Proximate causes of infertility and embryo mortality in captive zebra finches

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Data accessibility

Supporting data, model structures and R scripts can be found in the Open Science Framework at https://osf.io/tgsz8/.

Authors' contributions

W.F. and B.K. designed and planned the study. W.F., D.W. and K.M. collected reproductive performance data. Y.P. and W.F. analyzed the data with inputs from J.R. Y.P., W.F. and B.K. interpreted the results and wrote the manuscript with inputs from J.R. All authors contributed to the final manuscript.

Competing interests

We have no competing interests.

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Abstract

Some species show high rates of reproductive failure, which is puzzling because natural selection works against such failure in every generation. Hatching failure is common in both captive and wild zebra finches (*Taeniopygia guttata*), yet little is known about its proximate causes. Here we analyze data on reproductive performance (fate of >23,000 eggs) based on up to 14 years of breeding of four captive zebra finch populations. We find that virtually all aspects of reproductive performance are negatively affected by inbreeding (mean r = -0.117), by an early-starting, age-related decline (mean r = -0.132), and by poor early-life nutrition (mean r = -0.058). However, these effects together explain only about 3% of the variance in infertility, offspring mortality, fecundity and fitness. In contrast, individual repeatability of different fitness components varied between 15% and 50%. As expected, we found relatively low heritability in fitness components (median: 7% of phenotypic, and 29% of individually repeatable variation). Yet, some of the heritable variation in fitness appears to be maintained by antagonistic pleiotropy (negative genetic correlations) between male fitness traits and female and offspring fitness traits. The large amount of unexplained variation suggests a potentially important role of local dominance and epistasis, including the possibility of segregating genetic incompatibilities.

Introduction

Reproductive performance, including offspring survival, is subject to strong directional selection in every generation. Such strong selection works not only on individuals that live in their natural habitat, but also on those that live in captivity, unless artificial selection counters it. Thus, it is

68 puzzling that some populations (or species) have substantial difficulties with successful
69 reproduction, shown as high rates of infertility or embryo mortality. Prominent examples of
70 frequent reproductive failure include humans (De Braekeleer and Dao 1991; Sierra and
71 Stephenson 2006; Miyamoto et al. 2012), and other animals both in natural environments (Lyon
72 1986; Grossen et al. 2012) and in captive conditions (Ayalon 1978; Bunin et al. 2008; Gwaza et
73 al. 2016; Griffith et al. 2017). Given that directional selection constantly removes genetic
74 variants that lead to poor performance, one might suspect that reproductive failure typically
75 results from inbreeding (Briskie and Mackintosh 2004), because selection against recessive
76 deleterious mutations is inefficient, or from environmental factors (Jurewicz et al. 2009), such
77 as pollutants (Jackson et al. 2011). However, the range of possible explanations is much wider.
78
79 Reproductive failure and individual survival are complex traits and hence may be influenced by
80 multiple genetic components that can be evolutionary stable. For instance, reproductive failure
81 and mortality may be caused by selfish genetic elements that are self-promoting at the cost of
82 organismal fitness (Sandler et al. 1959; Lyon 1986; Safronova and Chubykin 2013; Lindholm et
83 al. 2016). Additive genetic variants can also be preserved under intra-locus sexual antagonism,
84 where genes that are beneficial to one sex impose detrimental effects on the other (Foerster et
85 al. 2007; Van Doorn 2009; Innocenti and Morrow 2010). Furthermore, there might be
86 evolutionary trade-offs between traits, such that individuals that invest more in reproduction
87 might show lower survival rates (Stearns 1989; Schluter et al. 1991). A few recent genetic and
88 genomic studies detected genetic variants (e.g. specific genes) involved in dominance effects or
89 rare variants that show main effects on reproductive traits (e.g. Christians et al. 2000;

Safronova and Chubykin 2013; Kim et al. 2017; Knief et al. 2017). As an extreme example, a balanced lethal system was identified in crested newts *Triturus cristatus*, where all embryos that are homozygous for chromosome 1 (about 50% of all embryos) die during development (Sims et al. 1984; Grossen et al. 2012).

Despite the development of new genomic tools, it remains difficult to identify and examine the genetic components that show antagonistic effects, or involve more than one locus, i.e. intra- and inter-locus genetic incompatibilities (Dobzhansky 1936; Fishman and Willis 2006; Johnson 2008; Eroukhmanoff et al. 2016). This difficulty is likely due to the complexity of interactions between multiple loci and between the genotype and the environment (Carrell and Aston 2011; Krausz and Riera-Escamilla 2018). If animals in captivity show high rates of reproductive failure because they are not adapted to a given artificial environment, selection can act on the standing genetic variance. This would result in a transient phase where fitness is heritable until the population is better able to cope with the new environment (e.g. due to behavioural and physiological adaptations to captivity). In general, the genetic basis of reproductive failure and variation in survival remains largely unclear in most species.

The zebra finch is a good model species to study how survival and reproductive performance of the two sexes are correlated at the additive genetic level. The zebra finch is a short-lived songbird that easily breeds in captivity (Zann 1996), and its reproductive performance varies extensively among individuals under controlled breeding conditions in both domesticated and

111 recently wild-derived populations (Griffith et al. 2017; Wang et al. 2017). In the wild, the rate of
112 hatching failure (infertile eggs and dead embryos) was estimated to be >15% (table 1). This
113 excludes clutches that failed completely, because nest desertion cannot be ruled out as the
114 reason of failure. In lab stocks, the average proportion of eggs remaining apparently
115 unfertilized ranged from 17% in aviary breeding to 30-35% in cage breeding (table 1), while
116 average embryo mortality rates varied between 24% and 75% (table 1). Average nestling
117 mortality rates were also high (table 1). Although some of the variation has been explained by
118 specific treatment effects (e.g. inbreeding, force-pairing, maternal stress; Hemmings et al. 2012;
119 Ihle et al. 2015; Khan et al. 2016), the high baseline levels of infertility, embryo and nestling
120 mortality remain largely unexplained.

121

122 To better understand this variation in reproductive performance and individual survival, we
123 here report on a comprehensive quantitative genetic analysis of lifespan, fecundity, infertility,
124 offspring mortality and other fitness-related traits that cover most phases of reproduction for
125 the two sexes (table 2). We quantified the effects of inbreeding, age and an individual's early
126 nutritional condition on all measured aspects of reproductive performance and survival.

127

128 Wild zebra finches have a remarkably large effective population size (Balakrishnan and Edwards
129 2009), where inbreeding is almost completely absent (Knief et al. 2015a). In contrast, in
130 captivity, mating between related individuals is practically inevitable in the long run (Knief et al.
131 2015*a*). The level of inbreeding typically correlates negatively with individual fitness and various

132 morphological and life-history traits, even though the estimated effect sizes can vary widely
133 (Charlesworth and Charlesworth 1987; Keller and Waller 2002; Bolund et al. 2010a; Forstmeier
134 et al. 2012; Hemmings et al. 2012; Hoffman et al. 2014; Huisman et al. 2016; Michaelides et al.
135 2016). The importance of inbreeding in predicting reproductive failure remains largely unclear.

136

137 Ageing, or senescence, typically leads to a decline in reproductive function at old age, e.g. in
138 birds (Bouwhuis et al. 2009; Lecomte et al. 2010) and humans (Speroff 1994; Shirasuna and
139 Iwata 2017). In zebra finches breeding in cages, male and female fertility declined when
140 individuals became older (Knief et al. 2017). More generally, the relationship between age and
141 reproductive performance is often quadratic, with an initial increase in performance due to
142 gained experience that may mask any early-starting decline caused by deterioration of the body
143 (Harely 1990; Bouwhuis et al. 2009; Lecomte et al. 2010).

144

145 The conditions that an individual experienced during early development may also affect fitness
146 later in life. Such permanent environmental effects have been demonstrated using brood size
147 manipulations and they may affect individual behavior and reproductive investment (Gorman
148 and Nager 2004; Tschirren et al. 2009; Rickard et al. 2010; Boersma et al. 2014). In zebra finches,
149 being raised in enlarged broods apparently did not affect later performance (Tschirren et al.
150 2009). However, a non-experimental measure of individual early-growth condition, namely
151 body mass measured at 8 days of age (which ranges from 2-12 grams), had a significant but
152 small effect on fitness later in life (Bolund et al. 2010*b*).

153

154 For this study, we used systematically recorded data on individual body mass at 8 days of age
155 and on reproductive parameters and survival for four captive populations of zebra finches with
156 an error-free pedigree. The aims of this study were (1) to estimate and compare the relative
157 importance of inbreeding, early nutritional condition and age on reproductive performance and
158 lifespan, (2) to estimate the relative importance of individual and pair identity (i.e. repeatability)
159 on reproductive performance, (3) to quantify the heritability of individual reproductive
160 performance and (4) to test if some of the heritable components can be maintained by
161 antagonistic pleiotropy, by analyzing the additive genetic correlations between reproductive
162 performance traits and lifespan across the two sexes.

