Tipping points in organismal adaptation

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Evolutionary tipping points in the capacity to adapt to environmental change

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Edited by Stephen W. Pacala, Princeton University, Princeton, NJ, and approved October 24, 2014 (received for review May 9, 2014)

In an era of rapid climate change, there is a pressing need to understand how organisms will cope with faster and less predictable variation in environmental conditions. Here we develop a unifying model that predicts evolutionary responses to environmentally driven fluctuating selection and use this theoretical framework to explore the potential consequences of altered environmental cycles. We first show that the parameter space determined by different combinations of predictability and timescale of environmental variation is partitioned into distinct regions where a single mode of response (reversible phenotypic plasticity, irreversible phenotypic plasticity, bet-hedging, or adaptive tracking) has a clear selective advantage over all others. We then demonstrate that, although significant environmental changes within these regions can be accommodated by evolution, most changes that involve transitions between regions result in rapid population collapse and often extinction. Thus, the boundaries between response mode regions in our model correspond to evolutionary tipping points, where even minor changes in environmental parameters can have dramatic and disproportionate consequences on population viability. Finally, we discuss how different life histories and genetic architectures may influence the location of tipping points in parameter space and the likelihood of extinction during such transitions. These insights can help identify and address some of the cryptic threats to natural populations that are likely to result from any natural or human-induced change in environmental conditions. They also demonstrate the potential value of evolutionary thinking in the study of global climate change.

fluctuating selection | global change | phenotypic plasticity | bet-hedging | adaptive tracking

Understanding how organisms cope with and adapt to changes in their environments is a central theme in evolutionary ecology (1). However, we currently lack the tools to predict the most likely evolutionary responses to changes in environmental conditions (2), including those currently experienced through global change (3, 4). Evolutionary responses to within- and among-year fluctuation in ecological parameters like ambient temperature or precipitation can be highly informative about the process of adaptation to environmental change, as well as about the potential consequences of the recently accelerated rates of global change and the associated increase in climatic variability and unpredictability (5–8). Earlier work indicates that some organisms face environmental uncertainty by hedging their bets with a strategy that minimizes fitness variance across all possible environmental conditions (conservative bet-hedging) (9), whereas others have evolved a mix of strategies to take advantage of alternative environmental scenarios in a probabilistic fashion (diversification bet-hedging) (9). In still other cases, organisms cope with environmental variation through phenotypic plasticity, which is the ability to respond to environmental cues through the adjustment of genotypic expression either during early development (irreversible or developmental plasticity) (10) or throughout life (reversible plasticity) (11). Finally, environmental variation is also known to result in correlated variation in mean population traits, as natural selection favors different phenotypes over evolutionary time (adaptive tracking) (12). Although an increasing amount of attention has been recently devoted to the conditions that promote these different forms of evolutionary response to environmental variation (hereafter “response modes”) (2, 9, 13–18), most studies have considered only one or a small subset of response modes (16, 17), and few have explored the general conditions under which one (or more) may be selected above the others (2, 18). Addressing these issues will be critical for improving our ability to predict whether and how populations will adapt to both natural and human-induced environmental change.

Here we develop a theoretical model that considers the joint evolution of a comprehensive range of evolutionary responses to environmental variation. Although we illustrate our model by exploring the effects of temperature, the principles we describe apply to other naturally fluctuating environmental variables (e.g., precipitation). We use the term insulation, I, as a broad descriptor of morphological (e.g., coat thickness) (19), behavioral (e.g., huddling), or physiological (e.g., sweating) characteristics that help counter thermal stress. To investigate the dynamics of adaptation to environmental variation, we use individual-based evolutionary simulations in which the pattern of variation in genotypic expression across a range of environmental conditions (i.e., the reaction norm of the genotype) (14) is assumed to be heritable and subject to mutation and natural selection. We begin by testing the consistency of evolutionary response to different types of environmental change and then use this general framework to explore how systems react to disruption in the nature of environmental oscillations. A nontechnical description

Significance

Environmental variation is becoming more frequent and unpredictable as a consequence of climate change, yet we currently lack the tools to evaluate the extent to which organisms may adapt to this phenomenon. Here we develop a model that explores these issues and use it to study how changes in the timescale and predictability of environmental variation may ultimately affect population viability. Our model indicates that, although populations can often cope with fairly large changes in these environmental parameters, on occasion they will collapse abruptly and go extinct. We characterize the conditions under which these evolutionary tipping points occur and discuss how vulnerability to such cryptic threats may depend on the genetic architecture and life history of the organisms involved.

