Figure 3.** Marginal posterior MCMC samples (bars), kernel density estimation (solid black line), posterior mean (red dotted line), 95% credible interval limits (black dashed lines), and prior (solid blue line) for the additive genetic variances ( $V_A$ ) in (A) juvenile survival, (B) adult female lifetime reproductive success (LRS), and (C) adult male LRS, and the additive genetic correlations ( $r_A$ ) between (D) juvenile survival and adult female LRS, (E) juvenile survival and adult male LRS, and (F) adult female and male LRS in song sparrows. Note that axis scales vary among plots. In A–C, the priors are depicted over the range of each posterior distribution, but extend to substantial positive values.

$V_A$  in adult male LRS than adult female LRS, as the posterior distribution of the male–female difference in  $V_A$  had a posterior mean of 1.14 (mode = 0.97, 95%CI limits: 0.19, 2.15).

Since  $V_A$  in female LRS was so small and the lower 95%CI limit for  $V_A$  in juvenile survival also converged toward zero, the pairwise  $COV_{AS}$  and  $r_{AS}$  among juvenile survival and female and male LRS were unsurprisingly estimated with considerable uncertainty (Table 2, Fig. 3). The posterior modes and means for  $r_A$  between juvenile survival and male LRS, and between female and male LRS, were slightly negative, but spanned zero for juvenile survival and female LRS, all with 95%CI limits that did not converge towards either  $-1$  or  $1$  (Table 2, Fig. 3).

Distributions of  $IGG$  and  $f$  for individuals included in analyses of juvenile survival and adult LRS (and ARS and survival) were quantitatively similar to those for individuals included in analyses of fitness (summarized above). The posterior mode for the regression of juvenile survival on  $IGG$  was negative, with a 95%CI that did not overlap zero (Table 2). Further analyses showed similar negative slopes for female and male juvenile

survival modeled as separate traits (Appendix S7). However, the posterior modes for the regressions of adult female and male LRS on  $IGG$  were small, with 95%CIs that spanned zero (Table 2). This implies that additive effects of immigrants' alleles decreased local juvenile survival, but not adult female or male LRS, relative to additive effects of founders' alleles.

The posterior modes for the regressions of juvenile survival and adult female and male LRS on  $f$  were all negative, demonstrating inbreeding depression (although the 95%CI for female LRS overlapped zero, Table 2). Further, inbreeding depression in LRS is most likely stronger in males than females (Table 2), as the posterior distribution of the male–female difference in  $f$  had a posterior mean of  $-7.12$  (mode =  $-5.51$ , 95%CI limits:  $-14.0$ ,  $-0.45$ ).

#### ADULT ANNUAL REPRODUCTIVE SUCCESS

During 1994–2015, there were 526 and 773 observations of ARS for adult females and males respectively, involving 254 and 331 Mandarte-hatched individuals. Mean female ARS was

4.9 banded offspring (median 5, variance 6.2, range 0–11, 6.7% zeroes, Fig. 1C) and mean male ARS was 3.2 banded offspring (median 2, variance 13.2, range 0–21, 32.7% zeroes, Fig. 1C).

In the bivariate QGGLMM, the posterior mode for  $V_A$  in female ARS was very small and the lower 95%CI limit converged toward zero (Table 3, Fig. 4A). However, there was a clear secondary peak away from zero (Fig. 4A), meaning that the posterior mean was slightly larger (Table 3), and 75% of the posterior density exceeded a minimal value of 0.01. Further, the posterior density near zero resembles the prior distribution, suggesting that the prior influences the posterior mode, while the data generate the second peak about the posterior mean. This implies the existence of very small, but probably nonzero,  $V_A$  for female ARS (Fig. 4A inset).

In contrast, the posterior mode and mean for  $V_A$  in male ARS were substantially larger and the lower 95%CI limit did not converge towards zero (Table 3, Fig. 4B). The permanent individual variances were very small in both sexes, but the year and residual variances were substantial, especially for males (Table 3). Consequently, despite the marked difference in  $V_A$ , the posterior means for  $h^2_{\text{latent}}$  and  $h^2_{\text{observed}}$  for ARS were similar for both sexes ( $\sim 0.06$ – $0.18$ ), but  $I_{A\text{-observed}}$  was substantially greater for male ARS than female ARS (Table 3, Figs. S6, S7).