163

164 **Methods**

165 Zebra finches are opportunistic breeders that are abundant throughout most of Australia.
166 Individuals become sexually mature around the age of 90 days and then form pairs for life
167 through mutual mate choice. Breeding pairs cooperatively incubate and raise nestlings until
168 they reach independence around the age of 35 days (Zann 1996). Captive zebra finches live for
169 about 4.5 years on average and maximally for 10 years (Zann 1996, our unpublished data). The
170 studied zebra finches originated from four populations held at the Max Planck institute for
171 Ornithology, Seewiesen, Germany. The population background, rearing conditions and breeding
172 seasons have been detailed elsewhere (see also the online appendix, tables A1 and A2). In brief,
173 we compiled and analyzed up to 14 years of zebra finch reproductive performance data from

(1) population 'Seewiesen', a domesticated population derived from the University of Sheffield, with a nine-generation long error-free pedigree (population #18 in Forstmeier et al. (2007b));

(2) population 'Krakow', a domesticated population that was generated by hybridizing between Krakow (#11 in Forstmeier et al. (2007b)) and Seewiesen populations;

(3) population 'Bielefeld', which was derived from the wild in the late 1980s (#19 in Forstmeier et al. (2007b));

(4) population 'Melbourne', which was derived from the wild in the early 2000s (see Jerónimo et al. (2018)).

Birds from the two recently wild-derived populations were smaller (ca. 11g) compared to domesticated birds (ca. 15-16g), and more shy, so we only bred them in large semi-outdoor aviaries (rather than in small cages, see table 2 for sizes of cage and aviary).

Between 2004-2017, we bred zebra finches in four settings with various treatments (see tables A1 and A2 for details): (1) cage breeding, (2) cage laying, (3) aviary breeding, and (4) aviary laying. In cages, single pairs were kept and hence partners were assigned. In aviaries, groups of birds were kept together and individuals could freely form pairs. Group size was typically 12, but ranged from 10 to 42, with sex ratio (proportion of males) ranging from 0.4 to 0.6. In a 'breeding' setup, pairs were allowed to rear their offspring, whereas in a 'laying' setup all eggs were collected for paternity assignment and replaced by plastic eggs that were removed after 7 or 10 days of incubation. The proportion of individuals that participated in more than one breeding season ranged from 0.23-0.84 (mean 0.47).

194 In this study, we focus on general effects on reproductive performance in zebra finches, not on
195 population-specific effects. Therefore, in all analyses, we only controlled statistically for
196 between-population differences in reproductive performance (main effects only, no
197 interactions).

198

199 ***Measures of the focal fixed effects: inbreeding, age and early nutrition***

200 We used the pedigree-based inbreeding coefficient 'F_{ped}', calculated using the R package
201 'pedigree' V1.4 (Coster 2015), as a measure of the degree of inbreeding of an individual (Wright
202 1922; Knief et al. 2015*a*). F_{ped} reflects the proportion of an individual's genome that is expected
203 to be identical by descent (Howrigan et al. 2011; Knief et al. 2015*a*). For instance, full-sibling
204 mating produces inbred offspring that are expected to have 25% of the genome identical by
205 descent (F_{ped} = 0.25). For practical reasons, all founders were assumed to be unrelated (F_{ped} = 0;
206 Forstmeier et al. 2004). However, their true level of identity by descent is likely about 5%
207 (judging from runs of homozygosity; Knief et al. 2015a).

208

209 For all birds, we recorded their exact hatch date. Thus, for models of reproductive performance
210 at the level of eggs, clutches, and breeding rounds (as the unit of analysis), we used the exact
211 age (in days) of the female or the male when an egg was laid, a clutch started, or a breeding
212 round started, respectively. At the start of reproduction, individuals were 69-2909 days old.

213

214 On the day of hatching, we individually marked all nestlings on the back using water-proof
215 marker pens (randomly using red, blue and green, and pairwise combinations of these colors
216 if >3 nestlings). We checked survival almost daily (daily on weekdays, occasionally during
217 weekends) until offspring became independent (age 35 days). As a measure of early-growth
218 condition, we determined body mass of each nestling to the nearest 0.1 g at 8 days of age
219 (hereafter 'condition'). Despite the fact that high-quality food was available to all parents *ad*
220 *libitum*, nestling body mass at this age ranged from about 1.5 g to 12.6 g (mean = 7.1 ±1.7 SD).
221 For 297 out of 6190 nestlings, body mass was measured on day 6, 7 or 9. For those individuals,
222 we estimated their mass on day 8, as follows. We constructed a linear mixed-effect model, with
223 nestling body mass as the dependent variable, with the actual age of the mass measurement as
224 a continuous covariate and with F_{ped} and population (1-4, see above) as fixed effects. We also
225 included the identity of the genetic mother as a random effect. Using the slope of daily mass
226 gain, we estimated mass at day 8 for those 297 individuals by adding or subtracting 0.97 g per
227 day of measuring too early or late. Because the four populations differ in body mass, we
228 normalized (Z-scaled) all measured or estimated values of mass at day 8 within each population
229 before further analysis.

230

231 We report effects of inbreeding, age and early condition always with a negative sign, such that
232 negative values of greater magnitude reflect stronger detrimental effects of being inbred, old,
233 or poorly fed. This allows to meta-summarize the results and to directly compare the strength
234 of the focal fixed effects on reproductive performance.

235

236 ***Measures of lifespan and reproductive performance traits***

237 Table 2 provides an overview of all traits included in this study. To allow direct comparison and
238 easy interpretation of the fixed effects and additive genetic correlations, we scored all traits
239 such that higher, positive values reflect better reproductive performance.

240

241 Lifespan was analyzed in the following subset of birds: 5 generations of birds from the
242 Seewiesen population (referred to as generations P, F1-F3, and S3, N = 1855 individuals) and 4
243 generations of birds from the Bielefeld population (F1-F4, N = 1067 individuals). Among those
244 birds, we used the 4 most complete generations P and F1-F3 Seewiesen for which we recorded
245 the exact lifespan for all (N = 1175 individuals) as a pool to impute missing lifespans. For 219 S3
246 Seewiesen birds and for 663 Bielefeld birds, no date of natural death was available (e.g.
247 because individuals were still alive or because their fate was unknown). For these individuals,
248 we used imputed life expectancy in all analyses, defined as the average lifespan of individuals
249 from the same pool that lived longer than the focal bird when last observed alive.

250

251 In aviaries, we identified social pairs by behavior (clumping, allopreening, and visiting a nest
252 together). All parentage assignments were based on conventional microsatellite genotyping,
253 following Forstmeier et al. (2007a). We assigned every fertilized egg to its genetic mother (N =
254 11704 eggs). When the egg appeared infertile (no visible embryo; Birkhead et al. 2008), we
255 assigned it to the social female that was attending the clutch (N = 3630 cases). In 36 cases
256 where two females used the same nest to lay eggs, we assigned the unfertilized eggs to the

female that laid the most similar eggs (in size and shape), based on eggs that were certainly laid by a given female (e.g. fertilized eggs and eggs in other clutches laid by that female). In cases where birds were not allowed to rear offspring, we quantified female fecundity as the total number of eggs laid by the focal female during the breeding period (see table A1 and A2).

In breeding experiments, we opened all unhatched eggs to check for visible signs of embryo development and classified them as either infertile or 'embryo mortality'. In experiments in which all eggs were incubated artificially for a few days to collect DNA from embryos, we classified eggs as infertile or not, but discarded information on embryo viability. Visual inspection of opened eggs has the disadvantage that early embryo mortality may get misclassified as infertility if it occurred before any visible signs of development. Misclassification cannot be avoided entirely, even with more time-consuming examination of eggs, which would be challenging to do for thousands of eggs (Bellairs and Osmond 2005; Birkhead et al. 2008; Murray et al. 2013). However, most cases of apparent infertility coincided with the absence of sperm on the perivitelline layer of the egg (fig. A1, see also Birkhead and Fletcher (1998)). Thus, we expect only a small fraction of misclassification.

In cages, we measured male fertility as a binary trait, i.e. whether an egg was fertilized or not. In 12 cases, one to five eggs (median: 1 egg) were fertilized by the previous partner of the female and those were counted as infertile eggs of the focal male. In aviaries, we assessed fertility by whether an egg that was laid by a male's social partner was sired by him or not. Thus,

in aviary conditions, fertility also reflects a male's ability to defend his paternity against extra-pair males. We also quantified male siring success as the total number of fertilized eggs sired by a focal male. This includes males that remained unpaired (without a social female).