Author contributions: C.A.B., F.J.W., J.W., and D.R.R. designed research; C.A.B. performed research; C.A.B. analyzed data; C.A.B., F.J.W., J.W., and D.R.R. wrote the paper.

The authors declare no conflict of interest.

This article is a PNAS Direct Submission. Freely available online through the PNAS open access option.

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This article contains supporting information online at www.pnas.org/lookup/suppl/doi:10.1073/pnas.1408589111/-/DCSupplemental.
of how our model can inform issues related to global change is included in the SI Text.

Results

Environmental variation includes both deterministic (i.e., climate) and stochastic (i.e., weather) components. For example, temperatures oscillate deterministically from cold winters to hot summers, but the actual values experienced in a given day vary stochastically from the seasonally expected average. We modeled these components as

\[ E_t = A \cdot \sin(2\pi t/LR) + B \cdot e \]

where \( t \) is time, \( L \) is the number of time steps per generation (i.e., lifespan), \( R \) is the relative timescale of environmental variation (i.e., number of generations per environmental cycle), \( e \) is a stochastic error term, and \( A \) and \( B \) are scaling constants reflecting the relative importance of deterministic and stochastic factors. This equation describes a simple sinusoidal oscillation in environmental conditions when \( R \) is intermediate or small and approximates a slow directional change when \( R \) is very large. Because \( R \) is a relative metric, the findings presented below are easily applicable to organisms with different lifespans.

In nature, changes in environmental conditions are often preceded by correlated changes in photoperiod, barometric pressure, or other environmental cues. For example, day length variation tends to be well correlated with seasonal temperature variation in temperate regions. Thus, we model the predictability of environmental conditions, \( P \), by altering the degree to which an environmental cue, \( C \), is correlated with future temperature values (SI Text and Fig. S1). When temperatures and cues are perfectly correlated, the environment is completely predictable, \( P = 1 \), and when they are not correlated at all, it is completely unpredictable, \( P = 0 \). In the simulations presented here, cues are provided to individuals before experiencing any changes in their environment (Methods).

Simulation runs in our model proceed in discrete time steps with nonoverlapping generations and individual lifespans of \( L = 5 \) time steps. Individuals possess seven genetic traits—loci \( h, s, a \), \( I_0, I_0', b \), and \( b' \)—that determine the amount of insulation to be produced under different environmental cues. Every genotype specifies two different reaction norms: one encoded by \( I_0 \) and \( b \), and another one encoded by \( I_0' \) and \( b' \). Loci \( I_0 \) and \( I_0' \) determine baseline degrees of insulation, whereas \( loci \) \( b \) and \( b' \) determine the degree to which insulation is made dependent on environmental cues. Each individual in our model expresses only one of these reaction norms through life: the one based on \( I_0 \) and \( b \) is chosen at birth with probability \( h \), whereas the one based on \( I_0' \) and \( b' \) is chosen with probability \( 1 - h \). In practice, this implies that locus \( h \) enables individuals with the same genotype to respond to environmental variation in two completely different ways (as in diversifying bet-hedging). Locus \( s \) is a genetic switch that determines whether the organism makes its insulation dependent on environmental cues (i.e., whether it allows for phenotypic plasticity; \( s > 0.5 \)) or not (\( s \leq 0.5 \)). Nonplastic individuals ignore environmental cues and exhibit a fixed insulation phenotype encoded by the baseline loci \( I_0 \) or \( I_0' \). Plastic individuals adjust their insulation phenotypes, \( I \), to the environmental cues they perceive using linear norms of reaction such that \( I = I_0 + b \cdot C \) or \( I = I_0' + b' \cdot C \). Locus \( a \) determines whether this cue dependence is only happening during ontogeny (irreversible or developmental plasticity) or also throughout the individual’s lifetime (reversible phenotypic plasticity). In practice, this means that individuals with \( a = 0 \) respond to environmental cues only during development—and therefore exhibit a single phenotype throughout life—whereas those with \( a > 0 \) alter their phenotypes with probability \( a \) at each time step after development. As in earlier studies (14), we assume that phenotypic plasticity is costly both during and after development. Thus, plastic individuals pay a one-time developmental cost, \( k_p \), and each phenotypic adjustment after development is assumed to incur in an additional cost of \( k_p \).

