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The Contemporary Evolution of Fitness

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Abstract

The rate of evolution of population mean fitness informs how selection acting in contemporary populations can counteract environmental change and genetic degradation (mutation, gene flow, drift, recombination). This rate influences population increases (e.g., range expansion), population stability (e.g., cryptic eco-evolutionary dynamics), and population recovery (i.e., evolutionary rescue). We review approaches for estimating such rates, especially in wild populations. We then review empirical estimates derived from two approaches: mutation accumulation (MA) and additive genetic variance in fitness (I_{Aw}). MA studies inform how selection counters genetic degradation arising from deleterious mutations, typically generating estimates of <1% per generation. I_{Aw} studies provide an integrated prediction of proportional change per generation, nearly always generating estimates of <20% and, more typically, <10%. Overall, considerable, but not unlimited, evolutionary potential exists in populations facing detrimental environmental or genetic change. However, further studies with diverse methods and species are required for more robust and general insights.

WHY AND HOW FITNESS MATTERS

fitness: various specific definitions, with the common overall objective of measuring the success of a potentially reproducing entity (see section titled Concepts of Fitness)

population mean fitness: individual absolute fitness, averaged across all individuals in a population

genetic degradation: deleterious effects on fitness caused by mutation, gene flow, genetic drift, recombination, or inbreeding

rate of evolution of fitness: the per-generation genetically based change in population mean fitness

plastic rescue: increased population mean fitness due to phenotypic plasticity in populations that would otherwise have gone extinct

Natural selection favors the adaptation of populations to their local environments, which should—to the extent possible—optimize phenotypic trait values and maximize individual fitness (Fisher 1930, Fisher 1958, Burt 1995, Orr 2009). Through this process of adaptation, evolution by natural selection should also tend to increase population mean fitness and, hence, increase population growth rates and the probability of population persistence (Saccheri & Hanski 2006, Kinnison & Hairston 2007, Hendry 2017); although that is not always the case (Rankin & López-Sepulcre 2006). The rates of these evolutionary changes are particularly important in two general contexts, here categorized as "environmental change" and "genetic degradation." The basic idea is that these two classes of processes are constantly acting to cause maladaptation and decrease organismal fitness. Hence, the rate at which maladaptation can be countered by natural selection determines just how effective adaptive evolution can be in facilitating the persistence, growth, and spread of populations and species. It is now widely established that phenotypic traits can show substantial adaptive evolution on contemporary time frames, such as years to a few centuries (Hendry & Kinnison 1999, Hendry et al. 2008). However, it is the rate of evolution of *fitness* that is the fundamental parameter underlying and linking evolutionary biology and population ecology, with far-reaching implications for endeavors to manage and conserve wild populations experiencing rapid environmental change and genetic degradation.

Environmental change should initially render most populations less well-adapted for their immediate local environments and therefore should decrease population mean fitness, leading to decreased population growth rates and, potentially, decreased population sizes (Lynch & Lande 1993, Gomulkiewicz & Holt 1995, Carlson et al. 2014, Bell 2017). These decreases are expected to be fastest when phenotype–environment mismatches are greatest and when adaptive phenotypic responses are slowest. Support for these expectations comes from studies showing that recent climate change negatively affects population growth in species such as pied flycatchers (*Ficedula hypoleuca*) in the Netherlands (Both et al. 2006), migratory birds in Europe (Møller et al. 2008), eelpout (Zoarcidae) in the Baltic Sea (Pörtner & Knust 2007), and flowering plants in New England (Willis et al. 2008).

In such cases of environmentally induced maladaptation, natural selection should favor adaptation to the changed environment, which should then increase population mean fitness and hence the population growth rate, potentially contributing to population recovery. This process is often termed "evolutionary rescue" (Lynch & Lande 1993, Gomulkiewicz & Holt 1995, Carlson et al. 2014, Bell 2017), with one alternative being "plastic rescue," in which adaptive phenotypic change instead reflects environmentally induced plasticity (Chevin et al. 2013, Kovach-Orr & Fussmann 2013). Although definitive examples of evolutionary rescue in the wild are very rare (Gomulkiewicz & Shaw 2013, Vander Wal et al. 2013, Carlson et al. 2014), evidence from laboratory studies is extensive (Bell 2017). For instance, yeast (*Saccharomyces cerevisiae*) populations challenged with high salt concentrations rapidly decrease in density as a result of maladaptation but then recover as they evolve the ability to tolerate high salt (Bell & Gonzalez 2011). In such cases of environmental change, the rate at which fitness evolves through natural selection strongly influences (*a*) the rate at which initial maladaptation-induced decreases in population size are arrested, (*b*) the probability of population persistence, and (*c*) how quickly and completely population sizes can rebound.

Even without external environmental change, all populations are susceptible to genetic degradation owing to deleterious mutations (Lynch & Gabriel 1990), maladaptive gene flow (Kirkpatrick & Barton 1997a, Lenormand 2002), some forms of recombination and segregation (Charlesworth & Barton 1996, Becks & Agrawal 2011), genetic drift (Barton & Partridge 2000), and inbreeding (Charlesworth & Willis 2009). As examples from the wild, maladaptive gene flow reduces

population size and patch occupancy in walking stick insects (*Timema cristinae*) (Farkas et al. 2013, 2016), and inbreeding depression reduces fitness in many small populations (Ellstrand & Elam 1993, Frankham 2015). If population sizes and growth rates are to be maintained in the face of such genetic degradation, natural selection must constantly weed out detrimental variants (Burt 1995). In the above examples, natural selection can act against immigrant genotypes (Nosil et al. 2005) and against inbred individuals that express recessive deleterious mutations (Keller & Waller 2002). In such cases, the rate at which fitness can evolve determines the extent to which genetic degradation can be offset by natural selection, thereby potentially preventing or reversing decreases in population size. This ability of selection to maintain populations that would otherwise decrease owing to genetic degradation is a form of "cryptic eco-evolutionary dynamics" (Kinnison et al. 2015, Hendry 2017).

cryptic eco-evolutionary dynamics: various manifestations, but herein defined as evolutionary change that maintains population size where it would otherwise decrease

The various factors just described can increase or decrease fitness through various mechanisms, from among which our current interest is centered on the rate at which natural selection increases population mean fitness. This rate can be quantified in several ways, most obviously the proportional increase in population mean fitness per generation (Burt 1995). By determining whether natural selection typically increases fitness by, for example, 0.1%, 1%, or 10% per generation, we can then address a series of fundamental questions in evolutionary biology. What rate of environmental change can adaptive evolution offset? How much maladaptive gene flow can a population receive before decreasing in abundance? How inbred can populations become before persistence becomes unlikely? These and many other theoretical and practical questions require a diversity of studies that quantify the rate of evolution of fitness.