For each fertilized egg that was incubated by the social parents, we recorded whether it hatched or not (binomial trait for the genetic parents). For each hatched egg that was reared, we recorded whether the nestling survived to independence (day 35; binomial trait for the social parents). We quantified the number of seasonal recruits as the number of genetic offspring that survived to independence within a given breeding season. Number of seasonal recruits was square-root transformed to approach normality.

Statistical models

All mixed-effect models were run in R, using the R packages 'lme4' V 1.1-18-1 (Bates et al. 2018). All animal models were run using VCE6 (Neumaier and Groeneveld 1998), because (a) it allows running a 12-trait multivariate animal model that consists of 2346 individuals with at least one trait value per individual and (b) it has a reasonable running time. To check the consistency of model outputs, we repeated all animal models in the R packages 'pedigreemm' V 0.3-3 (Vazquez et al. 2010; univariate animal models only) and 'MCMCglmm' (Hadfield 2015; univariate and bivariate animal models). All model details are listed with the supporting data and R scripts at https://osf.io/tgsz8/. Model outputs of all methods are given in the online appendix. The heritability and additive genetic correlation estimates were highly correlated

299 between methods (r>0.65, P<0.002). We report the VCE6 estimates, unless otherwise stated.
300 Figure A2 shows the exact range of each focal fixed effect and each performance trait value.
301 Here, we Z-transformed all covariates and response variables across populations to allow direct
302 comparison of the effect sizes for inbreeding, age and condition across all models. The 95% CIs
303 of fixed effects from mixed-effect models were calculated using the function 'glht' from the R
304 package 'multcomp' V1.4-10 while controlling for multiple testing (Hothorn et al. 2008).

305

306 Data analysis involved four consecutive steps (fig. 1):

307

308 *Step 1: Estimation of fixed effects and variance decomposition*

309 The goal of Step 1 was to estimate (a) all fixed effects on reproductive performance and (b)
310 individual repeatability of performance traits. All fixed and random effects of models used in
311 Step 1 are listed in tables A3-A4. In brief, we first fitted all models with a Gaussian error
312 distribution to compare and meta-summarize the estimated effect sizes of the fixed effects and
313 to estimate the variance components for the random effects. We used all observations with
314 information on the three fixed effects (age, F_{ped}, and early condition of the male, female and
315 the individual egg if applicable), and included population (fixed effect) and female, male, and
316 pair identity (random effects). We analyzed traits that were measured at either egg, clutch, or
317 season level. As applicable, we fitted as fixed effects the laying sequence of eggs within a clutch,
318 the order of hatching of offspring within a brood, the order of the clutches that were laid by a
319 female over the course of a season, the sex ratio in the aviary, and the duration of the season

(table A1). For models of embryo survival, we also controlled for whether or not the eggs were incubated in a nest that still contained offspring from a previous brood (7% of embryos). For models of nestling survival, we added as fixed effect pair type (pair formed through mate choice or through force-pairing; Ihle et al. 2015). For models of egg-based fertility, embryo and nestling survival, we also tested the effect of egg volume on egg fate (we calculated volume as $V = (\frac{1}{6})\pi Width^2 Length$ where egg length and width had been measured to the nearest 0.1 mm). For this analysis, we fitted the mean egg volume of each female and the centered egg volumes (centered within individual females) to distinguish between the effects of between- and within-female variation in egg size (van de Pol and Wright 2009). We estimated the variance components for male, female, and pair identity, and further controlled for clutch identity and identity of the setup (see appendix tables A1-A2), as applicable, by adding them as random effects. Lifespan had no repeated measurement, therefore we only included individual identity as a dummy random effect for practical reasons when running the model and extracting estimates in R. For this 'lm' model, the correlation between the residuals and the dummy random effect equals one, and the fixed effect estimates were unaffected by the dummy variable. Table 2 shows for which group of individuals, i.e. female, male or the offspring itself, we tested which focal fixed and random effects.

To allow direct comparison of the magnitude of fixed effects at the same level of measurement, we also aggregated data within clutches (e.g. proportion of infertile eggs within a clutch) and within individuals over the course of a season. Models on aggregated data were weighted by the number of eggs within a clutch or by the number of eggs or clutches for an individual within

342 a season (fig. 1). As expected, the proportion of variance explained by male, female and pair
343 identity increased from the egg level to the season level (see Results). However, the relative
344 proportions explained by female, male, and pair identity did not change notably. Therefore, we
345 focus on the analyses of fixed effect estimates at the breeding season level.

346

347 To compare the overall effect sizes between the focal fixed effects, we meta-summarized the
348 estimated effect sizes for inbreeding, age and condition using the weighted 'lmer' function from
349 the R package 'lme4'. The uncertainty of each estimate was accounted for by using the
350 multiplicative inverse of the standard error ($\frac{1}{SE}$) of the response variable as 'weight'. In this
351 meta-model, we used effect size estimates from models that had been aggregated at the
352 season level as the dependent variable. Note that effects of inbreeding of the egg on fertility in
353 cage-breeding and nestling survival were taken from egg-based models, because they cannot
354 be aggregated by clutch or season. Additionally, we tested whether effect sizes differed
355 between males, females and offspring (fixed effect with three levels) or among traits (random
356 effect with 11 levels; as listed in table 2).

357

358 Additionally, we tested for early-starting ageing effects by selecting reproductive performance
359 data for males and females that were <2 years old when reproducing. We then meta-
360 summarized the mean age effect estimates using the R function 'lm', weighted by the
361 multiplicative inverse of the standard error.

362

We calculated the amount of variance explained by each fixed effect (Nakagawa and Schielzeth 2010) as the sum-of-squares of the fixed effect divided by the number of observations (N-1) (Henderson 1953). In weighted models, we divided the variance components of the fixed effects and the residual by the mean weight value (Bates et al. 2018).

Step 2: Estimation of heritability of fitness-related traits

The goal of Step 2 was to estimate the heritability of reproductive performance traits using univariate Gaussian animal models. Because quantitative genetic models require large amounts of data, we restrict our analyses to the populations Seewiesen and Bielefeld. Note that the pedigrees of our four captive populations are not connected, so it was not useful to analyze them jointly.

We kept the general model structure from Step 1, but excluded the fixed effects of egg volume on male fertility, embryo and offspring survival (to avoid removing biological variation that is potentially heritable and hence of interest; note that the effect sizes of egg volume are small, see Results). For the embryo survival model, we excluded the non-significant fixed effects of male age, inbreeding, and condition. For the model on male fertility from cage-breeding, we excluded the non-significant effect of the level of inbreeding of the egg itself. To most effectively use the available information on reproductive performance, we included individuals with missing values for condition (N = 231 founder individuals and N = 23 individuals of the F2 generation; i.e. 7% of Seewiesen birds). These missing values were replaced by the population

384 mean. Individual identity was fitted twice, once linked to the individual correlation matrix
385 (pedigree) to estimate the amount of variance from additive genetic effects (V_A) and once to
386 estimate the remaining amount of variance from permanent environmental effects (V_{PE}) (Kruuk
387 and Hadfield 2007). Animal models on nestling mortality were run twice, once for the mother
388 and once for the father. We calculated heritability based on the total phenotypic variance, V_{Ph},
389 as $h^2 = (V_A/V_{Ph})$, and we also quantified V_A relative to the individual repeatability as
390 $(V_A/(V_A+V_{PE}))$.

391

392 We compared the estimates of heritability (and V_A relative to the individual repeatability),
393 between the domesticated population 'Seewiesen' and the recently wild-derived population
394 'Bielefeld' using the R function 'lmer'. We used the multiplicative inverse of the standard error
395 as 'weight' to control for variation in uncertainty of each estimate. We used the estimates of
396 heritability as the response variable, and fitted population as fixed effect (two levels) and trait
397 as a random effect (9 levels, only including traits that were measured in both populations).

398

399 *Step 3: Calculation of mean individual fitness-related traits values using BLUPs*

400 The only goal of Step 3 was to extract individual estimates of reproductive performance needed
401 for Step 4. We kept the model structure from Step 1, except that we used a binomial error
402 structure for binary traits, i.e. male fertility in cages and aviaries, embryo and nestling survival.
403 Missing values for condition (mostly founders of each population, 6% of all birds of the four
404 populations) were replaced with population means as in Step 2. For the embryo survival model,

405 we again excluded the non-significant effects of male inbreeding, age, and condition. We also
406 excluded (a) effects of egg volume from all egg-based models and (b) the effect of the level of
407 inbreeding of the egg itself from the model of male fertility measured in cages (see Step 2).

408

409 We extracted the best linear unbiased predictions (BLUPs) for female or male identity (as
410 applicable) as the estimated life-history trait value of that individual (table 2) for Step 4.