To establish a baseline for comparison, we began by evaluating the effects of environments with a constant temperature. As expected, this simple scenario led to the evolution of nonplastic insulation strategies with a mean population value that approximately matched the temperature experienced. We then considered completely stochastic environments (\( A = 0 \) and \( B = 1 \)), where individuals had no information about the potential state of the environment (\( P = 0 \)). Under these conditions, populations evolved to ignore uninformative cues, producing instead a fixed phenotype at the average environmental condition (\( I = 0 \); Fig. L4). In contrast, when we allowed these same stochastic environments to be completely predictable (\( P = 1 \)), the resulting reaction norms led to insulation levels that varied with the intensity of environmental cues (Fig. 1B). In completely deterministic environments (\( A = 1 \) and \( B = 0 \)) with rapid environmental variation (\( \log R = 0 \)), we observed that phenotypic plasticity also evolved only when individuals were able to anticipate environmental changes (Fig. 1 C and D). This result highlights a key aspect of adaptation to environmental change: the way in which environments vary (i.e., whether the pattern of environmental oscillations appears to be stochastic or deterministic) is less important to evolution than the degree to which individuals can anticipate the future state of the environment (20). Thus, the remaining simulations focus on the effects of predictability of environmental variation and assume, for simplicity, that \( A = 1 \) and \( B = 0 \) (SI Text).

We proceeded to explore evolutionary outcomes at different predictability levels and across a comprehensive range of timescales of variation (Fig. 2). For each set of conditions, we performed 100 replicated simulations. Each subplot in Fig. 2A depicts the 100 evolved mean reaction norms at generation 50,000 (e.g., \( I = \bar{b} \cdot C + \bar{b}_0 \), where \( \bar{b} \) and \( \bar{b}_0 \) correspond to the mean population values for \( b \) and \( b_0 \)). Overall, we find that evolution results in remarkably consistent outcomes for the majority of parameter combinations (Fig. 2A, SI Text, and Fig. S2) and that different response modes occur largely in nonoverlapping regions of parameter space (Fig. 2B, Table S1, SI Text, and Fig. S3). These findings are robust to the implementation of density-dependent selection, as well as to alternative coding schemes for genotype-to-phenotype mapping (SI Text and Fig. S4). In cases where environmental variation within a generation is both predictable and fast (\( P \) is large, \( R \) is small; upper left corner of Fig. 2B), each subplot in Fig. 2A shows a single cluster of reaction norms. This indicates that (i) similar reaction norms evolved in all 100 replicate simulations at that parameter combination, (ii) the evolved populations exhibit a high degree of plasticity (i.e., \( s > 0.5 \) and \( b \approx 1 \)), and (iii) individuals in these populations often adjust their phenotypes after development (\( b \approx 1 \); Table S1). As \( R \) becomes larger, locus \( a \) quickly evolves to \( a = 0 \) (depicted in blue in Fig. 2A) because the diminishing benefits of avoiding thermal mismatches no longer surpass the costs of phenotypic adjustment (13, 21, 22). We label this strategy irreversible plasticity because individuals in these populations exhibit plasticity exclusively during development. The transition from reversible to irreversible plasticity occurs at progressively shorter timescales in less predictable environments because the expected benefits of phenotypic adjustment decrease with higher potential for errors in anticipating environmental change.

