

ADAPTATION

Genetic variance in fitness indicates rapid contemporary adaptive evolution in wild animals

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The rate of adaptive evolution, the contribution of selection to genetic changes that increase mean fitness, is determined by the additive genetic variance in individual relative fitness. To date, there are few robust estimates of this parameter for natural populations, and it is therefore unclear whether adaptive evolution can play a meaningful role in short-term population dynamics. We developed and applied quantitative genetic methods to long-term datasets from 19 wild bird and mammal populations and found that, while estimates vary between populations, additive genetic variance in relative fitness is often substantial and, on average, twice that of previous estimates. We show that these rates of contemporary adaptive evolution can affect population dynamics and hence that natural selection has the potential to partly mitigate effects of current environmental change.

How fast are wild populations currently evolving in response to natural selection? The rate of adaptive evolution in nature is both of fundamental theoretical importance and of increasing practical relevance given the clear impact of human activities on the environments that wild organisms inhabit (1). There are numerous examples of phenotypic and genetic changes for traits under selection (2–5), which suggests that adaptive evolution can occur in wild populations over contemporary time scales. At the same time, however, many studies have found that trait changes do not correspond to adaptive expectations or suggest evolutionary stasis (6, 7). However, estimates of the rate of evolution of specific traits are unlikely to represent the overall rate of adaptation of a population, as natural selection acts on many traits concurrently. Instead, a comprehensive assessment of the rate of adaptive evolution in a population needs to integrate adaptive genetic changes across all traits that determine individual fitness, that is, the contribution of

an individual to the gene pool of the next generation.

According to Fisher's fundamental theorem of natural selection, the per-generation proportional change in mean absolute fitness caused by natural selection is given by the additive genetic variance in relative fitness, $V_A(w)$ (8–10). In nontechnical terms, $V_A(w)$ is the extent of heritable (transmitted from parents to offspring) genetic differences in the ability to reproduce. The realized change in mean fitness between generations may deviate from $V_A(w)$ because of concurrent effects of genetic mutations, gene flow, environmental change, or gene–environment interactions (8, 9, 11). Nonetheless, a nonzero value of $V_A(w)$ indicates that, all else being equal, natural selection contributes to an increase in mean fitness (8, 9). It also indicates that at least some of the traits that determine individual fitness are currently evolving in response to selection. Thus, $V_A(w)$ is arguably the most important evolutionary parameter in any population (9, 12).

Robust estimation of $V_A(w)$ requires accurate measures both of individual fitness and pairwise genetic relatedness for large numbers of individuals. Such data are difficult to collect for wild populations of animals or plants (13). Moreover, their analysis is made challenging by the distribution of individual fitness, which generally does not conform well to common statistical methods (14). Consequently, our knowledge of $V_A(w)$ in natural populations is currently limited: two reviews (12, 14) report estimates of $V_A(w)$ from 16 populations of 13 plant and (nonhuman) animal species with fitness measured over complete lifetimes (we discuss these results alongside our own). However, notwithstanding possible issues specific to each analysis (such as the omission of important nongenetic sources of similarity between relatives), most of these estimates were obtained from Gaussian models [for exceptions, see (10)], which generally do not fit the distribution of fitness well. In natural populations, the distribution of fitness of all individuals is typically both highly right-skewed, with most individuals having low values but a few having very high values, and zero-inflated, with an excess of zeros over and above what would otherwise be expected (zero inflation may, for example, be generated by high levels of juvenile mortality). Estimates of $V_A(w)$ from Gaussian models, and their associated uncertainty, may thus be unreliable (14, 15).

In this study, we addressed the gap in our knowledge of the value of $V_A(w)$ in the wild and its implications in terms of adaptation, trait evolution, and population dynamics. We developed and applied Bayesian quantitative genetic methods to data from long-term studies of 19 free-living vertebrate populations with high-quality lifetime reproduction and multigenerational relatedness data. Covering more populations and species than all previous studies combined, these 19 populations of 15 different species (six bird and nine mammal species) have contrasting ecologies, life histories, and social systems (10) (tables S1 and S2) and are located in diverse terrestrial biomes and continents (Fig. 1). Our analysis is restricted to birds and mammals because of their predominance among long-term studies with suitable data (13). The populations

