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The quantitative genetics of fitness in a wild seabird

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17 **ABSTRACT**

18 Additive genetic variance in fitness is a prerequisite for adaptive evolution, as a trait must be
19 genetically correlated with fitness to evolve. Despite its relevance, additive genetic variance in
20 fitness has not often been estimated in nature. Here, we investigate additive genetic variance in
21 lifetime and annual fitness components in common terns (*Sterna hirundo*). Using 28 years of data
22 comprising ca. 6000 pedigreed individuals, we find that additive genetic variances in the Zero-
23 inflated and Poisson components of lifetime fitness were effectively zero, but estimated with high
24 uncertainty. Similarly, additive genetic variances in adult annual reproductive success and survival
25 did not differ from zero, but were again associated with high uncertainty. Simulations suggested
26 that we would be able to detect additive genetic variances as low as 0.05 for the Zero-inflated
27 component of fitness, but not for the Poisson component, for which adequate statistical power
28 would require c. two more decades (four tern generations) of data collection. As such, our study
29 suggests heritable variance in common tern fitness to be rather low if not zero, shows how studying
30 the quantitative genetics of fitness in natural populations remains challenging, and highlights the
31 importance of maintaining long-term individual-based studies of natural populations.

32

33 **Keywords:** adaptive potential, additive genetic variance, heritability, lifetime reproductive
34 success, log-normal fitness

35 **INTRODUCTION**

36 Fisher's Fundamental Theorem of Natural Selection postulates that "*the rate of increase in fitness*
37 *of any organism at any time is equal to its genetic variance in fitness at that time*" (Fisher 1930).
38 As such, additive genetic variance in fitness, being equivalent to the change in mean fitness
39 resulting from selection, has been considered the single most useful statistic quantifying selection
40 (Burt 1995). Genetic variation in fitness also is a prerequisite for adaptive evolution, as a trait must
41 be genetically correlated with fitness to evolve through natural selection (Robertson 1966; Price
42 1970). Hence, understanding the quantitative genetics of individual variation in fitness is arguably
43 one of the most important aims in evolutionary ecology (Burt 1995; Ellegren and Sheldon 2008;
44 Walsh and Blows 2009; Gomulkiewicz and Shaw 2013; Shaw and Shaw 2014; Hendry et al. 2018).

45 Considerable debate has surrounded the question of whether or not additive genetic variation
46 in fitness is expected be low (e.g., Jones 1987; Burt 1995; Houle et al. (1996), Merilä and Sheldon
47 1999; Shaw and Shaw 2014), and particularly, under which conditions (e.g., Cheverud and
48 Routman 1995; Whitlock et al. 1995). Empirical estimates of additive genetic variance in fitness
49 from wild populations are relatively scarce (e.g., Gustafsson 1986; Kruuk et al. 2000; Merilä and
50 Sheldon 2000; McCleery et al. 2004; Coltman et al. 2005; Brommer et al. 2007; Foerster et al.
51 2007; Teplitsky et al. 2009; Wheelwright et al. 2014; McFarlane et al. 2014, 2015; Wolak et al.
52 2018; de Villemereuil et al. 2019), and have so far not shed much light on this debate, since
53 estimates vary substantially, with many estimates close to zero, and few large estimates (review
54 by Hendry et al. 2018). Overall, Hendry and colleagues (2018) tentatively concluded that the
55 evolvability of fitness (measured as the square of the coefficient of additive genetic variance in
56 fitness) is usually less than 0.2.

57 Data constraints might partially explain the paucity of studies testing for the heritability of
58 fitness in the wild and the heterogeneity among estimates of additive genetic variance, although
59 steadily growing datasets collected from long-term study populations gradually alleviate the
60 problem (Clutton-Brock and Sheldon 2010). This increased data availability was recently
61 accompanied by the development of (i) statistical tools designed to deal with the non-Gaussian
62 distributions that often characterize fitness data (de Villemereuil et al. 2016; de Villemereuil 2018),
63 as well as (ii) theoretical frameworks that facilitate the evolutionary inference of quantitative
64 genetic parameters based on these data distributions (Morrissey and Bonnet 2019). To date, only
65 four studies have modelled the quantitative genetics of fitness in wild populations assuming a non-
66 Gaussian distribution (McFarlane et al. 2014, 2015; Wolak et al. 2018; de Villemereuil et al. 2019).
67 Additive genetic variance in fitness was estimated to be very small in North American red squirrels
68 (*Tamiasciurus hudsonicus*) ($V_A \sim 0$, 95% = 5.2×10^{-07} - 1.1, McFarlane et al. 2014, see also
69 McFarlane et al. 2015). In birds, de Villemereuil et al. (2019) showed that hihis (*Notiomystis*
70 *cincta*) in New Zealand had negligible additive genetic variance in lifetime fitness (V_A Zero-Inflated
71 component ~ 0 , 95% CI = 1.4×10^{-11} - 0.0038 and V_A Poisson component = 0.0078, 95% CI = 2.3×10^{-10} -
72 5.7), while Wolak et al. (2018) found that the song sparrows (*Melospiza melodia*) of Mandarte
73 island in Canada harbored substantial additive genetic variance in female and male fitness (V_A
74 female = 2.01, 95% CI = 0.21 - 3.93; V_A male = 1.72, 95% CI = 0.27 - 3.39).