The posterior mode for the cross-sex additive genetic correlation ( $r_A$ ) in ARS was positive but small. Due to the small  $V_A$  in female ARS, the 95%CI was again wide and spanned zero, but did not converge toward either  $-1$  or  $1$  (Table 3, Fig. 4C).

The posterior modes for the regressions of ARS on *IGG* were small in both sexes, with 95%CIs that overlapped zero (Table 3). The posterior modes for the regressions of ARS on *f* were negative in both sexes, although the 95%CI for females again overlapped zero (Table 3). Again, inbreeding depression is most likely stronger in male than female ARS (Table 3), as the posterior distribution of the male–female difference in *f* had a posterior mean of  $-3.69$  (mode =  $-3.43$ , 95%CI limits:  $-6.93$ ,  $-0.57$ ).

#### ADULT ANNUAL SURVIVAL

For the focal 254 adult females and 331 adult males, the mean number of observations of annual survival (or mortality) was 2.1 (median 1, range 1–9, Fig. 5A) for females and 2.3 (median 2, range 1–9, Fig. 5B) for males, representing overall annual survival of 53.0 and 58.0%, respectively.

In the univariate QGGLMM, the posterior mode for  $V_A$  was effectively zero (Table 3, Fig. 5C). The posterior mean was slightly larger, but there was substantial posterior density close to zero compared to the prior distribution, and the lower 95%CI limit converged to zero (Table 3). Since there was also substantial year variance, the posterior modes for  $h^2_{\text{latent}}$  and  $h^2_{\text{observed}}$  were very small (Table 3; Fig. S8.3). The posterior modes for the regressions of adult annual survival on *IGG* and *f* were also small,

with 95%CIs that overlapped zero (Table 3). Analyses of adult longevity rather than annual survival, and of sex-specific annual survival, yielded similar conclusions (Appendix S8).

## Discussion

### ADDITIVE GENETIC VARIANCE AND CORRELATION IN SEX-SPECIFIC FITNESS

The sex-specific additive genetic variances ( $V_A$ ) in fitness, and the cross-sex genetic correlation ( $r_A$ ), are key parameters that determine the rate of fitness evolution and shape evolutionary responses to natural and sexual selection (Burt 1995; Brommer et al. 2007; Kirkpatrick 2009; Shaw and Shaw 2014). They also underlie the potential for evolutionary sexual conflict, which might constrain evolution yet help maintain overall  $V_A$  in fitness (Lande 1980; Chippindale et al. 2001; Kruuk et al. 2008; Bonduriansky and Chenoweth 2009; Long et al. 2012). However, these key parameters have rarely been estimated in wild populations, particularly using theoretically appropriate measures of fitness while accommodating non-Gaussian phenotypic distributions and accounting for genetic effects of immigration and inbreeding (Kruuk et al. 2008; Kirkpatrick 2009; Shaw and Etersson 2012; Gomulkiewicz and Shaw 2013; Shaw and Shaw 2014).