411

412 *Step 4: Estimation of additive genetic correlations*

413 The goal of Step 4 was to estimate additive genetic correlations between different performance
414 traits using multivariate animal models.

415

416 Before fitting a 12-trait animal model that estimates for each matrix (genetic and residual) all
417 12 variances and 66 covariances simultaneously, we aggregated the raw data to one phenotypic
418 value per individual for each trait. This was necessary because we are not aware of software
419 that can handle the full complexity of the underlying raw data (involving more than 26 different
420 fixed effects). Because simple averages of multiple measures can result in outliers when sample
421 size is small, we used the phenotypic BLUPs described above. BLUPs do not produce outliers
422 and account for all considered fixed and random effects (Robinson 1991; Houslay and Wilson
423 2017). Breeding values (genetic BLUPs) suffer from non-independence, because the phenotype
424 of one individual influences the breeding values of all its relatives (Hadfield et al. 2010). Note
425 that this is not the case for the phenotypic BLUPs we use here. However, the uncertainty that is

426 inherent to each BLUP is not taken into account, which may lead to underestimation of
427 standard errors (Houslay and Wilson 2017). To check the robustness of our results, we
428 compared our estimates with those obtained (a) using a smaller dataset from another
429 population ('Bielefeld') with the same method and (b) using bivariate animal models in
430 'MCMCglmm' V 2.26 (Hadfield 2015) (population 'Seewiesen'). The latter approach is
431 presumably less powerful than a full 12-trait animal model.

432

433 For each of the 12 traits, we fitted an intercept, and the pedigree as the only random effect to
434 separate additive genetic from residual variance. We ran these models for the largest and most
435 comprehensive dataset (population Seewiesen; N = 2346 individuals with at least one trait
436 value, BLUPs for 12 traits, 66 covariances) and for the more limited dataset (population
437 Bielefeld; N = 1134 individuals, BLUPs for 9 traits, 36 covariances; see Results).

438

439 We used the weighted 'lm' function in the R package 'stats' to summarize the estimated
440 additive genetic correlations within and between the major categories of traits, i.e. female,
441 male, offspring traits, and lifespan for each population separately (table 2). We fitted the
442 estimates of additive genetic correlations (for each pair of traits, weighted by the multiplicative
443 inverse of the standard error of each estimate) as the dependent variable with trait-class
444 combination as a predictor with seven levels. We removed the intercept to estimate the mean
445 additive genetic correlation for each pairwise combination of classes. We then computed the
446 eigenvectors of the additive genetic variance-covariance matrix of traits, using the R function

447 'eigen', and visualized the orientation of the traits in the additive genetic variation space
448 defined by the principle components PC1 and PC2.

449

450 **Results**

451 ***Effects of laying and hatching order, clutch order and egg volume on egg and embryo fate***

452 The fate of an egg and its embryo depended on the order of laying within a clutch, the order of
453 hatching within a brood, and the order of consecutive clutches within a breeding season (fig. A3;
454 table A3, models at the 'Egg' level). First-laid eggs in a clutch were significantly more likely to be
455 infertile or to contain a dead embryo. Fertility and embryo viability were the highest for the 3rd
456 egg (fig. A3). Male fertility significantly increased over the first three clutches and stayed high
457 afterwards. In contrast, clutch order did not affect the probability of embryo and nestling
458 survival.

459 The average effect of egg volume on measures of egg fate was small (mean r = 0.040±0.016 SE,
460 fig. A4). Effects of egg volume were largest for nestling survival after hatching, and smallest for
461 embryo survival (table A3, fig. A4). Despite large sample size (N = 9,785 eggs), embryo survival
462 was not significantly influenced by egg volume (between-female variation: r = 0.015±0.017 SE, P
463 = 0.37; within-female variation: r = 0.018±0.010 SE, P = 0.08; table A3). Additionally, embryos in
464 clutches that were incubated in the presence of nestlings from previous breeding attempts
465 were more likely to die before hatching (b = 0.192±0.048 SE, P < 0.0001; table A3). Overall, the
466 total amount of variance explained by laying and hatching order, clutch order and egg volume
467 on egg fate was less than 5% (table A4).

468

469 ***Effects of inbreeding, age, and early condition***

470 Individuals performed worse in virtually all studied reproductive traits when they were more
471 inbred, as they became older and when they weighed less at 8 days of age (figs. 2, A2 and A5,
472 table A3). Interestingly, reproductive performance did not show an initial increase at young age
473 (meta-summarized effect size of age among birds younger than 2 years: r = -0.013 ± 0.011 SE,
474 figs. 2*C*, *F*, 3 and A2). Inbred eggs were equally likely to be infertile than outbred eggs, while
475 inbred embryos and offspring were more likely to die (fig. 3*C*). Together, this suggests that most
476 infertile eggs were not cases of undetected early embryo mortality. Individuals lived shorter
477 when they were inbred and when they had low weight at day 8 (fig. 3, table A3). However, the
478 fixed effects of inbreeding, age and condition together explained on average only 2% of the
479 variance across all traits (fig. 4 and table A5).

480

481 Meta-summarized effect sizes of inbreeding (r = -0.117 ± 0.024 SE) and age (r = -0.132± 0.032 SE)
482 were similar in magnitude, and were about twice as large as the remarkably small effect of
483 early condition (r = -0.058 ± 0.029 SE; fig. 3 and table A4). There was no significant difference
484 between the categories male, female, and offspring in how strongly they were affected by
485 these three factors ($b \leq 0.012 \pm 0.028$ SE, P = 0.63; table A4). Fitting trait (fitness component, 11
486 levels) as a random effect explained 1.5% of the variance in effect sizes (P = 0.02; table A4),
487 suggesting that some components might be less sensitive than others (fig. 3; table A3). Female
488 traits significantly predicted offspring survival and male fertility (independent of whether they

489 were measured in a cage or in an aviary), whereas male traits showed no effect on offspring
490 survival (fig. 3).

491

492 ***Variance components and heritability***

493 Variance components for all reproductive performance traits are shown in fig. 4 (see also table
494 A4). Overall, individual reproductive performance traits were significantly repeatable (median R
495 = 0.28, range: 0.15-0.50). Female reproductive performance traits (clutch size, fecundity, and
496 female seasonal recruits) showed reasonably high repeatability for individual females (R ~ 0.26-
497 0.40). Likewise, male fertility, male siring success, and male seasonal recruits were highly
498 repeatable for individual males (R ~ 0.24-0.50). Female reproductive traits from aviary-breeding
499 were analyzed independently of whether the focal female had a partner or not (table 2), but
500 female clutch size measured in a cage showed no contribution from the male partner or from
501 pair identity. In contrast, male fertility depended on all three random effects, and was
502 repeatable for males ($R > 0.23$, $P < 0.0001$), but less so for females ($R < 0.18$, $P < 0.1$), and for the
503 particular pair combinations ($R < 0.23$, $P < 0.05$). The model on embryo survival showed
504 significant female and pair identity (genetic parents) effects that were similar in size (both $R =$
505 0.20, $P < 0.0002$), while genetic male identity explained no variance (fig. 4). In contrast, social
506 female ($R = 0.17$, $P = 0.017$) and social male ($R = 0.15$, $P = 0.039$) identity explained significant
507 amounts of the variance in nestling survival, while the effect of pair identity (parents that raised
508 the brood) was less clear ($R = 0.14$, $P = 0.11$).

509

Reproductive performance traits and lifespan in general had low narrow-sense heritability (V_A/V_{Ph}; Seewiesen: median $h^2 = 0.07$; Bielefeld: median $h^2 = 0.11$) and explained only a limited amount of the individual repeatability ($V_A/(V_A+V_{PE})$; Seewiesen: median = 0.29; Bielefeld: median = 0.32; see all heritability estimates in tables A6-A7). Heritability estimates from the recently wild-derived population 'Bielefeld' were similar to those from the domesticated 'Seewiesen' population (for 9 traits measured in both populations; mean difference in $h^2 = 0.02$, range: -0.10 - 0.13, meta-summarized difference after controlling for the uncertainty of each estimate: $\Delta b < 0.0001$; mean difference in $V_A/(V_A+V_{PE}) = 0.20$, range: -0.13 - 0.68, meta-summarized difference: $\Delta b = 0.0002$; table A8).

Additive genetic correlations

Reproductive performance traits were grouped into three classes: (1) aspects of male reproductive performance, (2) aspects of female reproductive performance and (3) aspects of offspring survival (table 2). Traits within each of these classes were on average positively correlated with each other at the additive genetic level (for the Seewiesen population, female traits: mean $r_A = 0.66$, $P<0.0001$; male traits: mean $r_A = 0.67$, $P<0.0001$; offspring survival traits: mean $r_A = 0.36$, $P = 0.09$; see fig. 5A). Results for the Bielefeld population are in fig. A6. The meta-summarized results are given in table A9 and all additive genetic correlation estimates are listed in tables A10-A11. Estimates of the additive genetic correlations from bivariate animal models using MCMCglmm are in fig. A7 (Seewiesen) and A8 (Bielefeld).