75 Here, we present phenotypic and pedigree data obtained from a 28-year individual-based study
76 on common terns (*Sterna hirundo*). The common tern is a Nearctic and Palearctic colonially
77 breeding, serially monogamous and migratory seabird. The study colony is located in the north of
78 Germany; common terns from this colony spend their winters in western Africa and return to the
79 breeding colony in early spring to breed or prospect potential breeding locations (Becker and

80 Ludwigs 2004). Common terns breed annually, both parents incubate and feed the chicks, and
81 extra-pair paternity is rare (González-Solís et al. 2001; Becker and Ludwigs 2004). Applying a
82 series of “animal models” to data from almost 6000 pedigreed individuals across five generations,
83 we investigate additive genetic variance for lifetime fitness (assessed as the total number of
84 fledglings produced by a locally-born fledgling), and two of its underlying annual components:
85 annual reproductive success and adult annual survival.

86

87 **METHODS**

88 **Study System**

89 Fitness and pedigree data were collected between 1992 and 2019 as part of a long-term study of a
90 common tern population located at the Banter See on the German North Sea coast (53°36'N,
91 08°06'E). The Banter See colony consists of six concrete islands, each of which is surrounded by
92 a 60-cm wall. Walls are equipped with 44 elevated platforms, each containing an antenna which
93 reads transponder codes. The individual-based study at the Banter See was initiated in 1992, when
94 101 adult birds were caught and marked with individually-numbered subcutaneously-injected
95 transponders. Since 1992, all locally hatched birds are similarly marked with a transponder shortly
96 before fledging and the presence and reproductive performance of marked individuals is monitored
97 following a standard protocol (Becker and Wendeln 1997). As part of this protocol, the colony is
98 checked for new clutches every 2–3 days throughout the breeding season (Zhang et al. 2015).
99 Parents are identified using portable antennae placed around each nest for 1–2 days during
100 incubation, which is shared by both partners. Pairs can rear up to three chicks per brood (mean
101 successful brood size 0.41 ± 0.65 SD chicks), and can produce replacement clutches after loss of
102 eggs or chicks. Second clutches are extremely rare (Becker and Zhang 2011).

103

104 **Fitness Data**

105 Our initial data selection included individuals that fledged between 1992 and 2016, because
106 previous work showed that 97% of fledglings, if they returned, did so within the first 3 years
107 (Vedder and Bouwhuis 2018). Since there is little standardized monitoring in areas around the
108 focal colony, we cannot directly quantify juvenile dispersal. However, we do know that there is (i)
109 a relatively high local return rate (26% of chicks fledged between 1992 and 2016 returned to the
110 colony, of which 14% recruited), and (ii) only rare reporting of external recruits (between 1992
111 and 2016, 32 fledglings from the Banter See were observed a total of 105 times in other European
112 breeding colonies). In addition, although we cannot directly observe an individual's death, we can
113 reliably assume it, because adult breeders at the Banter See are highly site-faithful, evidenced by
114 a resighting probability of breeding individuals close to one (Szostek and Becker 2012), and 96%
115 of breeders not skipping recording by the antenna system for two or more consecutive years after
116 first reproduction (Bouwhuis et al. 2015; Zhang et al. 2015). Based on this knowledge, we removed
117 all birds that were observed in 2018 and/or 2019 *and* were younger than 11 years old, because (i)
118 they are known to not be, or cannot yet be assumed to be, dead, and (ii) lifetime fitness of
119 individuals older than 10 years and those dead showed a high correlation ($r > 0.8$) in our dataset.
120 Hence, we included birds that have completed their life histories ($n = 5836$), as well as birds that
121 were still alive but older than 10 years ($n = 163$) to avoid introducing a cohort truncation bias by
122 non-randomly removing longer-lived birds (Hadfield 2008; Morrissey et al. 2012). To control for
123 any potential confounding effect, we modelled whether an individual was considered dead or alive
124 as a fixed effect (see below).

125 We quantified lifetime fitness as the number of local fledglings that a locally-hatched
126 fledgling produced during its lifetime, for a total of 5999 locally-hatched fledglings (Fig. 1A) and
127 decomposed it into two major components: juvenile survival and adult lifetime reproductive
128 success. Juvenile survival captures survival from fledgling to age 1, inferred from whether a
129 fledgling became a local recruit in later years, whereas adult lifetime reproductive success captures
130 adult survival and reproductive success from age 1 onwards. These two fitness components
131 correspond to the two mechanisms captured by the Zero-inflated Poisson distribution of lifetime
132 fitness. We further decomposed adult lifetime reproductive success into its two components:
133 annual reproductive success (ARS) and adult annual survival (AAS). ARS was measured as the
134 number of fledglings that an individual produced each year between age 1 and last registration,
135 assigning zeroes for years of skipped reproduction or registration, and for years prior to recruitment
136 (Fig. 1B). Similarly, AAS was adult survival (1/0) to the following breeding season, measured
137 every year from age 1 to last registration (inferring missing direct observations prior to recruitment
138 from later observations). In total, our data comprised 836 individuals with 6873 observations for
139 ARS and AAS.

140

141 **Pedigree**

142 The pedigree was constructed by assigning all fledged offspring to their social parents, then pruned
143 to remove individuals who were either not phenotyped or not ancestors to phenotyped individuals.
144 For the purpose of this study, the pruned pedigree comprised 6290 records. The maximum depth
145 was five generations, the number of paternities and maternities 2417 and 2520, respectively. The
146 numbers of full, paternal and maternal sibships were 2594, 10229 and 9807, respectively (see
147 Supplementary Material for further information on the population relatedness structure). This

148 social pedigree is a good approximation of the genetic pedigree, because common terns exhibit
149 very low levels of extra-pair paternity (González-Solís et al. 2001).

