

When evolution realizes large deviations of fitness: from speciation to dynamical phase transitions

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We explore the connection between evolution and large-deviation theory. To do so, we study evolutionary dynamics in which individuals experience mutations, reproduction, and selection using variants of the Moran model. We show that, in the large population size limit, the impact of reproduction and selection amounts to realizing a large-deviation dynamics for the non-interacting random walk in which individuals simply explore the genome landscape due to mutations. This mapping, which holds at all times, allows us to recast transitions in the population genome distribution as dynamical phase transitions, which can then be studied using the toolbox of large-deviation theory. Finally, we show that the mapping extends beyond the class of Moran models.

In stochastic systems with finite correlation times, time-averaged quantities converge to their typical values in the long-time limit. Doing otherwise requires deviating from the typical value for an extensive period of time, which becomes exponentially rare as time increases. The theory of large deviations explores such rare events, which play a crucial role in a variety of systems ranging from glassy dynamics [1, 2] to geophysical flows [3–5].

To explore the large deviations of an observable $A(t) = \int_0^t dt' a(t')$, a generalization of statistical mechanics to trajectory space has been introduced [6–12]. In practice, one considers a biased ensemble, where trajectories are weighted by an additional factor $e^{sA(t)}$, such that

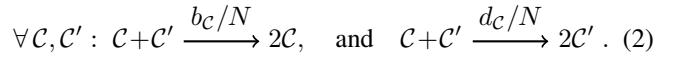
$$\mathcal{Z}_s(t) = \langle e^{sA(t)} \rangle \quad \text{and} \quad \mathcal{F}_s(t) = -\ln \mathcal{Z}_s(t) \quad (1)$$

play the role of partition function and free energy in trajectory space, respectively. The parameter s plays a role akin to that of temperature and allows controlling the values of A that are typical in the biased ensemble.

Algorithms that make large deviations typical have allowed detecting chaotic breathers and solitons in anharmonic chains of oscillators [13], to make glassy materials flow [2], or to induce traffic jams in transport models [14]. To do so, these methods rely on the simulations of ensembles of copies of the system that compete for survival: in a controlled way, trajectories are killed or cloned depending on their realization of $A(t)$ [15]. To this date, these numerical methods are without counterpart in the experimental world, where controlling the ‘temperature’ s has remained an open challenge. It is interesting to note that the dynamics underlying the aforementioned algorithms looks superficially akin to a population dynamics. A natural question is then whether evolutionary dynamics could be realizing large deviations and, if so, of what?

In this Letter, we address this question by considering evolutionary dynamics inspired by the Moran model [16]. A population of N individuals explores a space of genotypes $\{\mathcal{C}\}$ via mutations occurring with rates $W(\mathcal{C} \rightarrow \mathcal{C}')$, that are allowed to depend on \mathcal{C} and \mathcal{C}' [17]. In addition, individuals undergo reproduction by replacing other existing individuals, keeping the population constant. Not all genotypes are equivalent and

selection by competition occurs via genotype-dependent birth and death processes



From a physics perspective, the system amounts to N individuals undergoing non-interacting random walks in the genome space with rates W , complemented by interactions given by Eq. (2). We define $f_{\mathcal{C}} = bc - dc = sf_{\mathcal{C}}^0$ the fitness of genotype \mathcal{C} , where we have introduced a parameter s to control the relative strength of mutation and selection, and define the trajectory fitness as

$$F = \int_0^t dt' f_{\mathcal{C}(t')}^0. \quad (3)$$

Our first important result is that, in the large- N limit, the evolutionary dynamics described above realizes a large deviation of the trajectory fitness for the mutation dynamics *without selection*: the abundance $x_{\mathcal{C}}$ of genotype \mathcal{C} in the evolutionary dynamics at time t is given by its probability in the ensemble of non-interacting random walks induced by mutations only, weighted by $e^{sF(t)}$. Selection realizes a large deviation of the fitness F in the selection-free dynamics, which directly connects large-deviation theory and evolutionary dynamics.

From a large-deviation perspective, our mapping shows that Moran processes offer an alternative to existing simulation methods [15], which has the advantage of employing a constant population size. Here, we focus instead on the implications on the evolutionary side. More precisely, we show how we can recast the sudden variations of genome distributions as model parameters are varied in terms of dynamical phase transitions [2, 18–20]. Our main result is that the mapping allows us to assess the impact of the mutation and selection landscapes on the resulting genome diversity. Concretely, we show how first-order dynamical phase transitions amount to sudden jumps in the dominating genotypes, whereas continuous transitions lead to the emergence of bimodal genome distributions, akin to a sympatric speciation transition [21–25]. In the latter case, we show interesting finite-population effects:

genome coexistence emerges only in large enough population sizes. Finally, we close the Letter by extending our mapping beyond the case of Moran models, relaxing in particular the constraint of fixed population size.