When environmental conditions are fairly unpredictable, the rate at which environments change determines the resulting evolutionary outcome. If \( R \) is large (lower right corner of Fig. 2B), the slow rate of environmental change allows for beneficial mutations in \( I_0 \) to appear and approach fixation. The resulting pattern is a gradual change of the mean phenotype that tends to lag behind
the change in environmental conditions (adaptive tracking in Fig. S5A). However, at faster timescales (lower center and lower left in Fig. 2B), environmental change is too fast to be tracked by mutation and too unpredictable to be addressed through plasticity. Consistent with previous studies (9, 16), this extreme form of uncertainty forces individuals to hedge their bets. When individuals experience all possible conditions with similar probability (e.g., very low $R$), we observe the evolution of fixed phenotypes at $I \sim 0$. Although this insulation value rarely matches the actual conditions experienced, it matches the average environment and therefore minimizes overall thermal mismatch across the entire range of potential environmental conditions (Table S1). Thus, this strategy resembles conservative bet-hedging (9) in that it minimizes the variance in fitness among selection events and across individuals that share the same genotype. In contrast, when individuals of a given genotype experience only a fraction of the environmental cycle (e.g., $\log R = 0.5$), we observe the evolution of mixed strategies that produce alternative phenotypes with either heavy or light insulation in a probabilistic fashion (green in Fig. 2A). This strategy resembles the phenotypic polymorphism of diversification bet-hedging (9) (Table S1), because the different phenotypes produced by a single genotype minimize thermal mismatch in different scenarios (i.e., the larger $I$ phenotype does best when experiencing disproportionally more of the upper than the lower half of the environmental cycle, and the smaller $I$ phenotype does best in the opposite situation).

Having determined the most likely evolutionary outcomes under a comprehensive range of parameter combinations, we proceeded to explore how populations are affected by changes in the predictability or timescale of environmental variation (i.e., in the signature of their environment). The well-defined response mode regions observed in Fig. 2 allowed us to make a simple but important a priori prediction: changes in environmental signatures that require the evolution of an entirely different mode of response may be harder to cope with than those that do not. To test this hypothesis, we abandoned the assumption of a constant population size in our model and linked reproductive output to absolute rather than relative fitness (Methods). By relaxing this assumption, we were able to assess the demographic consequences (e.g., changes in population size and risk of extinction) of different environmental challenges. In this eco-evolutionary version of our model, maximal fecundity, $q$, was defined as the average number of offspring that an individual produces when it pays no plasticity costs and is able to exactly match its environment at every time step of its life. Thus, the mean fecundity of individual $i$, $\bar{F}_i$, is determined by the fraction of the maximum payoff that it is able to achieve, such that $\bar{F}_i = q W_i/W_{\text{max}}$ (Methods). Fig. 3 depicts the potential for extinction at each parameter combination (inner squares), as well as during transitions between adjacent combinations in parameter space when $q = 2.2$ (see Fig. S6 for alternative values of $q$). Each of the four possible transitions to an adjacent cell is depicted using trapezoids. For example, the color of the upper trapezoid within a given subplot indicates the effects of transitioning from that particular parameter combination to the one above it. As predicted, we found that the potential for extinction during these transitions is considerably higher when populations are forced into a different response mode region (a result that holds even if much larger changes in $P$ or $R$ are attempted).