150

151 **Quantitative Genetic Models**

152 We applied an animal model approach that combines the phenotypic information on individual
153 fitness components with information from the social pedigree (Kruuk 2004). As such, we fitted a
154 series of univariate animal models where fitness, or one of its components, was the response
155 variable.

156 To model lifetime fitness, we fitted a univariate animal model with a Zero-Inflated Poisson
157 error distribution. We fitted a Zero-Inflated Poisson distribution to better capture the nature of our
158 metric of lifetime fitness. Zero-inflation is often the result of a process that determines whether an
159 event occurs or not, which differs from the Poisson process that determines how many times an
160 event occurs. In this case, a Zero-Inflated Poisson model can explicitly model the two different
161 processes, as opposed to a Poisson model that assumes only a single process to be generating the
162 data (Korner-Nievergelt et al. 2015). We fitted random intercepts for individual identity linked to
163 the pairwise relatedness matrix and for hatch-year (to account for cohort effects; e.g., Vedder and
164 Bouwhuis 2018). Because we modeled lifetime fitness with a Zero-Inflated over-dispersed Poisson
165 distribution, we could estimate the covariance between the Zero-inflated and Poisson components
166 for each variance component. However, a model including additive genetic and hatch-year
167 covariances between the Zero-Inflated and Poisson components of the trait did not provide a better
168 fit to the data, hence we do not model such covariances. The main models presented also did not
169 control for shared environmental effects between siblings (maternal, paternal, or brood effects),
170 because we did not have information on parental identity for all individuals (maternal identities =

171 2382 and paternal identities = 2481; 1271 individuals have both maternal and paternal identities
172 known, see Supplementary Material for detailed information on the population relatedness
173 structure), and because most fledglings came from broods where only a single individual had
174 successfully fledged (3027 broods fledged one chick, 1145 broods two, 226 broods 3, while 4
175 individuals could not be assigned to a brood). However, we did explore the potential effects of a
176 shared environment (due to maternal, paternal effects, or brood effects) by running two additional
177 animal models which included one or two shared environmental effects as random effect(s). We
178 found that there was no major influence on our estimate of additive genetic variance in lifetime
179 fitness components, as expected given that the model presented in the main text returned a very
180 low (close to or zero) estimate of additive genetic variance (see Suppl. Material, Tables S1 and
181 S2).

182 As fixed effects, we modelled the trait intercept and whether the individual was alive or
183 dead at the end of the study period (categorical variable with two levels). Additionally, we
184 performed data simulations to investigate (i) whether we can effectively detect *small, but*
185 *substantial* additive genetic variances in fitness (*sensu* de Villemereuil et al. 2019) given our data
186 and pedigree structure, and (ii) the improvement of our statistical power to detect small additive
187 genetic variances in both components of lifetime fitness when the dataset and pedigree would
188 increase in size and depth (Supplementary Material, Figs. S1-S5).

189 To model ARS, we assumed a Poisson error distribution with a log link function and
190 checked whether the trait was underdispersed, which was not the case. We fitted random intercepts
191 for individual identity linked to the pairwise relatedness matrix, individual identity not linked to
192 the pedigree (to account for permanent environmental effects) and year of observation (to account
193 for temporal variation across years). As fixed effects, we modelled the trait intercept and age

194 (continuous trait ranging from 1 to 23 years), as fledgling production is known to linearly increase
195 with age (Zhang et al. 2015) (but see Supplementary Materials, Table S3, for results of the same
196 animal model without age effects).

197 To model AAS, we assumed a binary error distribution with a logit link function and fixed
198 the residual variance to one. We fitted random intercepts for individual identity linked to the
199 pairwise relatedness matrix, individual identity not linked to the pedigree (to account for
200 permanent environmental effects) and year of observation (to account for temporal variation across
201 years). As fixed effects, we modelled the trait intercept and age (continuous trait ranging from 1
202 to 23 years), as AAS is known to linearly decrease with age (Zhang et al. 2015; Vedder et al. 2021)
203 (but see Supplementary Materials, Table S3, for results of the same animal model without age
204 effects).

205 All quantitative genetic models were fitted using a Bayesian framework implemented in
206 the statistical software R (v. 3.6.1, R Core Team 2019) using the R-packages *MCMCglmm*
207 (Hadfield 2010) and *QGglmm* (de Villemereuil et al. 2016). Posterior distributions were plotted
208 using the R-package *wolakR* (github.com/matthewwolak/wolakR). Narrow-sense heritabilities (h^2)
209 were conditional on the variance explained by fixed effects and were estimated as the proportion
210 of the total phenotypic variance explained by the additive genetic variance. Evolvabilities (I_A) were
211 estimated by dividing the additive genetic variance by the squared population mean (Houle 1992;
212 Hansen et al. 2011).

213 For all models we used parameter-expanded priors (Hadfield 2010). We fitted different
214 priors for each fitness component (see Supplementary Material). The number of iterations and
215 thinning intervals were chosen for each model so as to ensure that the minimum MCMC effective
216 sample size for all parameters was 1000. Burn-in was set to a minimum of 5000 iterations. The

217 retained effective sample sizes yielded absolute autocorrelation values <0.1 and satisfied
218 convergence criteria based on the Heidelberger and Welch convergence diagnostic (Heidelberger
219 and Welch 1981). We drew inferences from the posterior mode and 95% credible intervals (95%
220 CI). To facilitate evolutionary inference (Bonnet et al. 2019; Morrissey and Bonnet 2019), we
221 back-transformed the latent-scale posterior distributions of the quantitative genetic parameters to
222 the data-scale (de Villemereuil et al. 2016).