Considerable variation in fitness clearly exists in wild populations; however, this variation stems from a combination of nonheritable stochastic contributions and heritable deterministic contributions—or "pluck" and "luck" as termed by Snyder & Ellner (2018). Hence, without focused studies on the former, it is uncertain just how much potential exists in contemporary wild populations for natural selection to drive evolutionary increases in fitness (Shaw & Shaw 2014). To date, the only article to have summarized the state of empirical knowledge on this potential was Burt (1995), who concluded that the amount by which natural selection increases mean fitness each generation (or degradation decreases mean fitness) will usually be between 0.1% and 30%; more tentatively, he suggested that values will typically fall between 1% and 10%. Given the subsequent 23 years of empirical studies, often with much improved methodology, it is now timely to provide a new empirical assessment of rates of evolution of fitness.

In the sections that follow, we first discuss key conceptual issues surrounding the definition, estimation, and interpretation of "fitness." We then highlight various approaches through which the rate of evolution of fitness can be estimated, emphasizing—to the extent possible—applications in contemporary wild populations experiencing real-world environmental change or genetic degradation. Finally, we provide a quantitative review of estimates generated through the two most commonly used approaches: mutation accumulation experiments and estimation of the additive genetic variance in fitness.

CONCEPTS OF FITNESS

Most broadly (and vaguely) construed, fitness is the "success" of a replicating biological unit. However, concepts and opinions vary when attempting to define precise quantities that can be measured and modeled, in part because the best definition depends on the question of interest and the biological system under consideration (Roff 1992, Brommer 2000, Orr 2009, Sæther & Engen 2015). We therefore first briefly note some of the fundamental distinctions, which helps to set the wider stage for our current specific focus. First, fitness can be considered as a property of biological

units at several different levels, such as cells, individuals, genotypes, families, groups, populations, species, or higher taxonomic levels. Second, absolute fitness is the fitness of a biological unit (e.g., number of offspring of an individual) ignoring the fitness of other units (e.g., other individuals in the population), whereas relative fitness is the fitness of a unit relative to those other units (e.g., number of offspring of an individual divided by the mean number of offspring per individual in the population). Third, fitness can be considered as the expected fitness of a unit given its genotype or phenotype or as the realized fitness of that unit, with the latter also being subject to various forms of stochasticity (Orr 2009). Fourth, the fitness of a unit averaged through time (e.g., long-term population mean fitness) is often indexed as geometric mean fitness, which weights low values more heavily, rather than as arithmetic mean fitness (Simons 2002)—and a number of other improvements are possible (Sæther & Engen 2015).

No single operational metric of fitness is universally applicable across all contexts, yet what all concepts seek to capture is some sense of the extent to which a given biological unit will increase or decrease in abundance through time. In laboratory studies, for example, two common operational metrics are the intrinsic rate of increase of a unit or the carrying capacity of that unit (Roff 1992, Kassen 2014), depending on whether or not the context of interest involves density dependence. These metrics are often unattainable in wild populations; although some studies have been able to track the "trajectory of abundance" of particular genotypes, phenotypes, or species through time (e.g., Nosil et al. 2018). Most field studies, however, are too short relative to the life span of the organism or are unable to accurately track specific biological units. Hence, most studies focus on short-term—usually one or a few generations—estimates of fitness that are expected to dictate how genotypes, phenotypes, or species will increase or decrease in the immediate future.

One particularly useful metric of fitness that is rooted in basic quantitative genetic theory is "lifetime reproductive success" (LRS), defined as the total number of zygotes produced per zygote (Arnold & Wade 1984). This metric, unlike the approach of measuring fitness as an individual parent's number of offspring that survive to reproduce, does not assign offspring survival as a parental trait and thus allows genetic variation to be distinguished from selection (Arnold & Wade 1984). However, adequately capturing lineage trajectories for iteroparous species with age structure and overlapping generations can require metrics of fitness that consider the timing of reproduction, as well as multiple aspects of population dynamics and environmental fluctuations, and resulting population density (Brommer 2000, Brommer et al. 2002, Moorad 2014, Sæther & Engen 2015).

For many organisms of interest, however, even LRS is difficult if not impossible to estimate in wild populations, or at least to estimate with a useful degree of precision and accuracy. Consequently, many studies instead estimate fitness components, especially survival and reproductive success, as seen in numerous studies estimating selection on phenotypic traits in the wild (Kingsolver & Diamond 2011). Although fitness components such as these do indeed contribute to fitness, they can trade off with each other, making it hard to directly or accurately predict overall fitness (Roff 1992). Moreover, fitness components are commonly estimated over only part of an organism's life cycle and thus might not predict the same parameters over the entire life cycle (Schluter et al. 1991). Examples illustrating the problem (Roff 1992) include early life survival trading off with late-life survival, and high reproductive effort in one period trading off with reproductive effort in other periods. Other commonly used fitness "surrogates," such as foraging rate, physiological growth rate, and body size, are even less likely to accurately reflect overall fitness and, hence, do not provide robust insight into the rate of evolution of fitness.

The above alternative concepts and definitions are not all equally relevant to our current interest in the rate of evolution of fitness. First, given our interest in the ability of populations to respond to environmental change and genetic degradation, we need to understand the evolution of population mean fitness—although note that one way to predict this evolution is to estimate the variance in

fitness among individuals (details below). Second, absolute (as opposed to relative) fitness is needed to understand the population dynamic consequences of natural selection and, hence, outcomes such as evolutionary rescue. Third, the desired inferences are about expected fitness, although in practice, estimation often comes about through the quantification of realized fitness. Fourth, practical concerns dictate that fitness assays for most organisms in most contexts must focus on short-term changes, precluding attention to geometric mean fitness across generations and other time-dependent metrics.

HOW TO ESTIMATE THE EVOLUTION OF FITNESS

When considering the evolutionary dynamics of fitness, an important distinction needs to be made between natural selection favoring individuals with higher fitness and the evolution of that fitness at the population level. Take, for illustration, a small inbred population in which fitness is low owing to the expression of recessive deleterious mutations (Ellstrand & Elam 1993, Frankham 2015). Introducing unrelated individuals into this population can rapidly increase population mean fitness by masking recessive mutations in the resulting heterozygous outbred offspring (Tallmon et al. 2004, Carlson et al. 2014, Frankham 2015, Whiteley et al. 2015). Although this immediate increase in fitness involves an evolutionary process (immigration of new alleles), it might not initially involve natural selection. However, natural selection then might act against inbred individuals that are homozygous for deleterious mutations, as opposed to outbred individuals that are heterozygous. The result of this selection should be an evolutionary decrease in the frequency of the deleterious mutations (i.e., purging), in which case natural selection has now caused an evolutionary increase in population mean fitness (Crnokrak & Barrett 2002).