Male and female reproductive performance traits were weakly negatively correlated at the additive genetic level (mean r_A = -0.14, P = 0.04; see 'MF' in figs. 5*A*, A7*A*). Accordingly, the eigenvectors for male and female fitness traits were pointing into different directions (figs. 5*B*, A7*B*). This pattern was somewhat consistent between the Seewiesen and Bielefeld populations (see figs. A6, A8 for Bielefeld population). However, the negative correlation between male and female fitness traits was no longer significant when estimated by the bivariate animal models in 'MCMCglmm', and disappeared in the 'Bielefeld' dataset (table A9). The orientation of offspring survival traits relative to male and female fitness traits was less consistent. In the Seewiesen population, female fitness traits were positively correlated with offspring survival traits at the additive genetic level (mean r_A = 0.61, P < 0.0001), while male fitness traits were not aligned with offspring survival traits (mean r_A = -0.11, P = 0.24; fig. 5). In contrast, in the Bielefeld population, both female and male fitness traits were positively correlated with offspring survival traits (fig. A6). Lifespan tended to be positively correlated with all reproductive performance traits (Seewiesen: mean r_A = 0.19, P = 0.02; Bielefeld: mean r_A = 0.60, P = 0.0006; figs. 5, A6; table A9).

Discussion

Effects of inbreeding, age, and early condition

Many studies have shown that inbreeding depression significantly influences morphological, behavioral, and fitness-related traits in zebra finches (Bolund et al. 2010*a*; Forstmeier et al. 2012; Hemmings et al. 2012; Opatová et al. 2016), and in other species (Amos et al. 2001; Reed

552 and Frankham 2003; Williams et al. 2003; Michaelides et al. 2016). This study confirms that
553 inbreeding negatively influenced all phases of offspring survival, reproductive performance and
554 lifespan. We found that the level of inbreeding of both genetic parents negatively influenced
555 egg fertility, suggesting that this is not only a matter of sperm functionality (Opatova et al.
556 2016), but also of female reproductive performance (e.g. egg quality or copulation behavior).
557 Male and female fitness estimates (seasonal recruits) were most strongly affected by
558 inbreeding (fig. 3), presumably because the successful rearing of offspring to independence
559 requires proper functionality at every step of reproduction.

560

561 Age effects on reproductive performance typically show an initial increase in performance both
562 in short- and in long-lived species (e.g. in great tits *Parus major* (Bouwhuis et al. 2009), Langur
563 monkeys *Presbytk entellus* (Harely 1990), red deer *Cervus elaphus* (Pemberton et al. 2009),
564 albatrosses *Diomedea exulans* (Lecomte et al. 2010), and bustards *Chlamydotis undulata*
565 (Preston et al. 2011)). Interestingly, in our captive zebra finches we found that reproductive
566 performance (especially male fertility, female clutch size, fecundity, and female effects on
567 embryo survival) did not show an initial increase after birds reached sexual maturity at about
568 100 days of age (figs. 2*C, F*, A2). This could be because zebra finches are short-lived
569 opportunistic breeders that reach sexual maturity earlier compared to most other birds (Zann
570 1996). Thus, zebra finches might have been selected to perform best early on. Alternatively,
571 this effect may not be present in the wild, where experience might play a more important role
572 in determining reproductive success.

573

574 Over the past decades, numerous studies focused on how early developmental conditions
575 affect behavior, life history, and reproductive performance later in life (Tschirren et al. 2009;
576 Rickard et al. 2010; Boersma et al. 2014). Here, we show that even dramatic differences in early
577 growth conditions of surviving offspring (see range of x-axis in fig. 2*B*), have remarkably small
578 (though statistically significant) effects on adult reproductive performance.

579

580 Overall, the proportion of variance explained by inbreeding, age, and early condition
581 (characteristics of conditions) was less than 3% (fig. 4; table A4). This indicates that individuals'
582 robustness against poor conditions appears more noteworthy than their sensitivity. As will be
583 discussed in the following paragraphs, the majority of the individual repeatability in
584 reproductive performance cannot be explained by such individual characteristics.

585

586 ***Repeatability and heritability of reproductive performance***

587 Individual zebra finches were remarkably repeatable in their reproductive performance. Our
588 variance-partitioning analysis showed that infertility is largely a male-specific trait, whereas
589 embryo and offspring survival are mostly related to female identity (fig. 4; table A4). The effects
590 of pair identity on infertility and offspring mortality may reflect behavioral incompatibility,
591 while the pair effect on embryo mortality more likely reflects genetic incompatibility (Ihle et al.
592 2015).

593 Although male and female zebra finches are highly repeatable in their reproductive
594 performance, the heritability of fitness traits was low and similar between the recently wild-

595 derived Bielefeld population and the domesticated Seewiesen population. Overall, our findings
596 indicate that there are some additive genetic components underlying zebra finch reproductive
597 performance.

598

599 ***Evidence for sexually antagonistic pleiotropy***

600 Some of the standing additive genetic variance in reproductive performance could be
601 maintained by intra-locus sexual antagonism between male fitness traits and female (and
602 offspring) fitness traits. This has for example also been suggested in studies on Drosophila
603 (Innocenti and Morrow 2010) and on red deer (Foerster et al. 2007). We found that male
604 fertility, siring success and seasonal recruitment were overall negatively correlated with female
605 fitness and offspring survival traits, suggesting that alleles that increase male fitness tend to
606 reduce female and offspring fitness (fig. 5). In contrast, lifespan and reproductive performance
607 tended to be positively correlated at the additive genetic level, which is suggestive of some
608 overall 'good gene variation' in our population (fig. 5). Some words of caution should be added
609 to these observations. VCE6 (figs. 5, A6) yielded higher absolute values of estimates than those
610 calculated with the R functions 'PedigreeMM' (heritability estimates only) and 'MCMCglmm'
611 (see appendix figs. A7-A8; also see tables A6-A7, A10-A11). Nevertheless, the additive genetic
612 correlation estimates are highly correlated between the two methods ($r > 0.7$, $P < 0.0001$; see
613 tables A10-A11). Estimating genetic correlations between traits with low heritability requires
614 large datasets, especially on additive genetic correlations of between-sex reproductive
615 performance where the traits of male fertility and female clutch size in cages are missing (N
616 performance traits: Seewiesen = 12, Bielefeld = 9; N birds have at least one entry of

617 reproductive performance data: Seewiesen = 2346, Bielefeld = 1134; hence these results are
618 presented in the online appendix only, fig. A6). Despite this lack of power in our second largest
619 data set of population Bielefeld, its overall orientation of traits in the additive genetic variation
620 space of the principle components PC1 and PC2 is very similar to population Seewiesen (note
621 that lifespan is in the center of all fitness traits and that aspects of female fitness do not align
622 with male fitness in figs. 5*B*, A6*B*, A7*B*, A8*B*).

623

624 Individual repeatability of fitness-related traits could arise from permanent environmental
625 effects (e.g. early developmental conditions and long-lasting diseases) or from genetic effects.
626 However, while food-shortage experienced during early development (reflected in body mass
627 at 8 days old) strongly predicted nestling mortality (our unpublished data), it only explained <1%
628 of variation in reproductive performance (mean r = -0.058, figs. 2*B*, *E*, 3 and also 4). Additionally,
629 our captive zebra finches were raised and kept in a controlled environment with no obvious
630 diseases detected. Additive genetic effects explained only about 30% of the large remaining
631 unexplained individual repeatability in fitness-related traits, suggesting that reproductive
632 performance might be (predominantly) dependent on genetic effects of local over- or under-
633 dominance and epistasis, i.e. incompatibility between loci. For instance, high levels of
634 reproductive failure could be maintained when alleles show non-additive effects, with selection
635 favouring the heterozygous genotype (see e.g. Sims et al. 1984; Grossen et al. 2012). In the
636 zebra finch, males that are heterozygous for an inversion on the Z chromosome produced fast-
637 swimming sperm and sired more offspring (Kim et al. 2017; Knief et al. 2017). Epistatic effects
638 that involve several genes (e.g. incompatibility between nuclear loci, or between mitochondrial

639 and nuclear genomes) could be evolutionary stable when certain combinations of genotypes
640 perform better than others, especially when combined with overdominance (Avent and Reid
641 2000; Arntzen et al. 2009; Hermansen et al. 2014; Knegt et al. 2016; Baris et al. 2017; Stryjewski
642 and Sorenson 2017).