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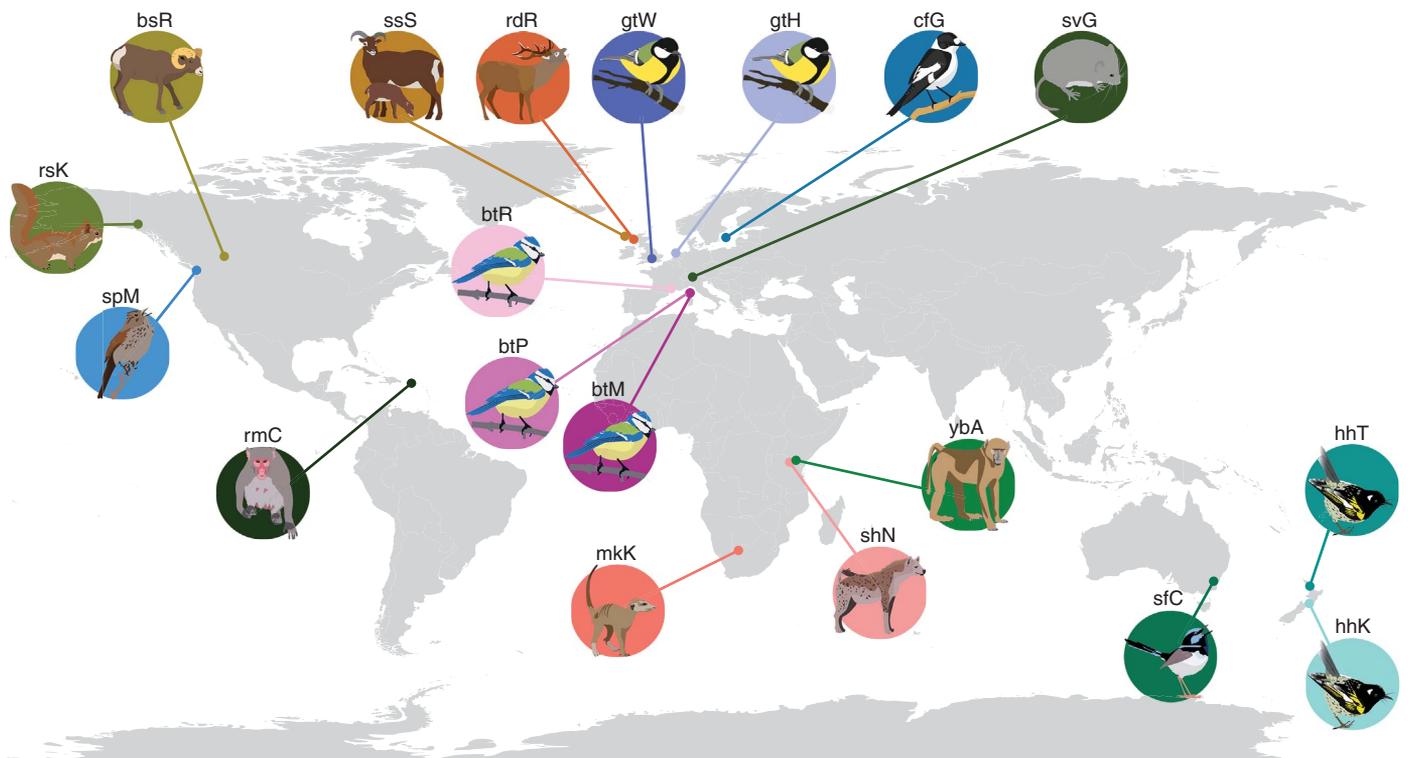


Fig. 1. Locations of the 19 long-term population studies. Abbreviations are as follows: bsR, bighorn sheep on Ram Mountain in Canada; ssS, Soay sheep on St Kilda, UK; rdR, red deer on the Isle of Rum, UK; gtW, great tits in Wytham Woods, UK; gtH, great tits in Hoge Veluwe, the Netherlands; cfG, collared flycatchers on Gotland, Sweden; svG, snow voles in Graubünden, Switzerland; rsK, red squirrels in Kluane, Canada; btR, blue tits at la Rouvière, France; spM,

song sparrows on Mandarte Island, Canada; btP, blue tits at Piriou, France; btM, blue tits at Muro, France; rmC, rhesus macaques on Cayo Santiago, Puerto Rico; ybA, yellow baboons at Amboseli National Park in Kenya; hhT, hihi on Tiritiri Matangi Island, New Zealand; shN, spotted hyenas in the Ngorongoro Crater, Tanzania; mkK, meerkats in the Kalahari, South Africa; sfc, superb fairy-wrens in Canberra, Australia; hhK, hihi in Karori, New Zealand.