It is also important to remember that not all variation in fitness is genetic, and therefore evolvable—neither is all genetic variation in fitness equally evolvable. Nongenetic effects, including stochasticity (environmental and demographic) and plasticity (including maternal effects), can increase or decrease the mean and variance in fitness among individuals in a population, thus contributing to changes in mean population fitness following environmental change. Such nongenetic changes could substantially affect population growth (Kovach-Orr & Fussmann 2013), but they are not the evolution of fitness, although they could become so if the plastic response then evolves (Chevin et al. 2013). Further, the specific type of genetic variation that dictates the rate at which natural selection can increase fitness is the additive genetic variance (Fisher 1930, 1958), as opposed to dominance and epistasis. Yet, those nonadditive genetic effects should not be ignored because they can strongly influence the mean and variance in fitness, as seen in recently evolved differences between populations being shaped by dominance and epistasis (Roff & Emerson 2006, Carroll 2007). Further, nonadditive genetic variance can be converted to additive genetic variance as a result of inbreeding or environmental change, thus contributing directly to the evolution of fitness (van Buskirk & Willi 2007).

The above processes can change both the mean and variance in fitness for a population, which highlights the two complementary ways of estimating the rate of evolution of fitness. First, studies can quantify genetically based changes in mean fitness within populations across generations, manifest either as increased fitness resulting from adaptive responses to environmental change or as decreased fitness resulting from multiple forms of genetic degradation (Burt 1995). Second, studies can quantify the additive genetic variance in fitness within populations, which thus predicts the per-generation increase in mean fitness expected to result from natural selection (Fisher 1930, 1958). Correspondingly, in the following sections, we describe a series of approaches for estimating the rate of evolution of fitness in contemporary populations from the perspectives of mean fitness following environmental change, mean fitness due to genetic degradation, and additive genetic

Table 1 Summary of different approaches for estimating the evolution of fitness, with some of their advantages, disadvantages, and practical difficulties

Some advantages	Some disadvantages and practical difficulties
Experimental introductions in nature	
Could be a realistic indicator of contemporary responses to (real) environmental changes	Can be hard to maintain a true ancestor for later comparison Ethical concerns preclude certain experiments True replicates are hard to implement, as are true controls Can be hard to control for environmental (plastic) effects
Resurrection experiments	
Could be a realistic indicator of past responses to (real) environmental changes	Evolution might not be linear Resurrected genotypes might not be representative of past populations Fitness is usually tested in artificial environments in the laboratory Some fitness determinants (e.g., host–parasite arms races) might undergo cyclical changes
Mutation accumulation	
A reasonable number of existing studies for comparison A clear single mechanism	Only considers one process influencing fitness Lethal mutations are typically ignored Mutations can be beneficial Most fitness assays are conducted in artificial laboratory environments
Gene flow manipulation	
Fairly easy to manipulate in nature A clear single mechanism	Only considers one process influencing fitness Gene flow can have beneficial effects Studies in nature tend to measure effects on adaptive traits, rather than on fitness
Recombination manipulation	
A clear single mechanism	Easiest to interpret the outcome when the environment is stable Difficult to determine what constitutes a stable environment Not easy to achieve tight control over recombination
Additive genetic variance in fitness	
Strong connections to the fundamental theorem of natural selection A reasonable number of existing studies for comparison Integrates multiple processes influencing fitness Can be estimated for wild populations	Represents potential evolution of fitness, which might not reflect the actual evolution of fitness Sex differences in variances (and covariances) can bias estimates of the overall rate of evolution Challenging to estimate and interpret given a non-Gaussian distribution of fitness Still very hard to estimate accurately and precisely in wild populations

variance in fitness (**Table 1**). For the first two approaches, few studies have been conducted in the wild, and so we start by describing idealized implementations in simplified laboratory contexts. For the third approach, enough studies have been conducted in nature that we can proceed directly to that context.

Evolution of Mean Fitness Following Environmental Change

The fundamental idea here is that environmental change shifts the optimum phenotype away from the mean phenotype currently expressed in the population, thus reducing mean fitness by causing a phenotype–environment mismatch (**Figure 1**, ❶). Additionally, environmental change can decrease population mean fitness by changing the shape of the fitness function or the mean and variance in phenotypes. Natural selection then should lead to adaptive evolution, which can be measured as changes in mean phenotypes toward the new optimum, changes in the variance in

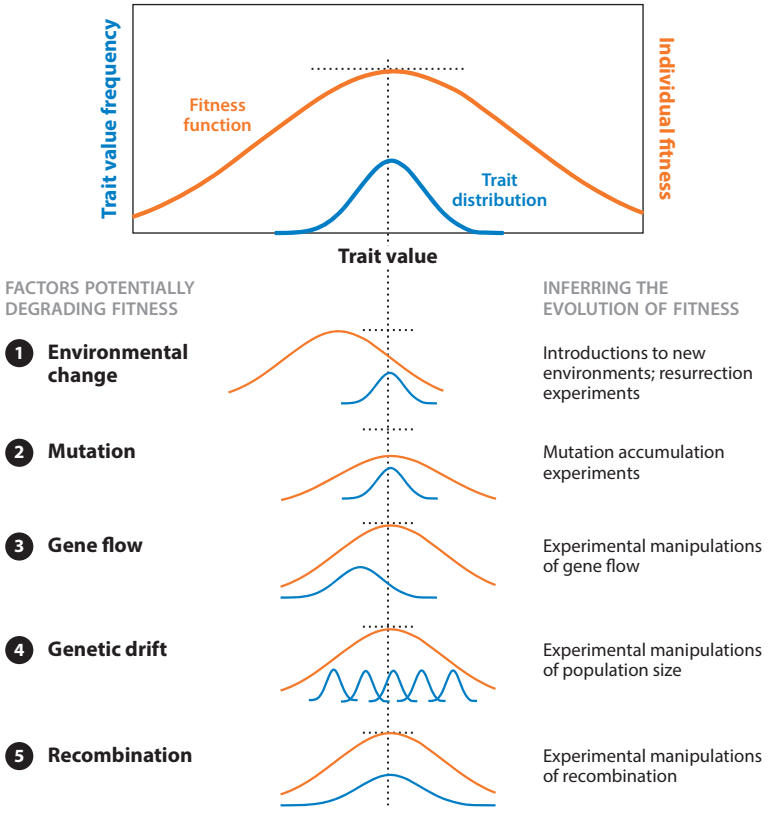

Figure 1

Conceptual depiction of how environmental change and various forms of genetic degradation (mutation, gene flow, genetic drift, and recombination) can influence fitness by altering fitness functions and trait distributions. The top panel shows the expectation for a well-adapted population in a reasonably stable environment, where selection around a particular phenotypic optimum (peak of the fitness function, *upper curve in orange*) generates a trait distribution (*lower curve in blue*) with a mean that is centered under the optimum and a relatively low trait variance (Haller & Hendry 2014). The lower panels show how this well-adapted scenario is modified by (❶) environmental change (shift in optimum), (❷) mutation (lowering of the fitness function for a given trait value), (❸) gene flow (shift in trait mean and increase in trait variance), (❹) genetic drift (shift in trait means in random directions and to varying degrees, combined with decreases in trait variance), and (❺) recombination or segregation (increase in trait variance). The dotted lines in the lower panels provide reference points to the original optimum (*vertical line*) and the original maximum fitness (*horizontal lines*) from the upper panel. The right-hand text outlines some methods for inferring the rate of evolution of fitness in each case.