643

644 Infertility, as one of the main and puzzling sources of reproductive failure, behaved as a male-
645 specific trait that may in part also depend on behavioral compatibility between pair members
646 (reflected in copulation behavior) and in part on the male's genotype at sexually antagonistic
647 loci. The intrinsic male fertility, measured in a cage, i.e. in the absence of sperm competition,
648 correlated negatively with all female and offspring survival traits at the additive genetic level
649 ('sexual antagonism'; median r_A = -0.30, range: -0.45 to -0.01; fig. 5; table A10). In contrast, in
650 the presence of sperm competition (aviary breeding), high male fertility, siring success and
651 seasonal recruitment should also be influenced by the competitive ability of the individual and
652 this could explain why these traits correlated positively with lifespan and trade-off less with
653 female traits and offspring rearing ability at the additive genetic level (figs. 4, A6; tables A10-
654 A11).

655

656 Embryo mortality, another main source of reproductive wastage, mostly depended on the
657 identity of the genetic mother and the identity of the genetic pair members. A previous study
658 using cross-fostering of freshly laid eggs also showed that embryo mortality is a matter of the
659 genetic parents rather than the foster environment (Ihle et al. 2015). The female component

suggested an overall female genetic quality effect, yet with limited heritability (pointing towards dominance variance or epistasis). The effect of the combination of parents on embryo mortality might reflect an effect of the genotype of the embryo itself, possibly involving multi-locus incompatibilities.

Conclusions

Our results suggest that sexually antagonistic pleiotropy between male and female fitness plus offspring rearing traits may maintain some of the existing additive genetic variation in reproductive performance traits in captive zebra finches. Additionally, there appears to be some 'good gene' (heritable) variation among reproductive performance traits and individual lifespan, which suggests an ongoing adaptation to the captive environment. We found that the level of inbreeding, age and – to a lesser extent – early rearing conditions predicted a small, but statistically significant amount of variation in individual reproductive performance and lifespan. However, those three effects were so small that they cannot be the main causes of reproductive failure. Although individual zebra finches were moderately repeatable in their reproductive performance, the heritability of those traits was low. Overall, our results suggest that alleles that have additive effects on fitness might be maintained through sexually antagonistic pleiotropy, and the major genetic causes of reproductive failure might be determined by genetic incompatibilities or local dominance effects.

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858

859 **Table 1:** Summary of rates of hatching failure, infertility, and embryo and offspring mortality reported in the zebra finch literature

Population	Sample description	Hatching failure (%)	Infertility (%)	Embryo mortality (%)	Nestling mortality (%)	Reference
Wild	1,156 eggs, clutches that produced no nestlings were removed	>17	-	-	-	Zann 1996
Wild	872 eggs, clutches that produced no nestlings were removed	16	-	-	9	Griffith et al. 2008
La Trobe University, Australia, domesticated	31 untreated and 25 CORT treated pairs, clutches that produced no nestlings and all first eggs were removed.	Untreated: 24; treated: 45	Untreated: 7; treated: 15	Untreated: 10; treated:29	-	Khan et al. 2016
Max Planck Institute for Ornithology, Germany, domesticated (from Sheffield, UK)	11,617 eggs	-	30	-	-	Knief et al. 2015b

Max Planck Institute for Ornithology, Germany recently wild-derived (from Bielefeld, Germany)	852 eggs, aviary	-	17	24	45	Ihle et al. 2015
Sheffield University, UK	161 eggs for infertility; 2,884 eggs for hatching failure and nestling mortality	52	35	-	31	Kim et al. 2017
Sheffield University, UK	1,524 eggs; 77 unrelated and 20 sib-sib pairs	-	Unrelated: 9; sib-sib: 11	Unrelated: 59; sib-sib: 75	Unrelated: 55; sib-sib: 68	Hemmings et al. 2012

Note: For population of La Trobe University, Australia, in treated pairs, females were given a corticosterone (CORT) mix after laying the first egg. The CORT mix was made of 0.5 mg crystalline corticosterone dissolved by 10 µl ethanol then mixed with 990 µl peanut oil (Khan et al. 2016). Hatching failure: proportion of eggs that do not hatch. Infertility: proportion of eggs that show no sign of development. Embryo mortality: proportion of fertilized eggs where the embryo died before hatching. Nestling mortality: proportion of nestlings that died before fledging.

860

861 **Table 2:** Description of reproductive performance traits in our zebra finch study

Trait	Fixed effects for	Random effects	BLUPs calculated for	Description
			Female	
Clutch size cage				
	Female	Female	Female	The number of eggs that were consecutively laid by a single female in a cage (contains one male and one female), allowing for laying gaps of maximally 4 days between subsequent eggs. For 2% (65 out of 3694) clutches that had >7 eggs, they were counted as 7.
	-	Male	-	
	-	Pair	-	
Clutch size aviary				
	Female	Female	Female	The number of eggs that were consecutively laid by a female in a communal breeding aviary, allowing for laying gaps of maximally 4 days between subsequent eggs. For 5% (173 out of 3663) clutches that had >7 eggs, they were counted as 7.
Fecundity aviary				
	Female	Female	Female	The total number of eggs laid by a female in a communal breeding aviary over the course of a breeding season (35-83 days), where no offspring rearing was allowed.
Seasonal recruits				
	Female	Female	Female	The total number of genetic offspring that survived to independence in a communal breeding aviary, i.e. age 35 days, within a breeding season (83-113 days for egg laying plus about 50 days for rearing).

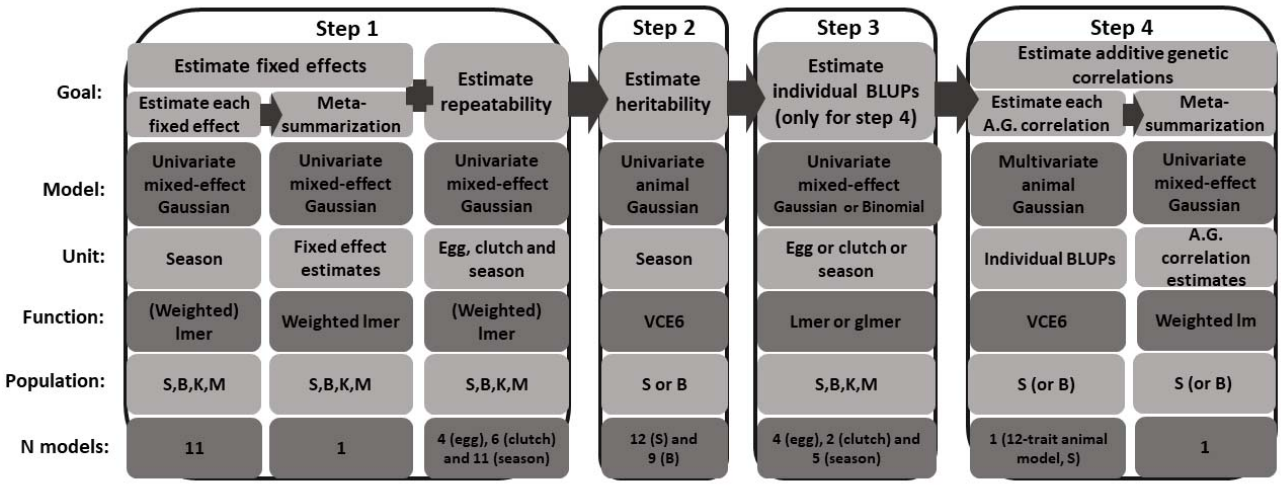
	Male		
Fertility cage			
Female	Female	-	Whether or not an egg was fertilized by the male in the cage (that contains one male and one female).
Male	Male	Male	
-	Pair	-	
Egg	-	-	
Fertility aviary			
Female	Female	-	Whether or not an egg laid by the social partner of the male in a communal breeding aviary was fertilized
Male	Male	Male	by the male (extra-pair fertilizations count as not fertilized).
-	Pair	-	
Siring success			
Male	Male	Male	The total number of eggs fertilized by a male in a communal breeding aviary over the course of a breeding season (35-113 days).
Seasonal recruits			
Male	Male	Male	The total number of genetic offspring that survived to independence in a communal breeding aviary, i.e. age 35 days, within a breeding season (83-113 days for egg laying plus about 50 days for rearing).
	Offspring		
Embryo survival			
Female	Female	Female	Whether or not a fertilized egg that was incubated by an individual in a cage (contains one male and one

	Male	Male		female) or a communal breeding aviary hatched.
	-	Pair	-	
	Embryo	-	-	
Nestling survival				
	Female	Female	Female	Whether or not a nestling that hatched in a cage (contains one male and one female) or a communal
	Male	Male	Male	breeding aviary survived to independence, i.e. age 35 days.
	-	Pair	-	
	Nestling	-	-	
			Individual	
Lifespan				
	Individual	-	Individual	The number of days from the date of hatching to the date of natural death.
				Some missing values were replaced by life-expectancy.