223

224 **RESULTS**

225 **Quantitative Genetics of Lifetime Fitness Components**

226 Among the 5999 common tern chicks that fledged between 1992 and 2016, lifetime fitness ranged
227 between 0 and 29 fledglings (Fig. 1A). 5231 (87.19%) fledglings obtained zero fitness, such that
228 the distribution of fitness was strongly zero inflated (Fig. 1A).

229 Raw mean fitness was 0.72 ± 2.52 SD fledglings. Although this would indicate the
230 population to be in overall decline (a mean lifetime breeding success of two fledglings would be
231 required for the population to be stable), population size actually varied dramatically across years
232 and did not decline (Fig. S6), partially because there was a substantial influx of non-locally hatched
233 breeders that immigrated into the population (ca. $74\% \pm 1$ of breeders was estimated to be
234 immigrant in any given year between 1992 and 2020). Since we do not capture or mark immigrants,
235 we can quantify the proportion of immigrants present in our colony in a given year but we cannot
236 include them in the pedigree or our individual-based models.

237 Simulations showed that, given our data structure and pedigree, we would not be able to
238 detect what might be considered a *small, but substantial* signal for the Zero-inflated component of
239 lifetime fitness: we generated a Zero-inflated component of fitness with an additive genetic
240 variance of 0.01, and found that the average posterior mode was similar to the simulated value of

241 V_A (average = 0.012 across the 100 replicates, Fig. S1), but the lower 95% CI limit was on average
242 zero across replicates (95% CI = 0 – 0.023 and lower 95% CI exceeded a value of 0.0001 only 72
243 times across the 100 replicates, Fig. S1). When we simulated larger values of additive genetic
244 variance (i.e., $V_A = 0.05$ or 0.1), our simulations showed that we would be able to detect those
245 (average = 0.053 and 95% CI = 0.028 – 0.083 across the 100 replicates for a simulated value of
246 0.05; and average = 0.102 and 95% CI = 0.064 – 0.145 for a simulated value of 0.1). Lower 95%
247 CI always exceeded a value of 0.0001 in both simulated cases (Figs. S3 and S4).

248 Our quantitative genetic analysis of empirical data suggested that the additive genetic
249 variance in the Zero-Inflated component of lifetime fitness was not different from zero, as the
250 posterior mode of the additive genetic variance was very close to, and the lower 95% CI limit
251 leaning towards, zero (Table 1, Fig. 2A-C). Taken together, our combination of analyses of
252 empirical and simulated data therefore suggested there to be low (lower than 0.05) to null additive
253 genetic variance in the Zero-inflated component of lifetime fitness, but that we lack power to
254 determine with higher precision whether such variance is effectively zero, or non-zero but very
255 small.

256 The results for the Poisson component of lifetime fitness are less straightforward.
257 Simulations showed that, given our data structure and pedigree, we would not be able to detect
258 either *small, but substantial* or larger signals for the Poisson component of fitness: we generated a
259 Poisson-component of fitness with a series of evolvability values ($I_A = 0.00, 0.01, 0.05$ and 0.1),
260 and found that the lower 95% CI limit was on average zero in all cases (i.e., lower 95% CI did not
261 exceed a value of 0.0001 in the vast majority of the 100 replicates, Fig. S1-4). Our analysis of the
262 empirical data suggested that the additive genetic variance of the Poisson component did not differ
263 from zero, given that the associated lower 95% CI limits of V_A , h^2 and I_A converged towards zero

264 (Table 1, Fig. 2D-F). Altogether, the combination of empirical analyses and data simulations
265 showed that we lacked power to determine where the additive genetic variance in the Poisson
266 component of lifetime fitness falls within a rather large range of values (between “larger than 0.1”
267 and zero).

268 Finally, simulation of a larger dataset with a deeper pedigree structure indicated that
269 increasing our study to include four more generations of pedigreed individuals would lead to an
270 important increase in statistical power, such that we would be able to detect additive genetic
271 variances of at least 0.05 in both components of lifetime fitness. Estimated values of additive
272 genetic variance were of similar magnitude to that of the simulated value (average posterior mode
273 of 0.05 across the 100 replicates for both components of lifetime fitness), with non-zero lower
274 95% CI in both cases (95% CI = 0.031- 0.064 for Zero-Inflated component, and 95% CI = 0.009 -
275 0.197 for Poisson component, Fig. S5).

276

277 **Quantitative Genetics of Annual Fitness Components**

278 We investigated the Annual Reproductive Success and Adult Annual Survival of 836 fledglings
279 that survived to adulthood and bred in our population (Table 2). Raw mean annual reproductive
280 success was 0.70 ± 0.81 SD with a maximum of three fledglings (Fig. 1B). The posterior
281 distribution of V_A for ARS converged toward zero (Table 2, Fig. 4A-C), suggesting that V_A is not
282 different from zero. Raw mean adult annual survival probability was 0.85 ± 0.36 SD. The posterior
283 modes of all quantitative genetic parameters for AAS were very close to zero (Table 2, Fig. 3A-
284 C), with the lower 95% CI limit of all parameter estimates converging towards zero, again
285 suggesting that V_A in AAS is not different from zero.