The nonuniformity of transitional extinction rates in our model is driven by at least two different mechanisms. First, some transitions imply moving into regions of parameter space that are particularly challenging for adaptation. For example, when environmental oscillations are quick and unpredictable (i.e., the bet-hedging region), baseline levels of extinction are high, particularly at lower $q$ values (Fig. S6). Thus, any population that is suddenly forced into this region will also be expected to have a high likelihood of extinction (Fig. 3A). The second contributor to extinction relates to the complexity of genetic changes required for adaptation during transition and is more readily observable after accounting for potential differences in baseline levels of extinction in the new environments. For example, when relative extinction rates are considered (Fig. 3B), we find that extinction is only more likely than expected when populations move into a different response mode region (even if this transition involves moving into regions of parameter space that appear to be easier for adaptation, such as into more predictable environments). The reason for the increased risk of extinction during these tipping point transitions is that adapting to a completely new strategy for phenotypic development often requires a radical restructuring of the genome, which can be particularly difficult to achieve as populations collapse. For example, in the transition from phenotypic plasticity to bet-hedging, plastic strategies...
maladapted (i.e., their expected number of offspring, \( W \), is less than 1) and population decline is swift (Fig. 4). Thus, given that adaptation to the new environment requires in this case resetting developmental switches (\( s \) and \( a \)) and adjusting almost every other locus in the virtual genome, the stochastic nature of mutation supply and the reduced standing genetic variation of declining populations are more likely to result in extinction (Fig. 4A) than in evolutionary rescue (Fig. 4B). Conversely, the relative extinction rates for the reverse transition are also high because the fitness of fixed strategies is low compared with that of plastic ones, and because many of the mutations that can potentially transform a fixed strategy into a plastic one will, in the absence of other necessary genetic changes, result in maladapted phenotypes. Another case with high relative rates of extinction during tipping point transitions is the change from conservative to diversifying bet-hedging, which involves similarly extensive genetic changes, including the resetting of \( h_i, I_0, y_0, b \), and \( b' \). In contrast, when genomic changes are relatively simple, as in the case of the transition between reversible and irreversible plasticity, the likelihood of adaptation during transition is much higher (Fig. 3).

**Discussion**

Our model suggests that evolutionary response to environmental variation may be more predictable than previously anticipated. Through evolutionary simulations, we showed that fundamentally different adaptive responses consistently evolve under different timescales and predictabilities of environmental variation.
In addition, our model indicates that the potential for extinction during tipping point transitions depends critically on the genetic architecture of relevant traits (37) and in particular on the number or magnitude of mutations required to achieve the genotypic optimum for the new selection regime. For example, we expect that populations will be more likely to go extinct when the strategy that needs to be evolved requires either de novo evolution (or loss) of complex organs and structures or a major readjustment of basic physiological/developmental pathways. Conversely, we expect lower vulnerability to extinction when the desired new strategy after transition is achievable through the evolution of simple genetic changes that do not interfere with major body plans.

In conclusion, our model provides a unifying theoretical framework for predicting evolutionary responses to environmental change and leads to a series of testable predictions regarding organismal capacity to adapt to natural or human induced changes in the environment. These predictions can be tested through experimental evolution of microorganisms or through comparative analyses of populations or species distributed along a gradient of environmental variation. Ultimately, evolutionary models like the one we present here can aid in determining the specific type of adaptation that organisms may use to cope with specific environmental changes, thereby improving our understanding of how populations and species may respond to either global change or other environmental challenges.

Methods

Norms of Reaction. The tendency of a genotype to be systematically expressed as different phenotypes across a range of environmental conditions is known as the genotype’s norm of reaction. Our model assumes that the effects of heat and cold stress are symmetric and that selection favors phenotypes that match the environmental condition in which they are expressed (see below). These simplifying assumptions imply that if individuals have perfect information about the environment, then they can maximize their returns with \( I = E \). Accordingly, we have parameterized reaction norms in our model as linear functions. Thus, \( I = I_0 + b \cdot C \) (or \( I = I_0 + b \cdot C \cdot W \) with probability \( 1 - h \)), where \( I_0 \) is the insulation level produced at \( C = 0 \), and \( b \) is a slope that determines the degree to which insulation levels change as a function of changes in environmental cues (for alternative genotype-phenotype mapping schemes, see SI Text and Fig. S3).

Fitness. Every individual in our model lives for \( L = 5 \) time steps. Each time step proceeds in a defined order. First, environmental conditions are updated and environmental cues are computed from \( E_t \) and \( P \) as described above.

\[ M_{t+1} = \sum_{i=0}^{n} M_{t,i} \]

where \( F_t \) is the current environmental state and \( l_{t,i} \) is the individual’s current phenotype. At the end of a generation, a nonplastic individual’s lifetime payoff, \( W_t \), is computed as a function of the sum total of phenotypic mismatches throughout life, such that

\[ W_t = \exp\left(-\tau \cdot \sum_{i=0}^{n} M_{t,i}\right), \]

where \( \tau \) is a constant that determines the strength of fitness decay as a function of total phenotypic mismatch. For plastic individuals (i.e., \( \sigma > 0.5 \))

\[ W_t = \exp\left(-\tau \cdot \sum_{i=0}^{n} M_{t,i} - k_d - n \cdot k_s\right), \]

where \( n \) is the total number of times an individual adjusts its phenotype during its lifetime.