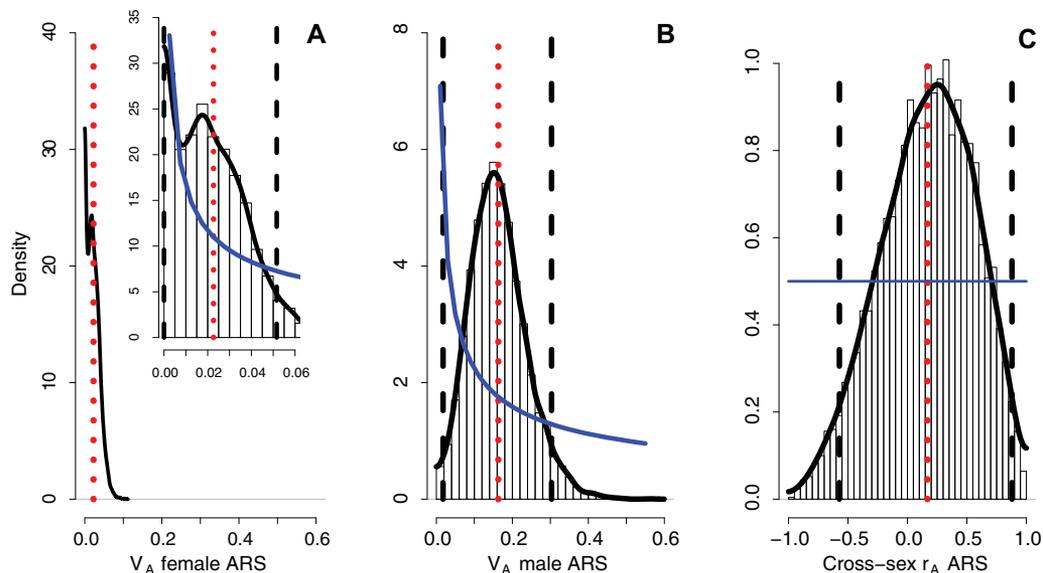
Our analyses of comprehensive fitness data from free-living song sparrows estimated nonzero latent-scale  $V_A$ s and heritabilities for fitness, of similar magnitudes, in both sexes. Such estimates do not concur with the common assumption that  $V_A$  for fitness is usually negligible (Charlesworth 1987; Shaw and Shaw 2014; Walling et al. 2014). Instead, our estimates support the view that nontrivial  $V_A$  in fitness can be readily generated and/or maintained in wild populations (e.g., Houle 1992; Kirkpatrick 2009; Zhang 2012; Shaw and Shaw 2014). Further, our inference that the cross-sex  $r_A$  for fitness is most likely to be positive implies that some  $V_A$  is shared between the sexes, potentially facilitating an increase in population mean fitness (Lande 1980). However, the upper 95%CI limit for the cross-sex  $r_A$  in fitness was less than one. For the special case of fitness, this implies that some sex-limited or sexually antagonistic genetic variation does exist, potentially facilitating the maintenance of overall  $V_A$ .

The few available estimates of sex-specific  $V_A$  in fitness in wild populations cannot readily be compared quantitatively because different studies used different fitness metrics, analytical methods, and estimation scales, with different degrees of paternity error and missing data. However, qualitatively concordant with our results,  $V_A$  for fitness was estimated to be nonzero and similar in both sexes in collared flycatchers (*Ficedula albicollis*, Merilä and Sheldon 2000; Brommer et al. 2007) and Swedish humans (*Homo sapiens*, Zietsch et al. 2014). In contrast,  $V_A$  was estimated to be zero or very small in both sexes in great tits (*Parus*

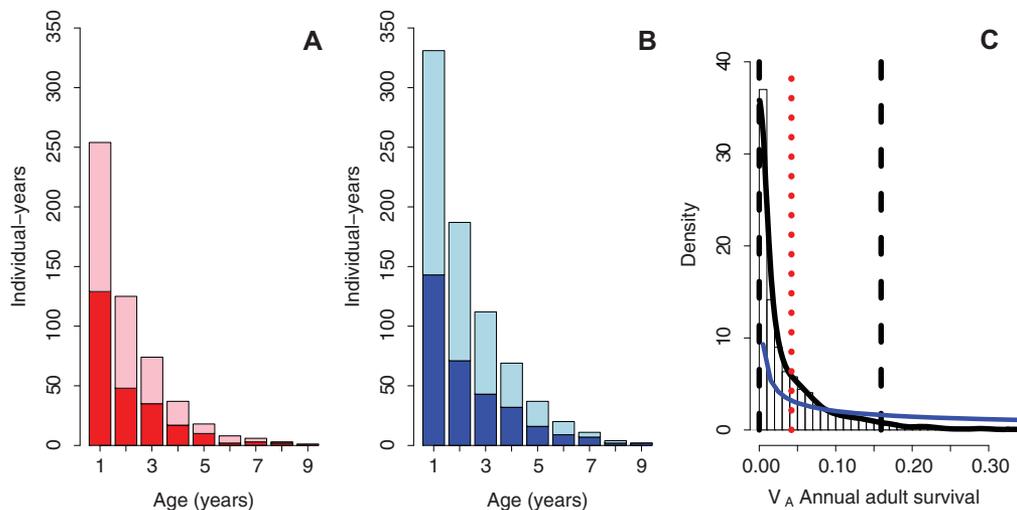
**Table 3.** Marginal posterior means, modes (in square brackets), and 95% credible intervals (in parentheses) from (A) the bivariate model for adult female and male annual reproductive success (ARS) and (B) the univariate model for adult annual survival.