286

287 **DISCUSSION**

288 The most direct measure of the adaptive potential of a population is its standing additive genetic
289 variance in fitness (Fisher 1930). Here, we estimated additive genetic variances in lifetime and
290 annual fitness components in a wild colony of common terns. On the one hand, our empirical
291 findings indicated no evidence for substantial (or different from zero) additive genetic variance in
292 lifetime fitness components, adult annual survival or annual reproductive success. On the other
293 hand, data simulations demonstrated an overall lack of statistical power to detect *small, but*
294 *substantial* signals (i.e., $V_A = 0.01$), although statistical power differed between the two
295 components of lifetime fitness: we would have power to detect slightly larger signals (additive
296 genetic variances of, at least, 0.05) for the Zero-inflated, but not Poisson, component of fitness. As
297 such, our work demonstrated that estimating additive genetic variance in fitness is very difficult in
298 wild populations, partly due to the expected low values of genetic variation in fitness in locally
299 adapted populations, but also to the challenges associated with collecting sufficient phenotypic
300 and pedigreed data.

301

302 **Quantitative Genetics of Lifetime and Annual Fitness Components**

303 There have been around 30 studies testing for additive genetic variance in fitness in the wild, with,
304 to our knowledge, only four using non-Gaussian animal models (McFarlane et al. 2014, 2015;
305 Wolak et al. 2018; de Villemereuil et al. 2019). Our estimate of the additive genetic variance for
306 the Zero-inflated component of lifetime fitness on the data-scale was effectively zero, with a zero
307 lower 95% CI limit (posterior mode $V_{A \text{ data-scale}} = 0.004$, 95% CI = 0 - 0.008, Table 1), similarly to
308 results for another bird species, the hihi (posterior mode $V_{A \text{ data-scale}} \sim 0$, 95% CI = 1.4×10^{-11} -
309 0.0038, de Villemereuil et al. 2019). For the Poisson component, de Villemereuil et al. (2019)

310 found a posterior mode of 0.0078 (95% CI = 2.3×10^{-10} - 5.7). Our posterior mode estimate was
311 overall larger (posterior mode $V_{A \text{ data-scale}} = 2.29$, Table 1), but associated with high uncertainty
312 (95% CI = 0.002 - 12.3), such that the estimates from both studies remain qualitatively similar.
313 Given that our estimates of additive genetic variance in fitness showed very low or null values,
314 our study implies that the adaptive potential of this natural population of common terns will be
315 extremely limited, although the actual potential remains partially unknown as our estimates were
316 associated with high uncertainty. Moreover, it is important to note that we could only investigate
317 the evolutionary potential of local recruits, as we did not have phenotypic and pedigree data to
318 investigate the evolutionary potential of the total colony (i.e., local recruits and immigrants).

319 Additive genetic variance in lifetime fitness can theoretically be decomposed into the
320 additive genetic variances in its underlying components. The two primary components of our
321 measure of lifetime fitness are juvenile survival and adult lifetime reproductive success. Our zero-
322 inflation in lifetime fitness is mainly due to low juvenile survival (i.e., 74% of fledglings did not
323 locally return to the colony), while the Poisson process generating the observed fitness distribution
324 is mostly capturing adult lifetime reproductive success. If we compare our nominally zero additive
325 genetic variance in the Zero-inflated component of lifetime fitness (Table 1) with estimates from
326 other studies that tested for additive genetic variance in juvenile survival, we observe some
327 differences. For instance, the study of Wolak et al. (2018) on the song sparrow population of
328 Mandarte Island reported evidence for non-zero V_A for juvenile survival.

329 Adult lifetime reproductive success is the sum of annual reproductive events across the life
330 of an individual, and hence, can be decomposed into annual reproductive success and adult annual
331 survival. Our quantitative genetic analyses of these two annual fitness components revealed a lack
332 of substantial additive genetic variance for both (Table 2). This finding again contrasts with one

333 from Mandarte’s song sparrows, where there was evidence for moderate levels of additive genetic
334 variance in ARS (especially for males) and close to zero in AAS, indicating that heritable ARS
335 was the primary component of heritable adult lifetime reproductive success in that population
336 (Wolak et al. 2018).

337

338 **Limitations of studying quantitative genetics of fitness in the wild**

339 Estimating quantitative genetic parameters with precision is a data-hungry endeavor. Researchers
340 therefore are faced with the challenge of collecting hard-to-quantify lifetime fitness data from an
341 unbiased sample of the population (i.e., avoiding the “missing fraction” bias) that comprises a
342 sufficiently large number of individuals of known relatedness (Burt 1995; Merilä and Sheldon
343 1999; Hendry et al. 2018). In addition, even when a large pedigree is available, additive genetic
344 variance in fitness is often expected to be low, for instance, when populations are locally adapted,
345 such that the power to detect small, close to zero, additive genetic variation in fitness may be low
346 as well. As pointed out by Burt (1995): “it is very difficult to get an estimate that is statistically
347 distinguishable from zero, and the sample sizes required to do so might easily lead to despair”.
348 Our data simulations reveal that we would need at least four more generations of terns to
349 statistically differentiate between an underpowered and a true zero estimate of additive genetic
350 variance for the Poisson component of lifetime fitness. Increasing our pedigree by four more
351 generations would require roughly two more decades of data collection, i.e. a non-negligible
352 amount of funding and logistic effort. This extrapolation should, however, be taken with care, as
353 it is challenging to predict the population dynamics for the next twenty years, and/or whether the
354 relatedness structure of the population will increase or decrease as the rates of emigration and
355 immigration may change with population growth (e.g., Szostek and Becker 2014). In light of the

356 multiple constraints posed by data requirements and expected low values, negative results with
357 respect to additive genetic variation in fitness should be discussed with caution. Nevertheless,
358 simulations aimed at determining the statistical power of a given dataset and pedigree structure
359 will help to distinguish a true negative result from a zero parameter estimated with high uncertainty
360 (e.g., de Villemereuil et al. 2019).