phenotypes, and—most relevant here—increases in population mean fitness (Hendry & Gonzalez 2008). Specifically, after an abrupt environmental change, the per-generation genetically based increase in population mean fitness provides a measure of the rate of evolution of fitness.

An ideal empirical implementation of this approach would center on "turning off" evolution in some populations but not others, as can be achieved relatively simply in some laboratory studies (Kassen 2014). For instance, populations of microbes can be divided into two groups, one of which adapts to a new environment and the other of which is preserved (e.g., frozen) in a state of nonevolutionary stasis. After populations of the first group evolve for some period of time in the new conditions, populations of the other (ancestral) group can be resurrected (e.g., thawed out)

and fitness can be compared between the evolving and nonevolving groups in each environment (Elena & Lenski 2003, Kassen 2014). For organisms that cannot be put into stasis, the (hopefully) nonevolving group is often maintained in the ancestral environment (Kawecki et al. 2012), although complications can arise owing to continuing evolution even in that group.

Under some conditions, this approach that "turns off" evolution can be applied in the wild. For example, organisms that can be frozen, or induced into dormant (or resting) stages, can be used to compare present and future genotypes in future environments. This approach will become easier in the future through initiatives such as Project Baseline, which collects and stores seeds for future germination and growth studies (Etterson et al. 2016, Franks et al. 2017). For organisms that have naturally entered dormant stages, "resurrection ecology" can be used to compare past and current genotypes in current environments (Jensen et al. 2012, Orsini et al. 2013, Franks et al. 2017). Indeed, this approach is increasingly used to assess adaptive evolution in response to specific environmental changes. As one example, resurrecting *Daphnia* and their parasites from cores of lake sediments has shown that the parasites were adapting to changing host genotypes over only a few years (Decaestecker et al. 2007). As another example, experimental germination of seeds produced in different years has demonstrated that postdrought plant genotypes are better adapted to drought conditions than are their predrought ancestors (Franks et al. 2007). The critical next step for such studies would be to assess the fitness of populations originating from different time periods when tested under contemporary conditions in the wild.

For organisms for which dormant stages are not produced or are not available, the equivalent studies instead compare derived populations evolving in new environments to their ancestors remaining in the original environment (Reznick & Ghalambor 2005). For instance, Gordon et al. (2009) studied guppy (*Poecilia reticulata*) populations introduced 8 years (13–26 generations) previously into new environments in the wild. The investigators marked and released, into the new environments, guppies that evolved in those new environments and also guppies from the ancestral environments, finding up to 59% higher survival in the former (2.3–4.5% increase per generation). Similarly, Kinnison et al. (2008) studied chinook salmon (*Oncorhynchus tshawytscha*) introduced 83 years (26 generations) previously into two sites with different seasonal migration distances. The investigators marked juveniles from the two populations and released them together at the site with the longest migration distance and also at a "control" site with a very short migration distance. Using a measure of fitness based on survival rates multiplied by fecundity, no difference was found between the populations at the control site, whereas a 120% difference was found between the populations at the long migration site, thus suggesting a 4.6% fitness increase per generation.

Although these two studies were meritorious for using real populations in real environments, neither was optimal for estimating the rate of evolution of fitness. In particular, the guppy study did not control for environmental effects and only examined one fitness component (survival). The salmon study was better in both of these regards, but it looked at divergence between two evolving populations rather than paired evolving versus nonevolving populations. Also, the salmon study did not consider some fitness components, most notably egg and juvenile survival. Nonetheless, these two studies illustrate that such experiments and inferences are potentially feasible in nature—given appropriate control over environmental effects, minimal (or known) genotype-by-environment interactions, and appropriate measures of fitness.

Evolution of Mean Fitness Following Genetic Degradation

The key principle here is that, even in a constant environment, a number of genetic degradation processes (mutation, gene flow, genetic drift, recombination) are continually decreasing

population mean fitness and potentially also altering the genetic variance in fitness. If fitness is to be maintained, natural selection must be constantly counteracting those degradation processes (Burt 1995), as is often discussed in the context of various forms of genetic "load" (Barton & Partridge 2000). Hence, if investigators can measure the extent to which each degradation process (or their combinations) decreases population mean fitness, an estimate can be generated as to how effective natural selection typically must be to counteract that process. Such estimates thus reflect the rate of evolution of fitness attributable to natural selection acting against that specific process.

Mutation is expected to increase the genetic variance in fitness and, because most mutations are deleterious, to thereby decrease population mean fitness (Lynch & Gabriel 1990, Burt 1995). One way to conceptualize this mutation load is a decrease in population mean fitness without a change in the distribution of a focal phenotype or in the location of the phenotypic optimum (**Figure 1**, ❷). Mutation load is typically quantified by establishing experimental mutation accumulation (MA) lines in benign conditions that either minimize mortality (i.e., minimal natural selection) or equalize individual contributions to reproduction (i.e., minimal sexual selection). These methods allow nonlethal deleterious mutations to accumulate across generations. Meanwhile, control lines are maintained under "normal" conditions in which natural and sexual selection continue to operate or, when possible, the actual ancestors are maintained under dormant conditions. The difference in mean fitness between MA and control (or ancestor) lines, divided by the number of experimental generations, thus yields an estimate of the per-generation rate at which natural selection must be increasing fitness by weeding out deleterious mutations under the normal conditions (Burt 1995). Of course, complications arise if beneficial mutations occur, if some deleterious mutations are lethal, or if the "normal" environment used for quantifying fitness is not realistic. Indeed, these multigeneration experiments are most easily (and therefore commonly) conducted in the laboratory, but realism can be increased by testing fitness in nature (Rutter et al. 2010, Roles et al. 2016) or, to some extent, by testing fitness under "stressful" conditions in the laboratory.