	Additive genetic matrix				Year matrix				$\beta_f$	$\beta_{GG}$	
	Female ARS	Male ARS	$V_{PI}$	Female ARS	Male ARS	$V_R$	$h^2_{latent}$	$h^2_{observed}$			$I_A$ -observed
Female ARS	$V_A = 0.02$ [<0.001]	$COV_A = 0.01$ [0.001]	0.01 [<0.001]	<b>0.04</b> [0.03]	$COV = 0.07$ [0.05]	0.05 [0.05]	0.18 [0.001]	0.06 [<0.001]	0.02 [<0.001]	-1.29 [-1.19]	0.22 [0.20]
	(<0.001, 0.05)	(-0.04, 0.06)	(<0.001, 0.04)	( <b>0.01, 0.08</b> )	(0.01, 0.15)	(0.04, 0.07)	(<0.001, 0.38)	(<0.001, 0.15)	(<0.001, 0.05)	(-2.82, 0.23)	(-0.38, 0.84)
Male ARS	$r_A = 0.17$ [0.18]	$V_A = 0.16$ [0.15]	0.05 [<0.001]	$r = 0.67$ [0.78]	<b>0.34</b> [0.25]	0.46 [0.46]	0.16 [0.12]	0.08 [0.08]	0.67 [0.40]	-4.98 [-4.98]	0.15 [0.11]
	(-0.57, 0.88)	(0.02, 0.30)	(<0.001, 0.15)	(0.30, 0.96)	( <b>0.11, 0.64</b> )	(0.34, 0.58)	(0.03, 0.31)	(0.01, 0.15)	(0.01, 1.64)	(-7.94, -2.07)	(-1.18, 1.61)
Adult annual survival	0.04 [<0.001]		0.37 [0.01]	0.69 [0.54]		1 (fixed)	0.02 [<0.001]	0.005 [<0.001]	NA	-0.49 [0.03]	0.33 [0.46]
	(<0.001, 0.16)		(<0.001, 1.44)	(0.20, 1.31)			(<0.001, 0.07)	(<0.001, 0.02)		(-4.81, 3.83)	(-1.48, 2.10)

Within the additive genetic and year matrices for ARS, variances are shown along the diagonal (bold) with covariances (COV) and correlations ( $r$ , italics) above and below the diagonal, respectively. Permanent individual ( $V_{PI}$ ) and residual ( $V_R$ ) variances, latent-scale heritabilities ( $h^2_{latent}$ ), observed-scale heritabilities ( $h^2_{observed}$ ) and evolvabilities ( $I_A$ -observed), and slopes of regressions on individual coefficient of inbreeding ( $\beta_f$ ) and immigrant genetic group coefficient ( $\beta_{GG}$ ) are also shown.  $I_A$  is not applicable (NA) for adult annual survival. Posterior modes and lower 95%CI limits that converged toward zero are reported as < 0.001.



**Figure 4.** Marginal posterior MCMC samples (bars), kernel density estimation (solid black line), posterior mean (red dotted line), 95% credible interval limits (black dashed lines), and prior (solid blue line) for the additive genetic variances ( $V_A$ ) in (A) adult female annual reproductive success (ARS), (B) adult male ARS, and (C) the cross-sex additive genetic correlation ( $r_A$ ) in song sparrows. On A and B, x-axis scales are standardized to facilitate comparison, but the y-axis scales differ. The panel A inset shows the marginal posterior distribution for female ARS on a larger scale. In A and B, the priors are depicted over the range of each posterior distribution, but extend to substantial positive values.



**Figure 5.** Phenotypic distributions of age-specific survival (or mortality) for adult (A) female and (B) male song sparrows, and (C) the marginal posterior distribution for additive genetic variance ( $V_A$ ) in adult annual survival. In A and B, dark and light shading indicate observations of mortality and survival, respectively. In C, plot attributes are as for Figures 2–4.

major, McCleery et al. 2004), bighorn sheep (*Ovis canadensis*, Coltman et al. 2005), North American red squirrels (*Tamiasciurus hudsonicus*, McFarlane et al. 2014), and savannah sparrows (*Passerculus sandwichensis*, Wheelwright et al. 2014); zero in females but more substantial in males in red deer (*Cervus elaphus*, Kruuk et al. 2000, but see Foerster et al. 2007) and Austrian humans (Gavrus-Ion et al. 2017); yet zero in males but more substantial in females in red-billed gulls (*Larus novaehollandiae*,

Teplitsky et al. 2009) and preindustrial Finnish humans (Pettay et al. 2005, Appendix S1).