have been monitored for between 11 and 63 years, providing fitness records for 561 fully monitored cohorts totaling 249,430 individuals of both sexes (10). For all datasets used here, an individual's fitness was measured as "lifetime breeding success," or the total number of offspring produced over its lifetime, irrespective of offspring survival. While there are numerous definitions of fitness, each motivated by different theoretical frameworks (16), measuring fitness as lifetime breeding success corresponds most closely to a life cycle-calibrated "zygote-to-zygote" definition of individual fitness, consistent with quantitative genetic theory (17). Individuals were identified soon after birth or hatching, and fitness was estimated for all known individuals in each population, including the often-large proportion that died as juveniles (10). We modeled absolute lifetime breeding success using a quantitative genetic form of a mixed-effects model known as an "animal model" (18), assuming that lifetime breeding success follows zero-inflated overdispersed Poisson distributions and including relevant covariates (such as inbreeding, genetic group, sex, and cohort; see tables S3 and S4, supplementary text S1 for model details, figs. S1 and S2 for eval-

uation of model goodness of fit, supplementary text S2, and fig. S3 for prior distribution). The zero-inflated Poisson models were fitted to absolute fitness data, and the resulting parameter estimates, obtained on link-function scales, were then back-transformed to derive estimates of $V_A(w)$ and other components of variances for relative fitness on the scale of the data (15). We first ran one model for each study population and subsequently combined results into a meta-analysis (10).

We found evidence for additive genetic variance in relative fitness in multiple populations. Our models provided estimates of $V_A(w)$ with posterior modes ranging from 0.003 to 0.497 (Fig. 2A). The 95% credible intervals (95% CI) for $V_A(w)$ excluded values below 0.001 in 10 of the 19 populations and excluded values below 0.01 in eight (thresholds explained in caption of Fig. 2A and supplementary text S2 and S3). Therefore, there was clear evidence that selection contributed to genetic changes, and hence a predicted increase in fitness, in roughly half of the study populations (9, 19). Across populations, the median of the posterior modes for $V_A(w)$ was 0.100, and the meta-analytic mean of $V_A(w)$ was 0.185, 95% CI [0.088; 0.303]. There was

also considerable variation among populations, with a meta-analytic among-population standard deviation in $V_A(w)$ of 0.11, 95% CI [0.01; 0.26]. The median and mean values of $V_A(w)$ were about four and two times larger than those of previous estimates (previous median: 0.023; previous mean: 0.092) (12, 14). Our values can be considered large given theoretical considerations (supplementary text S3 and fig. S4), and they were robust to the modeling of possible confounders: inbreeding, sex, linear environmental changes in mean fitness, gene flow due to immigration, and variance among cohorts and among mothers (10) as well as mother-by-cohort interactions, social group effects (supplementary text S4, table S5, and fig. S5), and the social inheritance of social dominance within families (supplementary text S5 and figs. S6 and S7). For completeness, we also present estimates relating to an alternative formulation of Fisher's fundamental theorem expressing change in terms of absolute fitness leading to the same conclusions (supplementary text S6 and fig. S8).

Previous work on adaptive evolution has often focused on the heritability of fitness, $h^2(w) = V_A(w)/V_P(w)$, where $V_P(w)$ is the phenotypic variance in relative fitness, or

the “opportunity for selection” (20). However, $h^2(w)$ may be a poor measure of the overall rate of adaptive evolution (20). In natural conditions, stochastic or unaccounted environmen-

tal variation is expected to dominate variation in individual fitness, even in the presence of large deterministic sources of variation in fitness (21), so that $h^2(w)$ may be small even

when $V_A(w)$ is large (21, 22). In line with this expectation, we found that $h^2(w)$ was generally small, with a meta-analytic average of 2.99%, 95% CI [0.80; 6.60%] and a value of <1% in