Gene flow is expected to decrease fitness, at least in the short term, when a population adapted to a given environment receives immigrant alleles from populations adapted to other environments (Kirkpatrick & Barton 1997a, Lenormand 2002, Garant et al. 2007). The resulting migration load can be conceptualized as a shift in the mean phenotype away from the local optimum, along with an increase in the variance in phenotypes and therefore in fitness (**Figure 1**, ❸). Migration load could be quantified in several ways. First, an investigator could experimentally, but realistically, increase gene flow among populations adapted to different environments and then quantify fitness changes in the recipient populations. For instance, Fitzpatrick et al. (2016) introduced guppies from one environment into a previously guppy-free site just upstream of guppy populations in another environment. The effect on the downstream population was an apparent increase in fitness due to gene flow, which highlights a complication of this approach. Specifically, gene flow can have positive effects on fitness, such as by reducing inbreeding or facilitating rapid adaptive responses to environmental change (reviewed in Garant et al. 2007). Second, an investigator could experimentally decrease gene flow between populations and then measure the rate at which fitness changes. Along these lines, several studies of wild populations have shown how a decrease in gene flow can be followed by rapid trait adaptation (Riechert 1993, Nosil 2009), and the important next step would be to quantify evolutionary changes in fitness. Third, Burt (1995) suggested that migration load could be used to estimate the evolution of fitness by (in essence) multiplying the proportion of immigrants by their fitness relative to residents. A problem here, however, is that immigrants can have little relevance to local population fitness if they do not interbreed with residents owing to low survival or reproductive success of the former (Hendry 2004, Nosil et al. 2005, Hendry 2017). Thus, investigators instead need to estimate the fitness effects of immigrant alleles into the resident gene pool. In one such approach, pedigree data that quantify

the introgression of alleles into a recipient population can be used to estimate the difference in the additive genetic value for fitness between immigrants and existing residents and hence infer the degree to which natural immigration affects the rate of evolution of fitness (Wolak & Reid 2016, 2017; Wolak et al. 2018).

genetic rescue: increased population mean fitness due to an influx of new alleles that offsets inbreeding depression in small populations

evolvability: the potential of a population to evolve in response to selection, with a formal definition being that of Houle (1992)

Genetic drift is expected to cause random deviations of mean phenotypes from the phenotypic optimum, especially when populations are very small or selection is very weak (Barton & Partridge 2000). This drift load can be conceptualized as a set of populations whose phenotypic distributions deviate to varying degrees and directions from the optimum (**Figure 1**, ❹). These phenotypic distributions also should become narrower given that drift reduces within-population variance. One way to leverage these effects into an estimate of the rate at which fitness can evolve would be to generate (or find a population that has experienced) a short-term population bottleneck and to then monitor the fitness change when the bottleneck is removed. Another effect of small population size can be an increase in inbreeding, which can have negative effects on fitness through the increased expression of recessive deleterious mutations and the decreased expression of overdominance (Reed & Frankham 2003, Charlesworth & Willis 2009). Inbreeding is certainly known to decrease fitness in nature (Ellstrand & Elam 1993, Keller & Waller 2002); accordingly, the immigration of unrelated individuals can cause genetic rescue through rapid increases in fitness (Frankham 2015). However, as noted earlier, it is hard to discern what component of this increase is due to natural selection versus the immediate benefits of hiding recessive deleterious mutations in a heterozygous state.

Genetic recombination and segregation within a population can decrease, in the short term, population mean fitness by disassociating alleles at different loci that work well together through positive epistatic interactions (Charlesworth & Barton 1996, Barton & Partridge 2000, Otto & Lenormand 2002, Becks & Agrawal 2011) or by simply decreasing the "precision" of adaptation (Hansen et al. 2006) (**Figure 1**, ❺). This recombination/segregation load can be assessed by manipulating sexual reproduction (Becks & Agrawal 2011) or by studying genomic regions of reduced recombination (e.g., Santos 2009), although various caveats attend these methods (Charlesworth & Barton 1996, Barton & Partridge 2000, Otto & Lenormand 2002). Also, any immediate short-term fitness cost of recombination or segregation can be more than offset through the resulting increase in genetic variation that can facilitate future adaptation. Indeed, natural selection generally favors recombination within populations, at least in variable environments (Otto & Lenormand 2002). Hence, any estimate of the immediate fitness costs of recombination or segregation needs to be separated from the longer-term benefits.

Additive Genetic Variance in Fitness

The above approaches each focus on a specific process (e.g., environmental change or mutation or gene flow) that putatively decreases fitness, which selection must then counteract. Those approaches thus generate inferences about the rate of evolution of fitness in relation to that specific process. However, multiple processes decreasing fitness could occur simultaneously in the wild. It is therefore important to estimate the overall rate of evolution of fitness considering all such effects at the same time.

The basic approach here is to follow Fisher's (1930, 1958) insight that the rate of increase of population mean fitness due to natural selection is proportional to the mean-standardized additive genetic variance in fitness (Burt 1995, Ewens 2004, Shaw & Shaw 2014). This evolvability was derived by Houle (1992) as $I_{Aw} = V_{Aw}/W^2$, where V_{Aw} is the additive genetic variance in absolute fitness and W is mean fitness. I_{Aw} consequently equals the square of the coefficient of additive genetic variance in fitness (i.e., CV_{Aw}^2). In general, I_{Aw} can be interpreted as the expected proportional change in population mean fitness given a unit strength of selection (Hansen

et al. 2011). It constitutes a metric that, unlike heritability (h^2), is not directly influenced by the magnitude of environmental variance, and it thus facilitates comparison of evolvability across populations and species (Houle 1992, Hansen et al. 2011). Of particular utility, I_{Aw} can be estimated in wild populations, although implementation is far from easy. The primary challenge is to obtain an unbiased sample of fitness estimates for enough individuals of known and varying relatedness that can be used to estimate V_{Aw} while distinguishing common environmental or intergenerational parental effects (Shaw & Shaw 2014), particularly maternal effects (Kruuk & Hadfield 2007). Another major challenge arises because fitness results from a temporal sequence of episodes of survival and reproductive success. This combination generates a phenotypic distribution that is typically non-Gaussian and hence challenging to accommodate in quantitative genetic analyses while retaining the utility of I_{Aw} as an appropriate standardized comparative metric (Hansen et al. 2011, Gomulkiewicz & Shaw 2013, Shaw & Shaw 2014, Wolak et al. 2018).

animal model: linear mixed effects model that includes a correlation structure based on additive genetic relatedness among individuals