Individual-Based Simulations. Our evolutionary model is based on populations of 5,000 individuals exposed to mutation and natural selection for 50,000 discrete, nonoverlapping generations (simulation runs were replicated 100 times at each parameter combination). Reproduction occurs only at the end of generation 5,000.

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**Fig. 4.** Representative examples of population dynamics during transitions through evolutionary tipping points in our model. (A) In the simulations depicted here, populations were forced to move from the region of reversible plasticity into that of bet-hedging by lowering \( P \) from 0.3 to 0.2 at \( \log R = 0 \) (all other model parameters as in the main text). (Top) Change in the correlation between cues and environmental values. (Middle) Evolution of traits before and after the transition (black = s, blue = a, green = h, gray = lo, and red = b; the time of transition is depicted by a dashed vertical line at generation 5,000). (Bottom) Associated changes in population size over time. (A) Even though the change in predictability is barely visible to the naked eye, populations immediately decline after predictability is reduced. (B) In most situations, populations become extinct because the mutations required to adapt to the new environment fail to arise. (C) However, in cases where beneficial mutations arise on time, these traits tend to reach fixation quickly and evolutionary rescue is complete.

[Image of population dynamics examples]
of each generation and is proportional to the payoffs accumulated during each individual’s lifetime ($W_i$). Thus, the number of offspring for individual $i$ is drawn from a Poisson distribution with mean $W_i / W_{\text{max}}$, where $W_{\text{max}}$ is the mean cumulative payoff for that generation. As a consequence, the average number of offspring per individual is equal to one and the size of the offspring population is very similar to that of the parent population. To compensate for the occasional differences between these two population sizes, we randomly removed or replicated offspring when needed to maintain a population of 5,000. All offspring in our model inherit the alleles at each locus from their parents, with a per locus mutation probability of $\mu = 0.001$ and mutational steps drawn from a normal distribution with a mean of zero and an SD of 0.05. The loci that encode slopes in the reaction norms ($b_i$ and $b_j$) are only allowed to mutate if individuals are plastic (i.e., when $s > 0.5$). Otherwise, these traits are set to zero and subsequently ignored unless $s$ evolves a value greater than 0.5.

**Simulating Transitions to Different Regions of Parameter Space.** To include the possibility of varying population sizes into our model, we replaced relative reversibility in plasticity ($\rho$) with $\rho = 0.05$. The loci that encode slopes in the reaction norms ($b_i$ and $b_j$) are only allowed to mutate if individuals are plastic (i.e., when $s > 0.5$). Otherwise, these traits are set to zero and subsequently ignored unless $s$ evolves a value greater than 0.5.

Parameter Settings. All simulations reported above are based on the following parameters unless otherwise stated: $s = 5$, $\rho = 0.02$, $\rho = 0.01$, $z = 0.25$, and $q = 2.2$. In every replicate, with the exception of transition simulations, the starting population was initialized by setting $h = 1$ (i.e., assuming that genomes only code for one norm of reaction), and by drawing the remaining traits for each individual at random from uniform distributions on $[0, 1]$ for $a$ and $s$; $[-1, 1]$ for $b_i$ and $b_j$; and $[-2, 2]$ for $b_i$ and $b_j$. Subsequent evolution was completely unbounded and determined solely by mutation and natural selection.

**ACKNOWLEDGMENTS.** C.A.B. was supported by US Geological Survey Grant/ Cooperative Agreement G10AC00624. J.W. was supported by the Centre for Biodiversity Dynamics at the Norwegian University of Science and Technology. D.R.R. was supported by National Science Foundation Grants IOS-1121435 and IOS-1257530.