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

32

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

35 INTRODUCTION

Fisher's Fundamental Theorem of Natural Selection postulates that "the rate of increase in fitness 36 of any organism at any time is equal to its genetic variance in fitness at that time" (Fisher 1930). 37 As such, additive genetic variance in fitness, being equivalent to the change in mean fitness 38 resulting from selection, has been considered the single most useful statistic quantifying selection 39 40 (Burt 1995). Genetic variation in fitness also is a prerequisite for adaptive evolution, as a trait must be genetically correlated with fitness to evolve through natural selection (Robertson 1966; Price 41 1970). Hence, understanding the quantitative genetics of individual variation in fitness is arguably 42 43 one of the most important aims in evolutionary ecology (Burt 1995; Ellegren and Sheldon 2008; Walsh and Blows 2009; Gomulkiewicz and Shaw 2013; Shaw and Shaw 2014; Hendry et al. 2018). 44 Considerable debate has surrounded the question of whether or not additive genetic variation 45 in fitness is expected be low (e.g., Jones 1987; Burt 1995; Houle et al. (1996), Merilä and Sheldon 46 1999; Shaw and Shaw 2014), and particularly, under which conditions (e.g., Cheverud and 47 Routman 1995; Whitlock et al. 1995). Empirical estimates of additive genetic variance in fitness 48 from wild populations are relatively scarce (e.g., Gustafsson 1986; Kruuk et al. 2000; Merilä and 49 Sheldon 2000; McCleery et al. 2004; Coltman et al. 2005; Brommer et al. 2007; Foerster et al. 50 51 2007; Teplitsky et al. 2009; Wheelwright et al. 2014; McFarlane et al. 2014, 2015; Wolak et al. 2018; de Villemereuil et al. 2019), and have so far not shed much light on this debate, since 52 estimates vary substantially, with many estimates close to zero, and few large estimates (review 53 54 by Hendry et al. 2018). Overall, Hendry and colleagues (2018) tentatively concluded that the evolvability of fitness (measured as the square of the coefficient of additive genetic variance in 55 56 fitness) is usually less than 0.2.

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

Here, we present phenotypic and pedigree data obtained from a 28-year individual-based study on common terns (*Sterna hirundo*). The common tern is a Nearctic and Palearctic colonially breeding, serially monogamous and migratory seabird. The study colony is located in the north of Germany; common terns from this colony spend their winters in western Africa and return to the breeding colony in early spring to breed or prospect potential breeding locations (Becker and Ludwigs 2004). Common terns breed annually, both parents incubate and feed the chicks, and extra-pair paternity is rare (González-Solís et al. 2001; Becker and Ludwigs 2004). Applying a series of "animal models" to data from almost 6000 pedigreed individuals across five generations, we investigate additive genetic variance for lifetime fitness (assessed as the total number of fledglings produced by a locally-born fledgling), and two of its underlying annual components: annual reproductive success and adult annual survival.

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87 METHODS

88 Study System

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

103

104 Fitness Data

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

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

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141 **Pedigree**

The pedigree was constructed by assigning all fledged offspring to their social parents, then pruned to remove individuals who were either not phenotyped or not ancestors to phenotyped individuals. For the purpose of this study, the pruned pedigree comprised 6290 records. The maximum depth was five generations, the number of paternities and maternities 2417 and 2520, respectively. The numbers of full, paternal and maternal siblingships were 2594, 10229 and 9807, respectively (see Supplementary Material for further information on the population relatedness structure). This social pedigree is a good approximation of the genetic pedigree, because common terns exhibit
very low levels of extra-pair paternity (González-Solís et al. 2001).

150

151 Quantitative Genetic Models

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

156 To model lifetime fitness, we fitted a univariate animal model with a Zero-Inflated Poisson error distribution. We fitted a Zero-Inflated Poisson distribution to better capture the nature of our 157 158 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 event occurs. In this case, a Zero-Inflated Poisson model can explicitly model the two different 160 processes, as opposed to a Poisson model that assumes only a single process to be generating the 161 data (Korner-Nievergelt et al. 2015). We fitted random intercepts for individual identity linked to 162 the pairwise relatedness matrix and for hatch-year (to account for cohort effects; e.g., Vedder and 163 164 Bouwhuis 2018). Because we modeled lifetime fitness with a Zero-Inflated over-dispersed Poisson distribution, we could estimate the covariance between the Zero-inflated and Poisson components 165 for each variance component. However, a model including additive genetic and hatch-year 166 167 covariances between the Zero-Inflated and Poisson components of the trait did not provide a better fit to the data, hence we do not model such covariances. The main models presented also did not 168 169 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 known, see Supplementary Material for detailed information on the population relatedness 172 structure), and because most fledglings came from broods where only a single individual had 173 successfully fledged (3027 broods fledged one chick, 1145 broods two, 226 broods 3, while 4 174 individuals could not be assigned to a brood). However, we did explore the potential effects of a 175 176 shared environment (due to maternal, paternal effects, or brood effects) by running two additional animal models which included one or two shared environmental effects as random effect(s). We 177 found that there was no major influence on our estimate of additive genetic variance in lifetime 178 179 fitness components, as expected given that the model presented in the main text returned a very low (close to or zero) estimate of additive genetic variance (see Suppl. Material, Tables S1 and 180 S2). 181

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

To model ARS, we assumed a Poisson error distribution with a log link function and checked whether the trait was underdispersed, which was not the case. We fitted random intercepts for individual identity linked to the pairwise relatedness matrix, individual identity not linked to the pedigree (to account for permanent environmental effects) and year of observation (to account for temporal variation across years). As fixed effects, we modelled the trait intercept and age (continuous trait ranging from 1 to 23 years), as fledgling production is known to linearly increase
with age (Zhang et al. 2015) (but see Supplementary Materials, Table S3, for results of the same
animal model without age effects).