Burt (1995) reported 12 estimates of I_{Aw} from 7 studies; however, most of those estimates were less than ideal because they were limited to fitness components, were based on estimates of LRS that were not zygote-to-zygote, or used parent–offspring regressions and hence excluded individuals that produced zero offspring (i.e., had zero fitness). Fortunately, recent years have seen several major developments that greatly facilitate appropriate estimation of I_{Aw} in wild populations. First, multiple field studies of wild vertebrate populations have reached sufficient duration to measure LRS in numerous individuals (Ellegren & Sheldon 2008, Clutton-Brock & Sheldon 2010). Second, such studies can increasingly, with the aid of detailed pedigrees and genomic information, measure relatedness among focal individuals (Bérénos et al. 2014, Postma 2014). Third, a generalized linear mixed model statistical approach, commonly known as the "animal model," facilitates estimating V_{Aw} while accounting for selection and resulting unbalanced family structures and unobservable phenotypes (Kruuk 2004, Charmantier et al. 2014). This accounting must be done because such effects are inevitable consequences of variation in fitness, and they can severely bias more traditional approaches to estimating V_{Aw} (Lynch & Walsh 1998, Hadfield 2008).

An additional dimension to the problem of estimating genetic variation in fitness can arise in species with separate sexes—that is, dioecious plants and gonochoristic animals. The reason is that the genetic basis of fitness can differ substantially between females and males, which can generate intralocus sexual conflict (cross-sex genetic correlation for fitness of less than one) and shape the overall magnitude of genetic variance in fitness and associated responses to selection (Brommer et al. 2007, Kruuk et al. 2008, Bonduriansky & Chenoweth 2009, Kirkpatrick 2009). Consequently, studies aimed at understanding the rate of evolution of fitness should estimate V_{Aw} in both sexes as well as the cross-sex additive genetic covariance (COV_A) and hence correlation (r_A) (Chippindale et al. 2001, Brommer et al. 2007, Kruuk et al. 2008, Kirkpatrick 2009). This ambition poses further data collection and analytical challenges for field studies because male fitness can be particularly hard to measure accurately and because cross-sex genetic correlations are notoriously difficult to estimate precisely (Shaw 1987, Poissant et al. 2010, Kruuk et al. 2014).

DATA ON THE EVOLUTION OF FITNESS

Two methods for quantifying the rate of evolution of fitness have been implemented in enough studies to warrant quantitative review: mutation accumulation and additive genetic variance in fitness. Even here, the available data are still too heterogeneous to warrant formal meta-analysis, yet a quantitative review can generate insight into general patterns, thereby updating the similar review of Burt (1995). Before commencing, we must note that our abiding interest in contemporary wild (or "natural") populations necessitates some caveats. For MA, fitness is rarely measured in nature, and assays are commonly restricted to fitness components rather than fitness itself. For

I_{Aw}, even studies of wild populations often involve somewhat unnatural conditions (e.g., use of nest boxes for birds or equal spacing in fields for plants) and, despite recent methodological advances, estimates still might be biased. Yet, despite these caveats, such data are still the best information currently available regarding the rate of evolution of fitness. Also, it is important to bear in mind that the two methods estimate different quantities because new deleterious mutations (MA) are only one factor contributing to variation in fitness (I_{Aw}).

Mutation Accumulation

We reviewed published MA studies to obtain data on the percent decrease in mean fitness (or major fitness component) per generation for eukaryotic organisms. The studies eliminated selection on all but the most deleterious (e.g., lethal and semilethal) mutations by variously using balancer chromosomes, extreme population bottlenecking (e.g., selfing, full-sib mating, or equalization of reproductive output), or curtailment of life span (to assay the effect of late-acting mutations; Bryant & Reed 1999). MA studies were generally conducted with many lines, and fitness assays were carried out under laboratory conditions ranging from benign to stressful or, for several plant studies, in natural habitats. In each study, MA line fitness was compared to a control set of lines or to the ancestor of the MA lines.

Given our interest in conditions that are as close to natural as possible, we excluded studies that used mutator lines (e.g., Heilbron et al. 2014) or lines constructed to carry more mutations than expected under rates of natural genomic degeneration (e.g., Sharp & Agrawal 2012). Also, to maximize precision and accuracy, we excluded studies based on few (<10) MA lines as well as studies that singled out a nonrandom subset of MA lines. We also excluded studies in which changes in mean fitness were not reported or were not discernable from the information provided. For all reported values, fitness change estimates were scaled to the whole genome level when the experimental design affected only a subset of the chromosomes (e.g., balancers in *Drosophila*).

A total of 51 estimates met our criteria, 39 of which came from 3 species and their relatives: *Caenorhabditis elegans*, *Drosophila melanogaster*, and *Arabidopsis thaliana* (**Supplemental Table 1**). The remaining estimates were from higher plants (5 studies on *Amsinckia* spp. and *Raphanus raphanistrum*) or single-celled eukaryotes (7 studies). As expected, most estimates indicated decreases in fitness in MA lines, with the rate of decrease varying substantially among organisms (**Figure 2**). Fitness decreases were largest for *D. melanogaster* and higher plants and smallest for *C. elegans*. Overall, the rate of decrease in fitness per generation due to MA (and therefore, the amount by which selection can be expected to increase fitness) is no larger than approximately 2%, with most studies (particularly those of *C. elegans* and simpler eukaryotes) falling closer to 0.1–0.5%, a value near the lower end of the spectrum of the estimates summarized by Burt (1995).

Several aspects of the included studies imply that they are unlikely to accurately reflect the rate of evolution of fitness in wild populations. First, lethal mutations are typically not assayed in MA studies. Second, only three of the estimates were obtained under field conditions, and only another eight under nonbenign "stressful" conditions (e.g., competitive stress, temperature stress, or mineral starvation). Third, in several *A. thaliana* studies, MA led to fitness increases rather than decreases, suggesting that not all mutations in the experiments were deleterious, although another possibility is genotype-by-environment interactions in which rapid cycling ecotypes bred in the laboratory or greenhouse are grown under new conditions. These biases compound to suggest that the decreases in fitness documented in MA studies are likely to underestimate the fitness consequences of deleterious mutations in nature. And, of course, mutation remains only one factor influencing fitness variation, dictating that changes detected in MA studies will be less than the expected rate of evolution of fitness in the wild.