Meanwhile, the posterior 95%CI for the cross-sex  $r_A$  for fitness in song sparrows excluded the substantial negative values previously estimated in wild populations (Foerster et al. 2007; Brommer et al. 2007; McFarlane et al. 2014; Appendix S1), with relatively little posterior density surrounding the small or slightly negative values estimated in laboratory populations (Chippindale

et al. 2001; Delcourt et al. 2009; Innocenti and Morrow 2010; Collet et al. 2016). Yet, cross-sex  $r_A$ s can change substantially when (laboratory) populations experience novel environments (Delcourt et al. 2009; Punzalan et al. 2014; Collet et al. 2016), migration load (Long et al. 2012), or inbreeding (Duffy et al. 2014). Positive values, such as those inferred in the song sparrows, might indicate populations where both sexes are displaced from their fitness peak, and consequently experience congruent directional selection (Long et al. 2012; Duffy et al. 2014; Punzalan et al. 2014). Overall, further rigorous and standardized estimates of  $V_A$  and  $r_A$  in sex-specific fitness from wild populations experiencing different ecological circumstances are clearly required to discern general patterns and evolutionary implications.

### ADDITIVE GENETIC VARIANCES AND CORRELATIONS IN FITNESS COMPONENTS

Values of  $V_A$  in sex-specific fitness, and the cross-sex  $r_A$ , must ultimately result from  $V_{AS}$  and cross-sex and within-sex  $r_{AS}$  in underlying sex-specific fitness components. Quantifying such parameters can consequently help identify mechanisms that maintain  $V_A$  in fitness, and identify sources of sexual conflict (Walling et al. 2014). Juvenile survival constitutes one primary fitness component. Indeed, 96% of observed song sparrow fitness values of zero represent individuals that did not (locally) survive to adulthood, generating the high frequency of zeroes in the overall fitness distribution. Such patterns are likely commonplace (Blomquist 2010; Wagenius et al. 2010; Gomulkiewicz and Shaw 2013). We therefore explicitly estimated  $V_A$  in juvenile survival, showing a moderate posterior mean with a clear peak of posterior density away from zero, but a lower 95%CI that converged toward zero. This broadly concurs with previous evidence that  $V_A$  in juvenile survival is moderate and similar in female and male song sparrows with a substantial positive cross-sex  $r_A$  (Reid and Sardell 2012, Appendix S7).

However, for adult LRS, which constitutes the remaining primary fitness component, there was a striking difference between the sexes:  $V_A$  for male LRS was substantial and clearly exceeded zero, while  $V_A$  for female LRS was very small. This implies that there is little opportunity for rapid evolutionary change in adult female LRS, and hence in other traits that are postulated to be genetically correlated with female reproductive success. The potential for evolution in mean absolute male LRS will consequently also be constrained. Yet, the large  $V_A$  in absolute male LRS estimated on the QGGLMM latent scale implies that relative male LRS on the observed scale will also exhibit  $V_A$ . Consequently, there could be rapid evolutionary change in relative male LRS, and hence in genetically correlated traits. This includes traits that shape mating systems, for example male acquisition of reproductive success through within-pair versus extra-pair paternity (Reid and Wolak 2018).

The small  $V_A$  in female LRS impedes precise estimation of the cross-sex  $r_A$  in LRS, and indeed renders such estimation somewhat redundant (since  $r_A$  is undefined if  $V_A$  is truly zero in one or both sexes). Nevertheless, the cross-sex  $r_A$  posterior mode was close to zero, further suggesting that additive genetic effects on adult LRS are largely independent in females and males. Together, our results imply that the moderate positive cross-sex  $r_A$  in fitness is primarily driven by the moderate positive cross-sex  $r_A$  in juvenile survival. Hence, cross-sex expression of additive genetic effects on juvenile survival ameliorates potential sexually antagonistic genetic variation in overall fitness resulting from sex-specific expression of adult LRS. These patterns are reminiscent of those observed in *Drosophila melanogaster*, where a positive cross-sex  $r_A$  in juvenile survival initially combined with a negative cross-sex  $r_A$  in adult reproductive success to generate a weak overall cross-sex  $r_A$  for fitness (Chippindale et al. 2001), but where the cross-sex  $r_A$  in adult reproductive success was no longer detectably different from zero after further generations of laboratory adaptation (Collet et al. 2016).