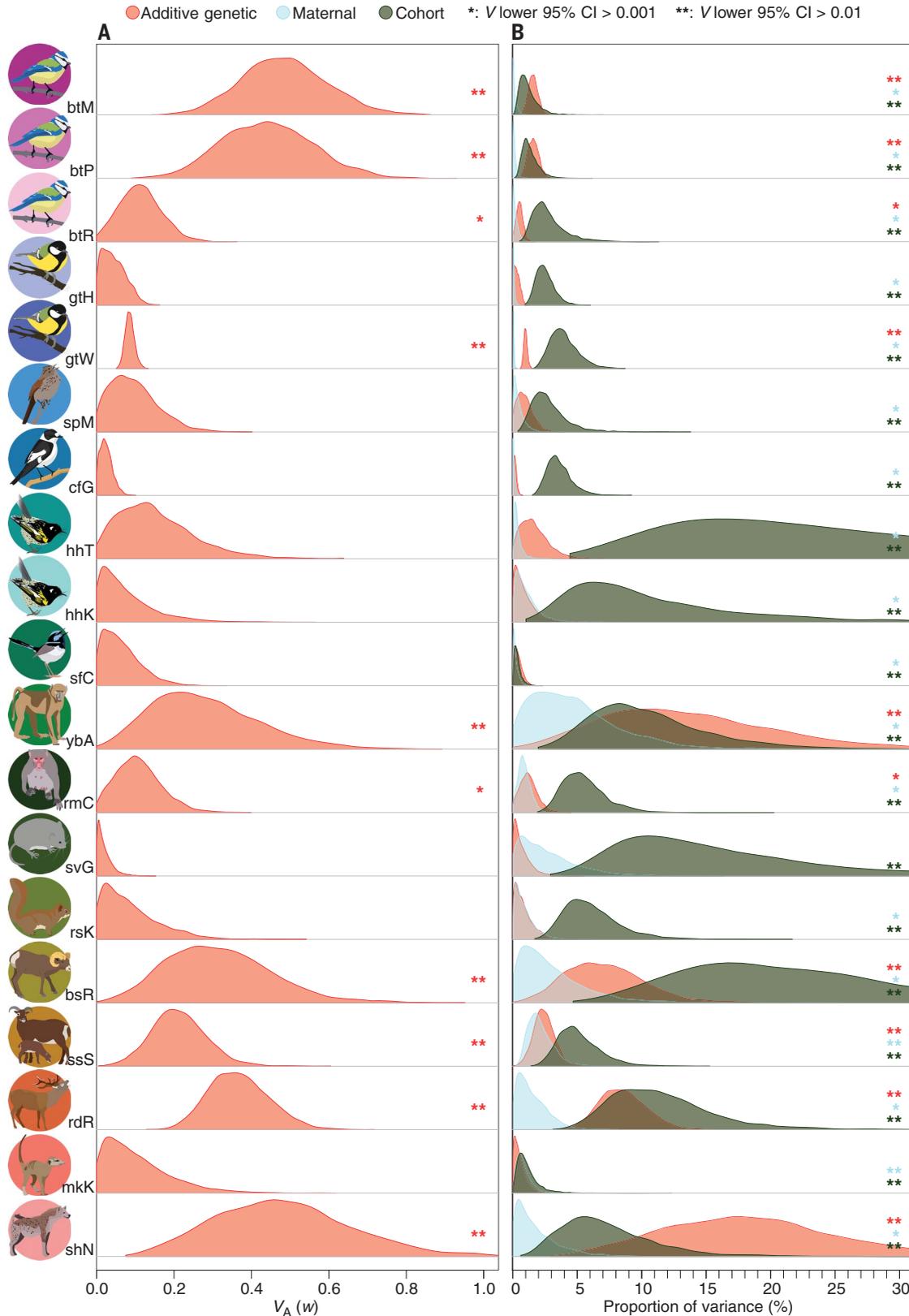


Fig. 2. Additive genetic variance and other components of variance in relative fitness. Panels show posterior distributions of (A) additive genetic variance in relative fitness, $V_A(w)$, and (B) proportion of phenotypic variance in fitness due to different variance components: additive genetic variance (i.e., heritability; red), maternal effect variance (light blue), and cohort variance (dark green). Species are ordered by phylogenetic proximity. Each distribution has an area of 1 but is scaled arbitrarily on the y axis to aid comparison. Single asterisk indicates that the 95% CI of a variance component does not overlap 0.001 [approximately the mode of the prior distribution for $V_A(w)$; supplementary text S2]. Double asterisk indicates that the 95% CI does not overlap 0.01 (the approximate threshold between small and moderate rates of adaptive evolution; supplementary text S3). Asterisks indicate absolute variance values, not proportions of variance. Abbreviations of population names are as in Fig. 1.

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11 populations (Fig. 2B), similar to previous estimates of $h^2(w)$ (14). Nevertheless, estimates of $h^2(w)$ were of similar magnitude to the proportion of variance explained by maternal effect and cohort variances (Fig. 2B, supplementary text S7, and tables S6 to S10 for parameter estimates on different scales). Furthermore, $h^2(w)$ was highly variable between populations and was sometimes substantial, with posterior modes ranging from 0.019 to 17.1%.