Supplemental Material >

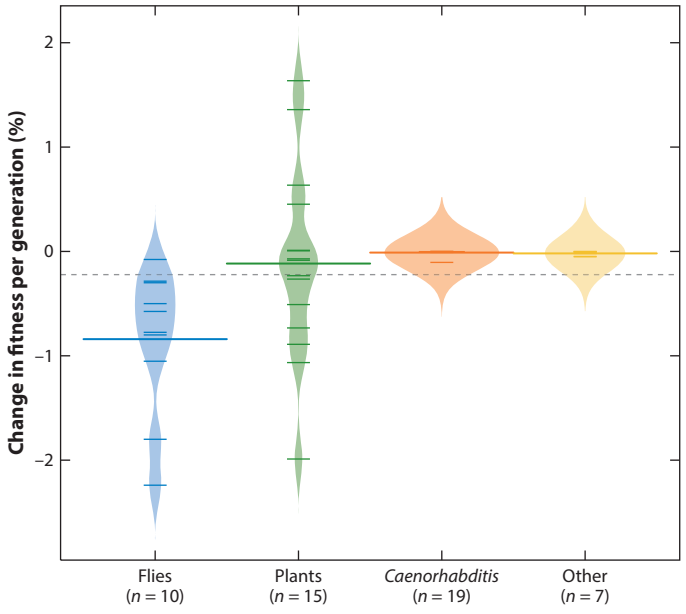

Figure 2

Percent change in mean absolute fitness (or fitness components) per generation as estimated from mutation accumulation experiments. Data are grouped into the following categories: flies (*Drosophila* and related genera), plants, *Caenorhabditis* (*Caenorhabditis elegans* and related taxa), and other (yeast, protozoans, and single-celled algae). Sample sizes indicate numbers of studies conducted with each category of organism. Long lines indicate medians, shorter lines indicate individual estimates, and shapes (*violins*) are traces of the kernel density estimates.

Additive Genetic Variance in Fitness

We searched the literature for studies estimating additive genetic variance in some measure of fitness in wild animal or plant populations. We retained studies that measured LRS as the total number of new offspring born or hatched, or seeds produced, by each new offspring, which represents the closest metric of zygote-to-zygote fitness achieved in studies of wild populations. To provide a comprehensive overview of available data, we also retained studies that measured LRS as total adult or recruited offspring as well as the few studies that considered relative fitness or metrics that account for reproductive scheduling and age structure. From each retained study, we extracted estimates of I_{Aw} and/or V_{Aw} (or h^2_w or CV_{Aw}), along with standard errors or 95% credible intervals (e.g., from Bayesian Markov Chain Monte Carlo estimation). We also extracted any further variance component estimates and statistics (e.g., phenotypic mean and variance) describing the distribution of fitness as well as any information on data transformations and key analytical methods. When neither I_{Aw} nor V_{Aw} were reported in the original article, we used other reported information to calculate them when possible. Full explanations of the search and exclusion criteria and data calculations and interpretations are available in the online **Supplemental Materials**.

Supplemental Material >

A total of 30 studies met our criteria: 25 on animals (including 8 on humans) and 5 on plants. A total of 82 estimates of I_{Aw} were extracted from 22 studies (**Supplemental Table 2**). These estimates ranged from 0 to 0.85 (**Figure 3**). However, 73 (89%) estimates were <0.2, and 24 (29%) estimates were reported as zero or negative (**Figure 3**). Thus, overall, the available evidence supports Burt's (1995) tentative conclusion that I_{Aw} is usually less than 0.3—or perhaps this conclusion now can be tightened to usually less than 0.2. No marked differences in I_{Aw} were

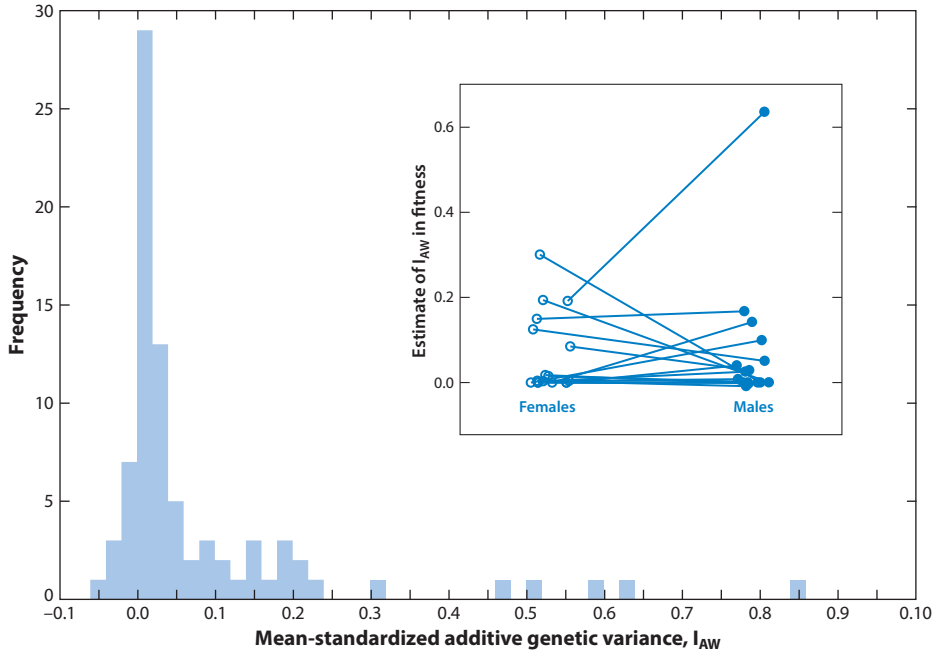

Figure 3

Distribution of estimates of the mean-standardized additive genetic variance in fitness (I_{Aw}) from free-living populations (*main histogram*), and comparison between paired estimates of I_{Aw} in females (*open circles*) and males (*filled circles*) in the same measure of fitness from the same study (*inset panel*; *lines join paired sex-specific estimates*). Some estimates of I_{Aw} derived from parent–offspring regressions are slightly negative. Across all 82 estimates, mean I_{Aw} was 0.08 $\pm$ 0.16 SD (median 0.02, IQR 0.00–0.08). Across 17 sets of paired sex-specific estimates, mean I_{Aw} was 0.06 $\pm$ 0.09 SD (median 0.01, IQR 0.00–0.12) for females and 0.07 $\pm$ 0.16 SD (median 0.01, IQR 0.00–0.05) for males and consequently did not differ significantly between the sexes ($t_{16} = -0.16$, $p = 0.88$). Abbreviations: IQR, interquartile range; SD, standard deviation.

Supplemental Material >

evident between plants, nonhuman animals, and humans—or between studies that measured LRS within or across generations (**Supplemental Materials**). Definitive conclusions on whether I_{Aw} differs among these and any other categories of interest will require additional data and carefully standardized comparative studies.