Further decomposition of adult LRS in song sparrows revealed little or no  $V_A$  in adult annual survival, and identified ARS as the primary source of  $V_A$  in male LRS. The substantial difference in  $V_A$  in ARS, and hence LRS, between males and females likely reflects the population's ecology and mating system. Due to the typically male-biased adult sex-ratio and frequent extra-pair paternity, males accumulate ARS by securing a territory and a social mate, defending within-pair paternity and accruing extra-pair paternity (Sardell et al. 2010; Lebigre et al. 2012; Reid et al. 2011a, 2014a,b; Losdat et al. 2015). In contrast, females accumulate ARS through their own fecundity. Consequently, while components of ARS such as within-pair paternity can be conceptualized as "emergent" traits of pairs rather than individuals (Reid et al. 2014a), males and females are likely to differ substantially in the suite of physiological and behavioral traits that generate high ARS, and hence in underlying genetic effects. Previous analyses revealed nonzero  $V_A$  in components of annual male extra-pair reproductive success and a positive  $r_A$  with within-pair paternity success per brood (Reid et al. 2014b; Losdat et al. 2015), but a negative  $r_A$  between net paternity success and juvenile survival (Reid and Sardell 2012). Together, these positive and negative correlations, alongside among-year variation in adult sex-ratio and hence the social context in which male reproductive success is expressed, could help maintain substantial  $V_A$  in male ARS (and hence LRS, Reid and Wolak 2018).

### GENETIC EFFECTS OF IMMIGRATION

Immigration, and resulting gene flow, is one primary mechanism that can maintain  $V_A$  in fitness and associated evolutionary potential in any population, and also rapidly increase mean fitness by alleviating inbreeding depression. However, the overall genetic

effects of immigration, and the evolutionary consequences, depend on genetic properties of naturally occurring immigrants compared to existing natives (Ingvarsson and Whitlock 2000; Tallmon et al. 2004; Edelaar and Bolnick 2012; Carlson et al. 2014). We utilized the multigeneration song sparrow pedigree, that links all Mandarte-hatched individuals to their immigrant and “founder” ancestors and hence describes expected introgression of immigrants’ alleles, to directly estimate the relative mean additive genetic values for local fitness of the defined immigrant and founder genetic groups. Unlike analyses that examine demographic and evolutionary consequences of dispersal by directly comparing observed phenotypes of immigrants (or dispersers) and residents (e.g., Marr et al. 2002; Pasinelli et al. 2004; Nosil et al. 2005; Pärn et al. 2009; Bonte et al. 2012), our analyses do not utilize immigrants’ own phenotypes and consequently cannot be confounded by environmental effects of dispersal on those phenotypes. Our analyses showed that immigrant song sparrows carry alleles that, when expressed in subsequent Mandarte-hatched generations, have negative additive effects on local fitness in both sexes.

Such effects could stem from three main processes. First, there could be divergent selection among song sparrow demes and resulting local adaptation. Immigrants to Mandarte might consequently be locally maladapted and hence have low mean additive genetic value for local fitness, as assumed by classical migration load models. Second, dispersal could be nonrandom, such that immigrants have low additive genetic value for local fitness even without any local adaptation. Third, low additive genetic value for fitness measured on Mandarte could reflect  $V_A$  in dispersal, such that offspring of immigrants are more likely to emigrate and hence have zero local fitness (e.g., Doligez and Pärt 2008). These three processes are not mutually exclusive and are not distinguished by our current analyses. However, the overall effects resulted primarily from immigrants’ low additive genetic value for local juvenile survival, and therefore reflects some combination of effects on early-life mortality and/or emigration. To indicate biological effects, the estimated latent-scale effect of  $\beta_{IGG} = -2.6$  (Table 2) implies a decrease in local juvenile survival probability of approximately 0.04 given an increase in individual *IGG* coefficient of 0.1 spanning the current mean of  $\sim 0.5$ , which is not trivial. In general, such reduced local survival of immigrants’ descendants would reduce the effective rate of gene flow below that expected given the observed immigration rate (Garant et al. 2007).