What do our estimates of $V_A(w)$ imply about the evolution of traits in our study populations? $V_A(w)$ is the partial increase in fitness expected to result from the combined responses to selection across heritable traits (23). Therefore, a nonzero $V_A(w)$, as was found for at least half of our study populations, implies that for one or several traits, the responses to selection tend to cause adaptive change, although the total change may be affected by mutations or environmental change (19). The value of $V_A(w)$ sets an upper bound for the possible per-generation response to selection of any trait (19). Given the meta-analytic estimate of $V_A(w) = 0.185$ and a trait with a heritability of 0.3 [an average value for trait heritability in wild populations (24)], the maximal rate of response to selection is 0.24 standard deviations per generation (10, 19). Across our 19 populations, the upper bound of response to selection for a trait with a heritability of 0.30 varies from 0.05, 95% CI [0.01; 0.13], to 0.39, 95% CI [0.29; 0.50], standard deviations. These upper bounds are substantial: For comparison, in natural populations the rates of phenotypic change, irrespective of whether the change is known to be adaptive, are rarely above 0.03 standard deviations (~10% of estimates) and only very rarely above 0.13 standard deviations (~5% of estimates) (2). Furthermore, evolutionary studies of wild populations, including several conducted in our study populations, have often failed to detect phenotypic change in response to current selection (5, 6, 25). Our results may therefore appear at odds with these observations. However, attempts to estimate genetic evolution of traits, as opposed to just phenotypic trends, remain rare and underpowered (25). Genetic evolution of traits may be masked at the phenotypic level, either because phenotypic plasticity hides genetic change (6) or because direct evolution is counterbalanced by the evolution of “indirect genetic effects,” that is, the effect of other individuals’ genotypes (26). Moreover, approaches to estimating genetic change for a trait, such as estimation of trends in individual genetic merit (“breeding values”) (27) or by estimation of polygenic scores (28), may have limited statistical power. Finally, if $V_A(w)$ is ultimately driven by the cumulative effects of many traits evolving in response to selection, the evolutionary change in each trait will be small and even more

difficult to identify statistically. Any or all of these scenarios could prevent observed rates of phenotypic change in single traits from reaching the upper bound of what might be possible given the observed levels of $V_A(w)$.

Irrespective of the rates of adaptive evolution in the potentially many traits that contribute to $V_A(w)$, our estimates of their combined effect, summarized in $V_A(w)$, indicate that adaptive evolution may have substantially affected recent population dynamics (see supplementary text S6 and S8 and fig. S8). For instance, in a thought experiment assuming that no forces oppose adaptive evolution and that $V_A(w)$ remains constant, 11 of our 19 populations would recover from an arbitrary one-third reduction in fitness in fewer than 10 generations (supplementary text S8). Moreover, the median $V_A(w)$ of 0.10 means that in half the populations, natural selection tends to increase mean absolute fitness by at least 10% every generation. Whereas such a change would lead to exponential population growth if not counterbalanced, none of our study populations showed any exponential increase in population size such as that predicted by the thought experiment (supplementary text S9). This indicates that any adaptive evolution was countered by simultaneous deleterious effects of other processes such as mutation, gene flow, or environmental changes (19). The presence of these counterbalancing forces, as well as potential changes in future selective pressures and the potential instability of $V_A(w)$ in future environments, makes it impossible to project whether the contemporary adaptive evolution that our results indicate is sufficiently fast and lasting to ensure population persistence. Other studies that focused on specific traits, rather than on the net effect of selection on fitness, suggest that short-term phenotypic changes in response to climate change are overall insufficient to ensure the persistence of populations (29, 30). Crucially, however, our finding that most populations harbor biologically meaningful levels of additive genetic variance in fitness indicates that the machinery of adaptive evolution often operates at a substantial pace on generation-to-generation time scales. Without ongoing adaptive genetic changes, these populations would presumably have had (often substantially) lower growth rates over recent generations.

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SUPPLEMENTARY MATERIALS

[science.org/doi/10.1126/science.abk0853](https://doi.org/10.1126/science.abk0853)
Materials and Methods

Supplementary Text S1 to S10
Figs. S1 to S10
Tables S1 to S10
References (31–242)
MDAR Reproducibility Checklist
Data S1
Code S1

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Rapid change

Human impacts are leading to exceedingly rapid alteration of our world, from land conversion and habitat loss to climate change. Some have proposed that rapid adaptation could help some species persist in the face of these changes, but questions remain about whether adaptation could occur rapidly enough to make a difference. Bonnet *et al.* looked at additive genetic variance, which determines the contribution of selection to genetic change that increases fitness, in long-term data from 19 species and found it to be higher than expected—often substantially higher (see the Perspective by Walsh). These results suggest that many species may have some capacity to adapt to our changing world. —SNV

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