A total of 14 studies (all on animals) explicitly estimated V_{Aw} in some measure of male fitness alongside female fitness. A total of 17 female–male pairs of estimates of I_{Aw} were extracted from 11 of these studies. Across these pairs, no obvious overall difference in I_{Aw} was evident between the sexes (**Figure 3**). Because several studies estimated V_{Aw} in one or both sexes to be zero, only 5 studies attempted to estimate the cross-sex r_A, returning 6 estimates. These estimates varied substantially, from very negative to very positive, including within the same study, and they are all very imprecise (**Supplemental Table 3**).

Although the quantitative genetic analysis of fitness in wild populations has seen a revolution over the last 20 years, Burt's (1995) listing of caveats regarding quantitative interpretation still certainly applies. For instance, some values of $I_{Aw} = 0$ might simply represent bounded estimates stemming from low statistical power, whereas some high values of I_{Aw} might be inflated by common environmental effects on relatives, or further biased by transgenerational parental effects (e.g., Kruuk & Hadfield 2007). Sex-specific biases in I_{Aw} also might exist owing to, for example, more

error in paternity than maternity, more (or nonrandom) missing fitness records in one sex or the other, sex-biased dispersal, or sex-specific pedigree structure (**Supplemental Materials**). Finally, it is notable that some recent state-of-the-art studies estimate V_{Aw} using generalized linear mixed models with non-Gaussian error distributions, thereby providing appropriate latent-scale V_{Aw} estimates (e.g., Milot et al. 2011, McFarlane et al. 2014, Wolak et al. 2018). Such approaches as yet preclude direct estimation of I_{Aw} and associated evolutionary interpretation in the absence of appropriate back-transformation onto the phenotypic scale.

evolutionary rescue: increased population mean fitness due to adaptive genetic change in populations that would otherwise have gone extinct

Supplemental Material >

CONCLUSIONS AND PROSPECTS

The rate at which fitness can evolve in response to selection in contemporary wild populations is a fundamentally important parameter that underpins our understanding of how much environmental change and genetic degradation populations can withstand. This parameter determines the potential for evolution to facilitate population increases (such as in range expansions), prevent population declines (sometimes termed "cryptic" eco-evolutionary dynamics), and allow population recovery following deleterious environmental change (evolutionary rescue). Reviewing literature available at the time, Burt (1995) concluded that the amount by which natural selection increases mean fitness each generation (or degradation decreases mean fitness) will usually be between 0.1% and 30%; more tentatively, he suggested that values will typically fall between 1% and 10%. On the basis of the larger number of studies that have now estimated additive genetic variance in fitness in wild populations, often using vastly improved data sets and analytical methodologies, we can conclude that nearly all estimates are below 20% and that the majority are below 10%. By contrast, estimates from MA studies are approximately an order of magnitude lower, the majority being below 1%. This qualitative difference between estimation approaches makes sense given that mutation is only one of several processes that contribute to additive genetic variation in fitness. Hence, the different approaches are complementary: Additive genetic variance in fitness estimates the overall rate of fitness evolution, whereas process-specific methods, such as MA experiments, estimate how natural selection must counter that specific process.

Considerable debate has surrounded the question of how much variation in fitness, and therefore how much evolutionary potential, exists in wild populations (Shaw & Shaw 2014). Our empirical review highlights that some genetic variation in fitness does commonly exist in contemporary wild populations, which in some cases explains a nontrivial proportion of the phenotypic variance (i.e., generating moderate heritability; see also **Supplemental Table 2**). Hence, although nongenetic stochasticity certainly will be an important component of realized fitness, genetic influences will also shape expected fitness ("luck" and "pluck" sensu Snyder & Ellner 2018). These estimates could be useful for predicting evolutionary rescue by comparison with initial population declines following disturbance, which should reflect the rate at which environmental change decreases population mean fitness. Such estimates also might help to establish the time frames over which genetic change versus existing phenotypic plasticity will be most important for population persistence (Chevin et al. 2013, Kovach-Orr & Fussmann 2013). Further, from a genetic degradation perspective, estimates of the fitness costs of MA (or migration or drift) could be used to parameterize models seeking to optimize population sizes and gene flow rates for endangered, isolated, or declining populations.

The estimates reviewed here also inform other key areas of interest in evolutionary biology, such as the benefit of mate choice and the associated evolution of secondary sexual traits and mating systems. Classically, additive genetic variance in fitness is a key parameter that limits the rate at which mating preferences and behaviors, and associated traits, can evolve through so-called good genes effects (i.e., indirect selection) (Burt 1995, Kirkpatrick & Barton 1997b, Arnqvist & Kirkpatrick 2005). Our survey indicates that some scope for such evolution does often

exist. However, recent work emphasizes the importance of sex-specific additive genetic variances and cross-sex genetic correlations (Kirkpatrick & Hall 2004, Brommer et al. 2007). We found that studies estimating all three parameters are still remarkably scarce and, in particular, available estimates of the cross-sex genetic correlation are too imprecise to allow any broad inference (**Supplemental Table 3**; see also Kirkpatrick 2009). More estimates, with greater precision, are clearly needed, in turn requiring further major advances in data collection and in quantitative genetic analysis and interpretation.

Supplemental Material >

Although the general bounds for the rate of evolution of fitness that we identified seem likely to hold in future investigations, it is important to note substantial variation among taxa and experiments. As just one example, estimates of decreases in fitness due to mutation accumulation are much higher for flies than for plants or *Caenorhabditis* (**Figure 2**). To confirm the reality of potential taxon-specific rates, and to then explore the reasons for such variation, additional studies are needed, including studies of other taxa. Similarly, estimates of additive genetic variance in fitness in wild populations are mostly from humans, ungulates, passerine birds, and higher plants (**Supplemental Table 2**). Additional studies will also facilitate formal meta-analysis of rates of evolution of fitness and how they vary among taxa and methods. Beyond further expansion of existing databases on mutation accumulation and additive genetic variance in fitness, we have highlighted several alternative experimental approaches that could generate useful estimates of the evolution of fitness in nature, including experimental introductions, resurrection studies, and the experimental manipulation of gene flow. In addition, it would be useful to conduct studies that combine and compare approaches so as to infer different forces contributing to variance in fitness and its rate of evolution, such as mutational inputs and gene flow and other genetic or environmental changes.

Burt (1995) stimulated general interest in how rapidly fitness can evolve. Although his initial motivation was driven largely by an interest in the evolution of mate choice (A. Burt, personal communication), the implications for responses to contemporary environmental change and ongoing genetic degradation have become increasingly apparent and pertinent. Now, 23 years hence, we have enough data to support—and refine—Burt's (1995) conclusions while still identifying additional data needs and emerging questions. We hope that further major improvements and refinements rapidly become possible through accumulating new data using existing and new approaches and methods. The rate of evolution of fitness in contemporary wild populations is a critical parameter toward which our attention should be squarely directed.

DISCLOSURE STATEMENT

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Errata

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