However, our analyses also demonstrate very strong inbreeding depression in fitness in both sexes, resulting from inbreeding depression in juvenile survival and in adult LRS and ARS, particularly in males. Similar patterns of inbreeding depression have previously been documented in the Mandarte population, using different data subsets and methods (Keller 1998; Reid et al. 2014c; Nietlisbach et al. 2017). This inbreeding

depression reflects covariance between individual fitness and  $f$ , where the underlying variance in  $f$  stems from immigrant-native outcrossing; resulting F1 offspring are defined as outbred and have relatively high fitness (Keller 1998; Marr et al. 2002; Reid et al. 2006, 2014c; Wolak and Reid 2016). The estimated latent-scale effect size of  $\beta_f = -9.3$  (Table 2) implies an increase in juvenile survival probability of approximately 0.25 for outbred offspring ( $f = 0$ ) compared to inbred offspring with  $f = 0.1$  (see also Keller 1998; Reid et al. 2014c). This effect could cause rapid initial introgression of immigrants’ alleles, and hence increase the short-term effective rate of gene flow (e.g., Ingvarsson and Whitlock 2000; Garant et al. 2007; Hedrick et al. 2014). Indeed, the mean *IGG* coefficient of  $\sim 0.5$ , calculated across the focal 2821 fitness-phenotyped individuals, implies that an average Mandarte-hatched song sparrow inherited half its genome from immigrant ancestors despite the relatively small number of contributing immigrants ( $n = 26$ ) and that only 195 (7%) of the phenotyped individuals were direct F1 offspring of immigrant-native pairings.

However, once immigrants’ descendants start to inbreed, as is inevitable for initially high-fitness lineages in small populations (Reid et al. 2006; Bijlsma et al. 2010; Hedrick et al. 2014), increased expression of recessive alleles with detrimental effects on local fitness would occur. This process would exacerbate the decrease in fitness that is expected following recombination in F2 and subsequent generations and resulting outbreeding depression (Marr et al. 2002; Frankham 2016). The combination of heterosis that exacerbates initial introgression and low overall additive genetic value for fitness could potentially generate substantial migration load; almost all population members might be pulled below the fitness peak, substantially decreasing population mean fitness but potentially generating a positive overall cross-sex  $r_A$  for fitness and alleviating sexual conflict (Long et al. 2012; Duffy et al. 2014; Punzalan et al. 2014). Such multigenerational dynamics of immigrants’ alleles should, in future, be explicitly quantified using pedigree and genomic data (from song sparrows and other systems), and through theory that simultaneously considers heterosis and migration load (e.g., Lopez et al. 2009). Meanwhile, our analyses demonstrate that structured quantitative genetic analyses can explicitly estimate  $V_A$  in fitness alongside multiple genetic consequences of immigration in wild populations, and thereby elucidate the contributions of gene flow to the magnitude and maintenance of overall  $V_A$  in fitness and resulting evolutionary dynamics.

#### AUTHOR CONTRIBUTIONS

M.E.W. and J.M.R. jointly conceived and designed the analyses and wrote the manuscript. M.E.W. conducted the analyses, with contributions from J.M.R. P.A. ensured the long-term field data collection. P.N. and L.F.K. conducted the genotyping. All authors contributed to editing the final draft.

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**DATA ARCHIVING**

Data have been archived in the Dryad Digital Repository: <https://doi.org/10.5061/dryad.p7p1jb3> (Wolak et al. 2018).

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## *Supporting Information*

Additional supporting information may be found online in the Supporting Information section at the end of the article.

**Appendix S1.** Literature summary.

**Appendix S2.** Overall approach and data specifications.

**Appendix S3.** Pedigree structure and genetic groups.

**Appendix S4.** Details of model specification and implementation.

**Appendix S5.** Additional details of results.

**Appendix S6.** Prior sensitivity analysis.

**Appendix S7.** Sex-specific juvenile survival.

**Appendix S8.** Additional analyses of adult survival.

**Appendix S9.** Additional analysis of adult annual survival and annual reproductive success.