361 In addition to the difficulty of estimating the heritability of fitness with precision, our
362 knowledge of the genetic architecture of fitness components is limited. Extending our genomic
363 understanding of fitness variation in wild populations will bring important insights into how
364 genetic variation underpinning fitness may be maintained, and overall will help to better predict
365 the evolutionary dynamics of natural populations (Merilä and Sheldon 1999; Mackay 2001; Huang
366 and Mackay 2016). Despite the clear benefits, genomic research based on quantitative trait loci
367 (QTL) approaches or genome-wide associations in natural populations was a challenge (Slate
368 2004; Slate et al. 2010; Jensen et al. 2014), partially due to the low power to detect QTL, for
369 instance because studies suffered from low-density linkage maps and/or relatively few genotyped
370 individuals. Nowadays, the use of powerful next-generation genomic techniques, however, allows
371 to increase the power in such studies.

372 A better understanding of the genetic architecture of fitness will also provide added
373 benefits, as, for instance, it would allow a deeper understanding of the genetic underpinnings of
374 complex traits such as fitness, which might be subjected to different pleiotropic effects (Mackay
375 2001). For instance, antagonistic pleiotropy is often assumed to underlie the negative phenotypic
376 correlation between the two main components of lifetime fitness: survival and reproductive
377 success (also observed in the terns: Vedder et al., 2021).

378

379 **Conclusion**

380 Our quantitative genetic study of fitness in a wild population of common terns reported low to zero
381 estimates of additive genetic variance in lifetime and annual fitness components, which were at
382 the same time associated with high uncertainty. Those analyses, however, were overshadowed by
383 a lack of statistical power to detect additive genetic variation in fitness more accurately and
384 precisely. The continuation of long-term individual-based studies should be safeguarded (also see
385 Clutton-Brock and Sheldon 2010), such that the maturation of long-term studies will offer
386 improved opportunities for testing genetic variation in natural populations, which, thanks to the
387 recent development of appropriate statistical and theoretical frameworks (de Villemereuil et al.
388 2016; Bonnet et al. 2019; Morrissey and Bonnet 2019), will help to improve our understanding of
389 the genetics of fitness in the wild. Ultimately, a robust quantification of the standing additive
390 genetic variation in fitness will inform us about the rate of adaptation of populations, and allow a
391 better understanding of their viability in the face of the deleterious environmental effects resulting
392 from current climate and global changes.

393

394 **AUTHOR CONTRIBUTIONS**

395 M.M. conceived the study with input from S.B. and A.C. M.M. designed and conducted the
396 analyses, and wrote the manuscript. S.B. manages the tern data and collated the dataset. All authors
397 contributed to editing the final paper.

398

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408

409 **DATA ARCHIVING**

410 Data have been archived in the Dryad Digital Repository: <https://doi.org/10.5061/dryad.8kpr4xqj>
411 (Moiron et al. 2022).