Note: Traits were measured either in the context of single pairs breeding in a small cage or of multiple pairs breeding communally in a large aviary. Fixed effects (focal) are inbreeding coefficient, age, and early condition (mass at day 8). Random effects (focal) are the variance components explained by female, male or pair identity. 'BLUPs' stands for best linear unbiased predictions, estimated from univariate models where we controlled for significant fixed and random effects. For the offspring trait of embryo survival, female, male and pair identities refer to the genetic parents of the embryo, whereas for nestling survival, female, male and pair identities refer to the social parents that raised the nestling. Cage dimensions, before 2012: 60x40x45 cm (L x W x H), afterwards: 120x40x45 cm. For details of housing conditions see Bolund et al. (2007). A semi-outdoor aviary measured 500x200x200 cm (L x W x H).

862

863 **Figure 1.** Steps of data analysis from univariate mixed models to multivariate animal models.
864 Shown are the goals of the analysis, the model properties, the unit of analysis (i.e. whether
865 rows in the data represent single eggs, clutches, individuals in a breeding season, single fixed
866 effect estimates, or individuals overall), the software functions used for analysis (for models on
867 aggregated levels, 'weight' stands for the number of eggs or clutches used for each aggregation,
868 whereas in meta-summarization models, weight stands for the multiplicative inverse of the
869 standard error of each estimate) and the population abbreviations for data used for the analysis.
870 'S', 'B', 'K', and 'M' stand for 'Seewiesen', 'Bielefeld', 'Krakow', and 'Melbourne', respectively.
871 Number of models conducted within each step with their specific details (e.g. unit, population
872 or the model type) used for analysis. 'A.G.' stands for 'additive genetic'.



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874

875 **Figure 2**. Reproductive performance traits (continuous or count traits in A-C, binomial traits in
876 D-F) as a function of inbreeding coefficient (F_{ped}; A, D), early condition (mass at day 8,
877 separately for populations that differ in body size (B, E), and age (C, F). Clutch size, fecundity,
878 siring success, seasonal recruits, and lifespan are continuous or count traits (Z-scaled), whereas
879 the proportions of eggs fertilized, embryos survived, and nestlings survived are binomial traits.
880 Note that these are composite figures of all effects that were examined (see fig. A2 for plots of
881 single traits with absolute trait values), such that the fate of one embryo may be shown twice,
882 once as a function of the embryo's own F_{ped} and once as a function of its mother's F_{ped} (hence
883 the high sample sizes, N_{obs}). The age category zero contains measurements until day 365. Red
884 lines show smoothed regressions with 95% CIs, circle size (A-C) and bar width (D-F) reflect
885 sample sizes.

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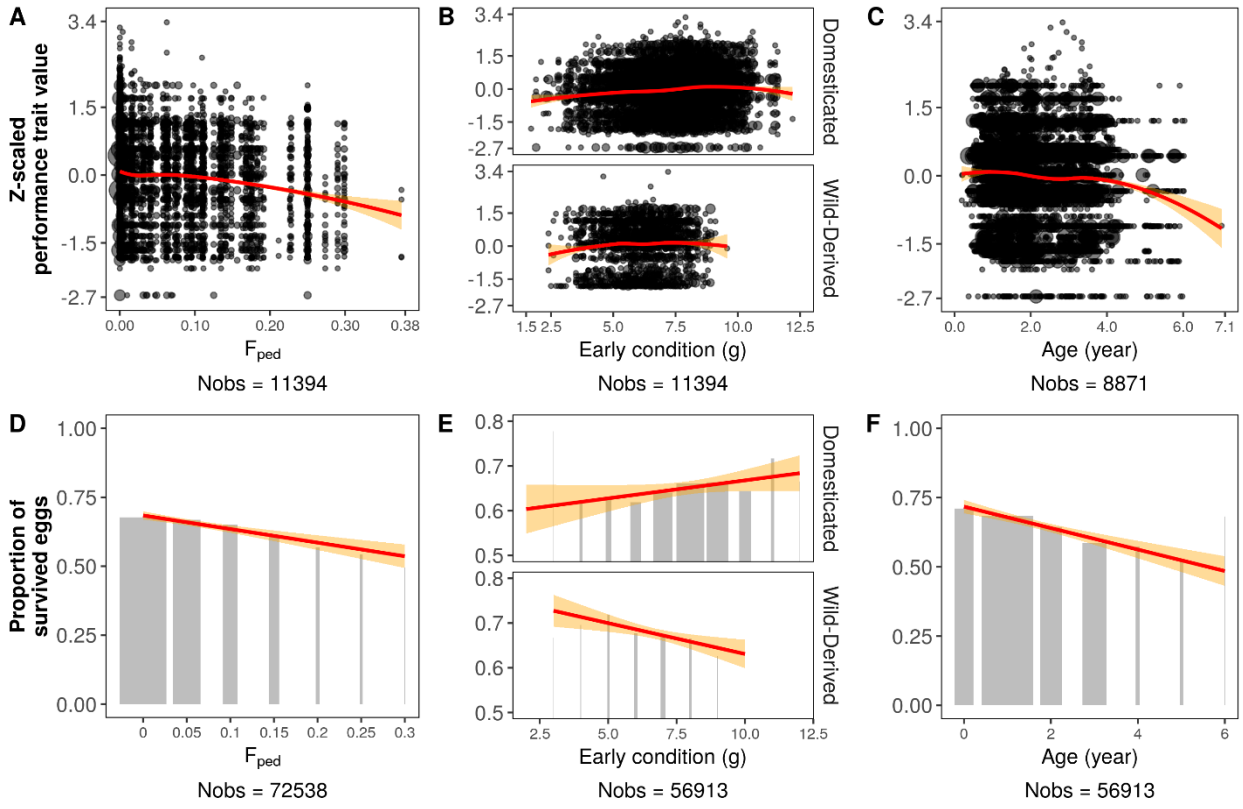
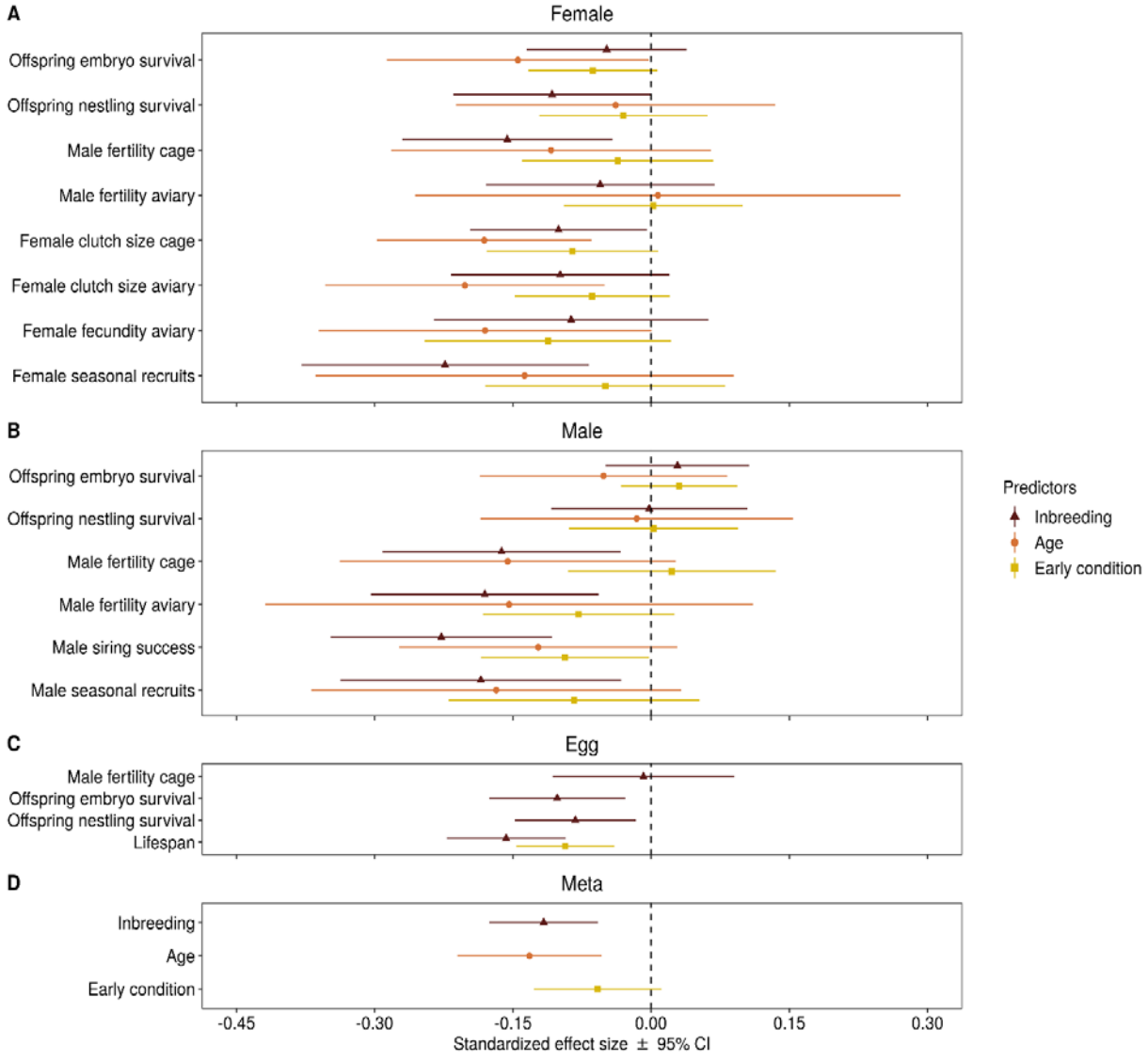