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

All quantitative genetic models were fitted using a Bayesian framework implemented in 205 the statistical software R (v. 3.6.1, R Core Team 2019) using the R-packages MCMCglmm 206 (Hadfield 2010) and QGglmm (de Villemereuil et al. 2016). Posterior distributions were plotted 207 using the R-package *wolakR* (*github.com/matthewwolak/wolakR*). Narrow-sense heritabilities (h²) 208 were conditional on the variance explained by fixed effects and were estimated as the proportion 209 210 of the total phenotypic variance explained by the additive genetic variance. Evolvabilities (I_A) were estimated by dividing the additive genetic variance by the squared population mean (Houle 1992; 211 Hansen et al. 2011). 212

For all models we used parameter-expanded priors (Hadfield 2010). We fitted different priors for each fitness component (see Supplementary Material). The number of iterations and thinning intervals were chosen for each model so as to ensure that the minimum MCMC effective sample size for all parameters was 1000. Burn-in was set to a minimum of 5000 iterations. The retained effective sample sizes yielded absolute autocorrelation values <0.1 and satisfied convergence criteria based on the Heidelberger and Welch convergence diagnostic (Heidelberger and Welch 1981). We drew inferences from the posterior mode and 95% credible intervals (95% CI). To facilitate evolutionary inference (Bonnet et al. 2019; Morrissey and Bonnet 2019), we back-transformed the latent-scale posterior distributions of the quantitative genetic parameters to the data-scale (de Villemereuil et al. 2016).

223

224 **RESULTS**

225 Quantitative Genetics of Lifetime Fitness Components

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

Raw mean fitness was 0.72 ± 2.52 SD fledglings. Although this would indicate the 229 population to be in overall decline (a mean lifetime breeding success of two fledglings would be 230 231 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 breeders that immigrated into the population (ca. 74% ± 1 of breeders was estimated to be 233 234 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.

Simulations showed that, given our data structure and pedigree, we would not be able to detect what might be considered a *small, but substantial* signal for the Zero-inflated component of lifetime fitness: we generated a Zero-inflated component of fitness with an additive genetic variance of 0.01, and found that the average posterior mode was similar to the simulated value of V_A (average = 0.012 across the 100 replicates, Fig. S1), but the lower 95% CI limit was on average zero across replicates (95% CI = 0 - 0.023 and lower 95% CI exceeded a value of 0.0001 only 72 times across the 100 replicates, Fig. S1). When we simulated larger values of additive genetic variance (i.e., V_A = 0.05 or 0.1), our simulations showed that we would be able to detect those (average = 0.053 and 95% CI = 0.028 - 0.083 across the 100 replicates for a simulated value of 0.05; and average = 0.102 and 95% CI = 0.064 - 0.145 for a simulated value of 0.1). Lower 95% CI always exceeded a value of 0.0001 in both simulated cases (Figs. S3 and S4).

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

The results for the Poisson component of lifetime fitness are less straightforward. 256 257 Simulations showed that, given our data structure and pedigree, we would not be able to detect either small, but substantial or larger signals for the Poisson component of fitness: we generated a 258 Poisson-component of fitness with a series of evolvability values ($I_A = 0.00, 0.01, 0.05$ and 0.1), 259 260 and found that the lower 95% CI limit was on average zero in all cases (i.e., lower 95% CI did not exceed a value of 0.0001 in the vast majority of the 100 replicates, Fig. S1-4). Our analysis of the 261 262 empirical data suggested that the additive genetic variance of the Poisson component did not differ from zero, given that the associated lower 95% CI limits of VA, h² and IA converged towards zero 263

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

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

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277 Quantitative Genetics of Annual Fitness Components

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

286

287 **DISCUSSION**

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

301

302 Quantitative Genetics of Lifetime and Annual Fitness Components

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

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

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

Adult lifetime reproductive success is the sum of annual reproductive events across the life of an individual, and hence, can be decomposed into annual reproductive success and adult annual survival. Our quantitative genetic analyses of these two annual fitness components revealed a lack of substantial additive genetic variance for both (Table 2). This finding again contrasts with one from Mandarte's song sparrows, where there was evidence for moderate levels of additive genetic
variance in ARS (especially for males) and close to zero in AAS, indicating that heritable ARS
was the primary component of heritable adult lifetime reproductive success in that population
(Wolak et al. 2018).

337

338 Limitations of studying quantitative genetics of fitness in the wild

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

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

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

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

378

379 Conclusion

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

M.M. conceived the study with input from S.B. and A.C. M.M. designed and conducted the analyses, and wrote the manuscript. S.B. manages the tern data and collated the dataset. All authors 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.8kprr4xqj
411 (Moiron et al. 2022).

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540 FIGURE LEGENDS

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



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



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



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

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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	Nindividuals	Pop. Mean	VP	VA	h ²	IA	
Zero-inflated		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	5999	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)	
		(2:00,2012)	(,0 .))	((*,***=*)	(*,**=	

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; h^2 , heritability; I_A , evolvability) presented in the table are reported on the data-scale.

Table 2. Posterior modes and 95% Credible Intervals (in brackets) for data-scale variance estimates from quantitative genetic analyses

C	Fitness omponent	Nobservations	Nindividuals	Pop. Mean	VP	VA	h ²	ΙΑ
	ASS			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)
		6873	836					
	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)

567 of annual reproductive success (ARS) and adult annual survival (AAS).

All statistics (Pop. Mean, population mean; V_P , phenotypic variance; V_A , additive genetic variance; h^2 , heritability; I_A , evolvability)

569 presented in the table are reported on the data scale.