412

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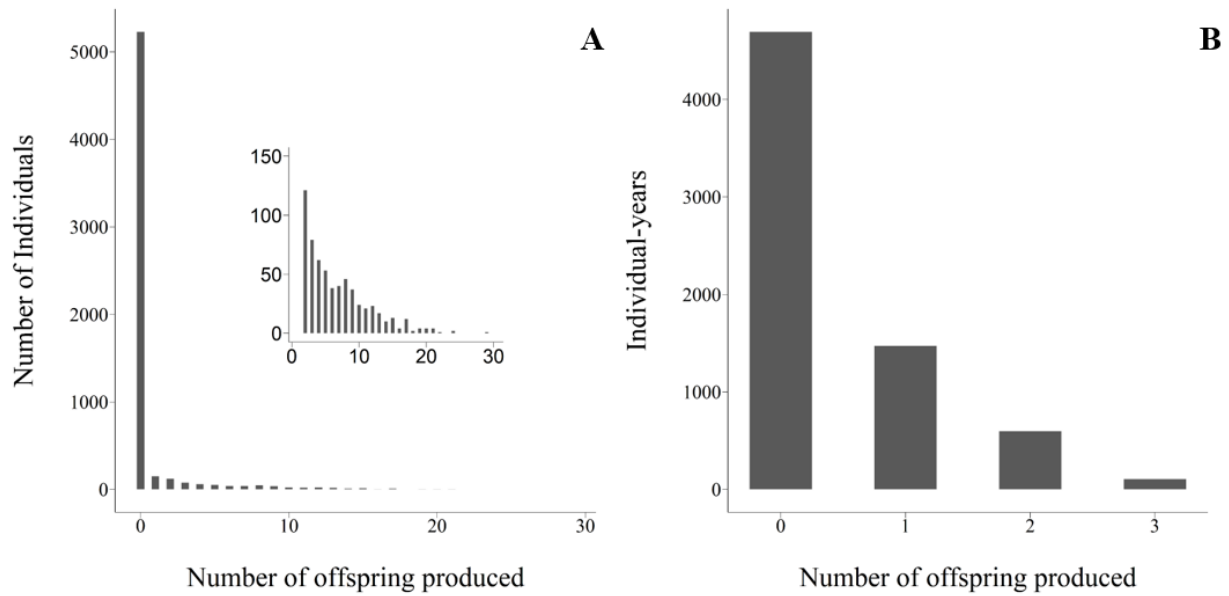
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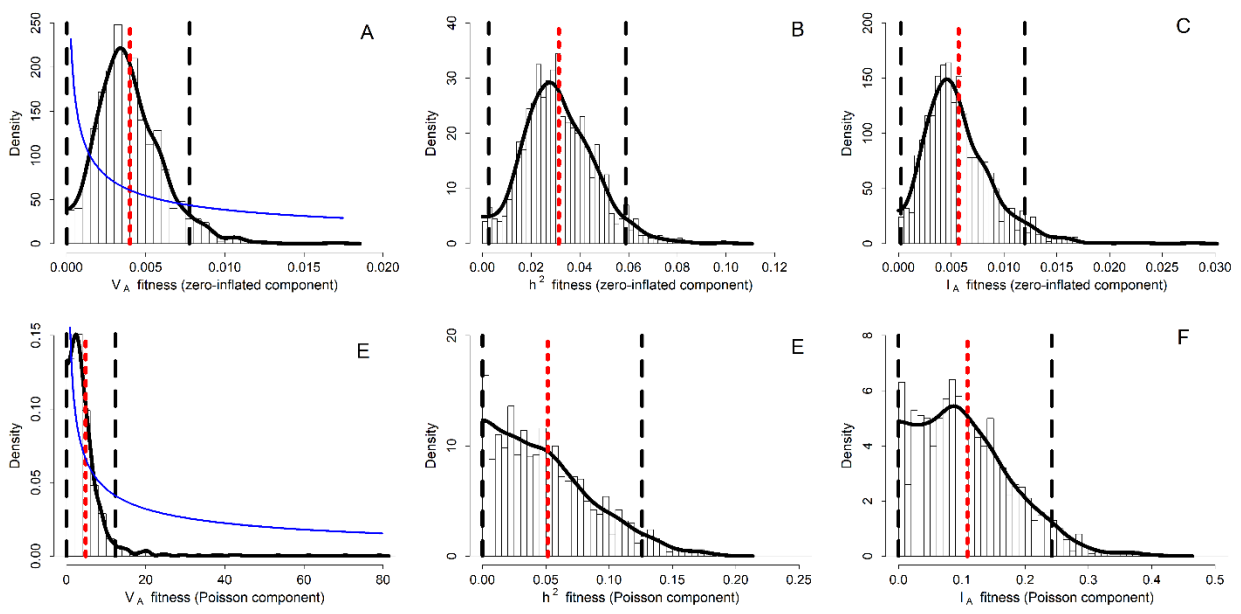
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540 **FIGURE LEGENDS**

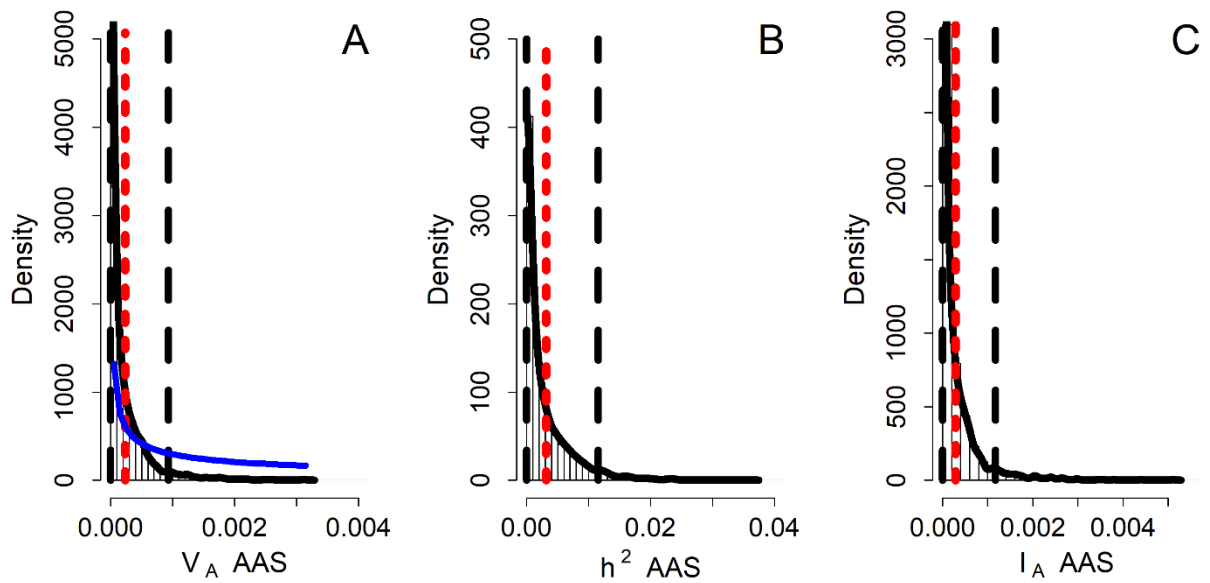
541 **Figure 1.** Phenotypic distributions of A) lifetime fitness measured as the total number of fledglings
542 a locally-hatched fledgling produced in its lifetime (with the inset showing the distribution for non-
543 zero fitness in more detail), and B) annual reproductive success, measured as the number of
544 fledglings an adult breeder produced in a year.