887

888 **Figure 3.** Standardized effect sizes with their 95% confidence intervals for inbreeding (F_{ped}), age
889 and early condition (mass at day 8) on zebra finch fitness components estimated in univariate
890 Gaussian mixed-effect models where all response variables were measured at the level of
891 individuals within seasons, and all measurements were Z-scaled (table A3). Note that the effect
892 of inbreeding of the offspring on its own mortality was taken from egg-based models. Negative
893 effects of condition indicate low fitness of relatively light-weight individuals at 8 days of age.
894 Panels separate effects of condition, age and inbreeding of the female (A), the male (B), and the
895 individual egg itself (C). Panel (D) shows the meta-summarized effect sizes for reproductive
896 performance and lifespan (table A4). The X-axes indicate effect sizes in the form of Pearson
897 correlation coefficients.



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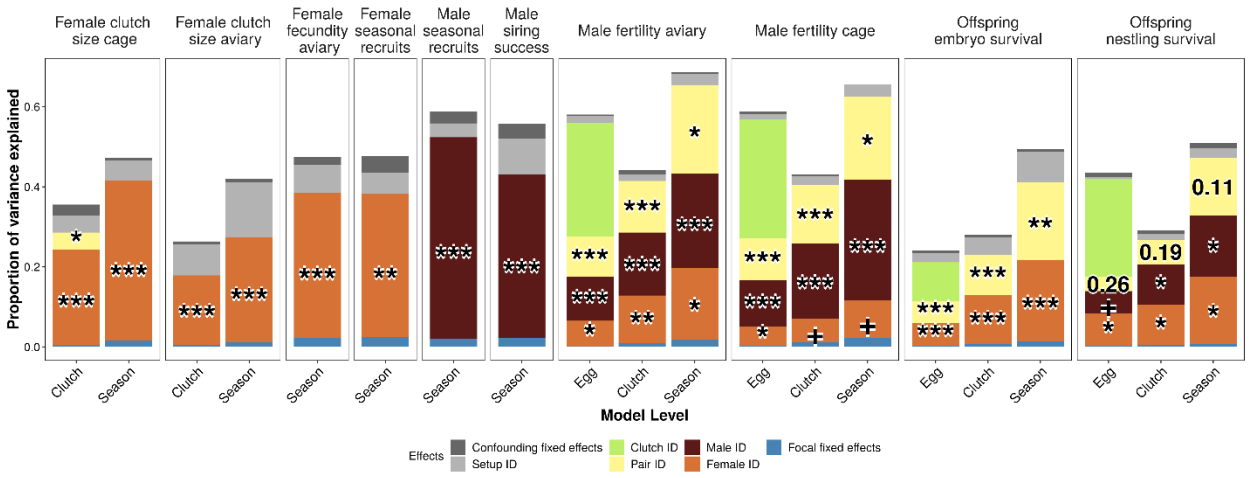
899

900 **Figure 4.** Variance components estimated in univariate Gaussian mixed-effect models (table A5).
901 Each dependent trait is shown in a separate panel. Within panels, the x-axis separates models
902 according to the unit of analysis, based on either egg fate (Egg), values per clutch (Clutch), or
903 values per individual within a breeding season (Season). The y-axis indicates the proportion of
904 variance explained by random effects after accounting for fixed effects. 'Focal fixed effects'
905 refers to the total variance explained by inbreeding, age, and early condition combined. For the
906 key variance components, numbers show non-significant P-values, otherwise '+' indicates P <
907 0.1, '*' indicates P < 0.05, '**' indicates P < 0.001, and '***' indicates P < 0.0001. Note that
908 Models of 'female clutch size aviary', 'female fecundity aviary' and 'female seasonal recruits'
909 were analyzed without 'Male ID' and 'Pair ID', and likewise 'male seasonal recruits' and 'male
910 siring success' was analyzed without 'Female ID' and 'Pair ID' because not all birds form a pair
911 bond; 'Male ID' explained no variance in models of 'clutch size cage' and 'embryo survival'
912 while 'Pair ID' explained no variance in 'clutch size cage' model.



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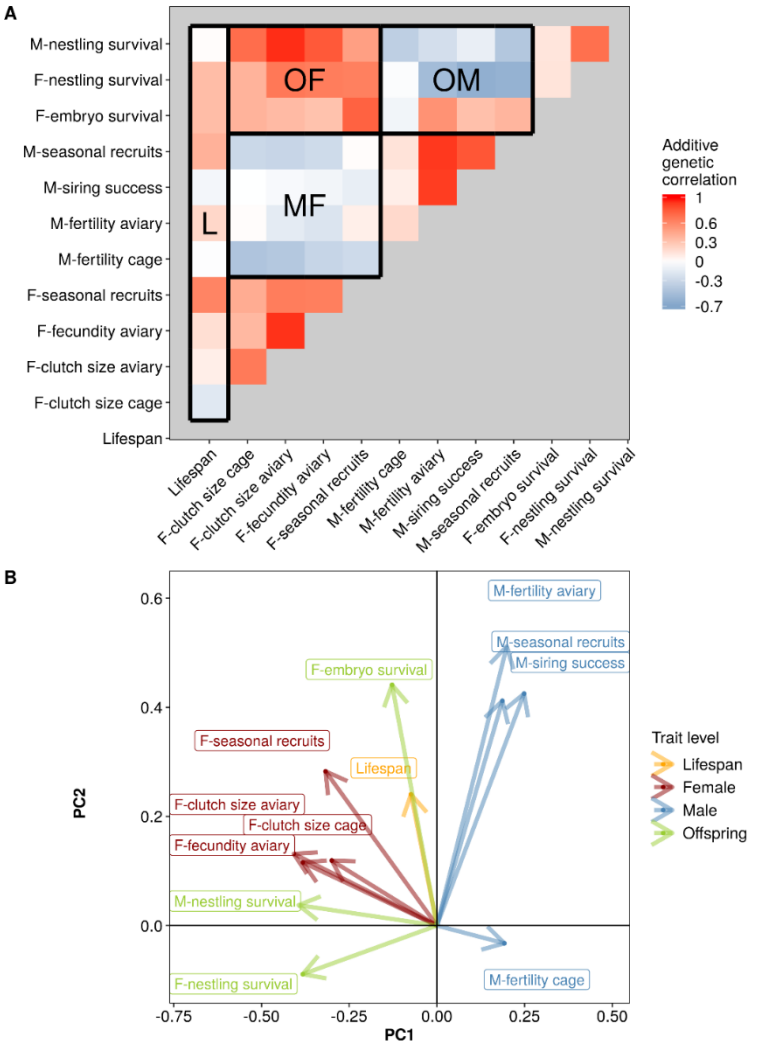
915 **Figure 5.** G-matrix of reproductive performance traits and lifespan estimated from multivariate
916 animal models for the Seewiesen population (shown are estimates from VCE; for estimates of
917 MCMCglmm bivariate models see fig. A7; see also figs A6 and A8 for estimates from the
918 Bielefeld population; estimates are given in tables A10-A11). (A) Heatmap of additive genetic
919 correlations between components of male (M), female (F), and offspring (O) fitness, and life
920 span (L). Red indicates a positive genetic correlation between traits while blue indicates a
921 negative correlation. Blocks marked in bold emphasize correlations between categories (e.g.
922 MF stands for correlations between male and female fitness components). (B) The first two
923 principal components of the G-matrix, showing eigenvectors of the 12 fitness components.
924 Note that aspects of male fitness do not align with aspects of female and offspring fitness.



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