545 **Figure 2.** Posterior MCMC samples (bars), kernel density estimation (solid black line), posterior
546 mean (red dotted line), 95% Credible Intervals (black dashed lines) and prior (solid blue line) for
547 the A) additive genetic variance (V_A), B) heritability (h^2) and C) evolvability (I_A) of the Zero-
548 Inflated component of lifetime fitness, and the D) additive genetic variance (V_A), E) heritability
549 (h^2) and F) evolvability (I_A) of the Poisson component of lifetime fitness. Distributions are reported
550 on the data scale.

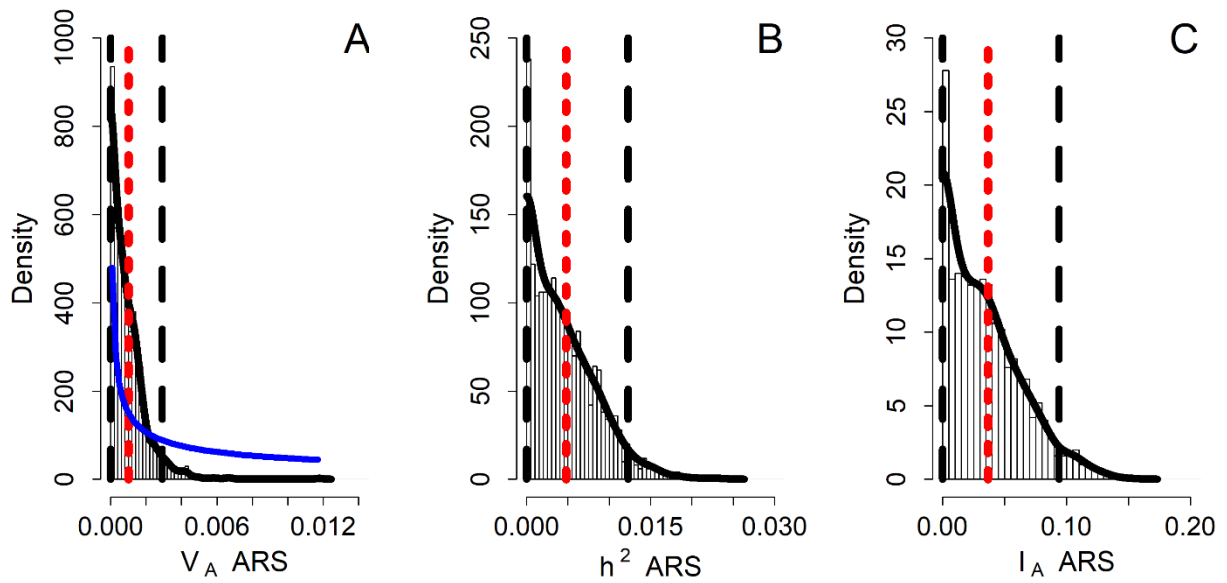


551 **Figure 3.** Posterior MCMC samples (bars), kernel density estimation (solid black line), posterior
552 mean (red dotted line), 95% Credible Intervals (black dashed lines) and prior (solid blue line) for
553 the A) additive genetic variance (V_A), B) heritability (h^2) and C) evolvability (I_A) of adult annual
554 survival (AAS). Distributions are reported on the data scale.



555 **Figure 4.** Posterior MCMC samples (bars), kernel density estimation (solid black line), posterior
556 mean (red dotted line), 95% Credible Intervals (black dashed lines) and prior (solid blue line) for
557 the A) additive genetic variance (V_A), B) heritability (h^2) and C) evolvability (I_A) of annual
558 reproductive success (ARS). Distributions are reported on the data scale.

559



560

561 **TABLES**

562 **Table 1.** Posterior modes and 95% Credible Intervals (in brackets) for data-scale variance estimates from quantitative genetic analyses
 563 of lifetime fitness components.

Fitness component	N_{individuals}	Pop. Mean	V_P	V_A	h²	I_A
Zero-inflated	5999	0.854 (0.777,0.908)	0.119 (0.083,0.173)	0.004 (0,0.008)	0.031 (0.003,0.059)	0.006 (0,0.012)
Poisson		5.71 (3.86,10.2)	17.2 (20.4,549)	2.29 (0.002,12.3)	0.023 (0,0.126)	0.088 (0,0.242)

564 The results are shown for the Zero-inflated and Poisson components of the model. All statistics (Pop. Mean, population mean; V_P,
 565 phenotypic variance; V_A, additive genetic variance; h², heritability; I_A, evolvability) presented in the table are reported on the data-scale.

566 **Table 2.** Posterior modes and 95% Credible Intervals (in brackets) for data-scale variance estimates from quantitative genetic analyses
 567 of annual reproductive success (ARS) and adult annual survival (AAS).

Fitness component	N_{observations}	N_{individuals}	Pop. Mean	V_P	V_A	h²	I_A
ASS	6873	836	0.940 (0.855,0.972)	0.056 (0.029,0.126)	0.000 (0,0.001)	0.0001 (0,0.012)	0.000 (0,0.001)
ARS			0.142 (0.108,0.236)	0.157 (0.115,0.365)	0.000 (0,0.003)	0.000 (0,0.012)	0.000 (0,0.094)

568 All statistics (Pop. Mean, population mean; V_P, phenotypic variance; V_A, additive genetic variance; h², heritability; I_A, evolvability)
 569 presented in the table are reported on the data scale.