The model. In the large population limit, the abundance x_C of individuals of genotype C evolves according to:

$$\partial_t x_C = \sum_{C'} \mathbb{W}_{CC'} x_{C'} + \sum_{C'} (f_C - f_{C'}) x_C x_{C'} \quad (4)$$

where we defined the stochastic matrix $\mathbb{W}_{CC'} = W(C' \rightarrow C) - r_C \delta_{CC'}$, with $r_C = \sum_{C'} W(C \rightarrow C')$ the rate at which individuals mutate out of genotype C . The non-linear term involving the effective selection rates $f_C = s f_C^0$ stems from the competition between genotypes induced by the selection process in Eq. (2). By definition, abundances sum to 1 at all times and indeed Eq. (4) preserves $\sum_C x_C = 1$. For compactness, we use bra-ket notation and introduce a vector $|x\rangle$ whose components are the abundances $x_C = \langle C|x\rangle$, where $\{|C\rangle\}_C$ denotes the canonical basis labeled by genotypes C , and $\langle \cdot | \cdot \rangle$ is the canonical scalar product. Equation (4) describes a selection-mutation model for the “quasispecies” $|x\rangle$ that attracted a lot of attention in population genetics [26–30], and belongs to the class of Eigen models [31].

Mapping onto a large-deviation problem. Consider the linear part of Eq. (4), which describes the (ergodic) random walk in genotype space induced by the mutation rates W . We first study how this random walk makes the trajectory fitness F defined in Eq. (3) evolve in time, *in the absence of selection*. To do so, we consider the joint probability $P(F, C, t)$ for the system to be in configuration C , with a trajectory fitness F , at time t . Its Laplace transform $\hat{P}_s(C, t) = \int dF e^{sF} P(F, C, t)$ evolves according to $\partial_t |\hat{P}_s\rangle = \hat{\mathbb{W}}_s |\hat{P}_s\rangle$ with $\hat{\mathbb{W}}_s = \mathbb{W} + \mathbb{f}$, where \mathbb{f} is the diagonal matrix with $\mathbb{f}_{CC} = f_C$. We note that the biased operator $\hat{\mathbb{W}}_s$ is not probability conserving and, starting from an initial state $|P_i\rangle$, the large-time behavior of $|\hat{P}_s\rangle$ is given by:

$$|\hat{P}_s(t)\rangle = e^{t\hat{\mathbb{W}}_s} |P_i\rangle \underset{t \rightarrow \infty}{\sim} e^{t\psi_s} |R_s\rangle \langle L_s| P_i \rangle \quad (5)$$

where $\psi_s \in \mathbb{R}$ is the (unique) eigenvalue of $\hat{\mathbb{W}}_s$ with the largest real part. The matrix $\hat{\mathbb{W}}_s$ is not symmetric and we denote $|R_s\rangle$ and $|L_s\rangle$ the right and left eigenvectors associated to ψ_s , respectively. Introducing the flat vector $|-\rangle$, whose entries equal 1, we use the normalization $\langle L_s|R_s\rangle = \langle -|R_s\rangle = 1$. Importantly, the Perron–Frobenius theorem ensures that $|L_s\rangle$ and $|R_s\rangle$ exist, are unique, and have strictly positive entries.

Varying the value of s in $\hat{\mathbb{W}}_s$ allows exploring the large deviations of the fitness F : ψ_s is indeed the scaled cumulant-generating function of F while the Laplace transform $|\hat{P}_s\rangle$ weights $P(F, C, t)$ with the exponential factor $e^{sF(t)}$ [11]. Using $s = 0$ thus leads to an unbiased sampling of the random walks induced by mutations only, whereas $s \neq 0$ favors mutation histories with atypical values of the fitness F .

Our first main result is that the Moran process realizes the large deviations of the trajectory fitness generated by the

selection-free mutation dynamics. Indeed, it is known that non-linear evolutionary dynamics such as Eq. (4) map onto linear processes [27] by introducing a properly chosen normalization factor. This has been used, in particular, to map evolutionary dynamics onto quantum Ising chains [30, 32–34]. In our context, one directly checks by substitution that the solution to the (non-linear) Moran Eq. (4) is given at all times by that of the linear biased dynamics $\hat{\mathbb{W}}_s$ through:

$$|x(t)\rangle = \frac{|\hat{P}_s(t)\rangle}{\mathcal{Z}_s(t)}, \text{ with } \mathcal{Z}_s(t) = \langle -|\hat{P}_s(t)\rangle = \langle e^{sF(t)} \rangle. \quad (6)$$

At $t = 0$, $F = 0$ so that $\mathcal{Z}_s(0) = 1$ and $|x(0)\rangle = |\hat{P}_s(0)\rangle = |P_i\rangle$. The implication of the mapping in Eq. (6) is that selection, which takes the form of the birth-death events in Eq. (2), is completely equivalent to favoring mutation histories that realize atypical values of the trajectory fitness F .

Consequently, the distribution of abundances $|x(t)\rangle$ reaches at large time a unique stationary state given by $|x_s^*\rangle = |R_s\rangle$ while the cumulant-generating function $\psi_s = \langle -|\hat{\mathbb{W}}_s|R_s\rangle = \langle -|\mathbb{f}|R_s\rangle$ gives the population fitness at steady state, i.e. the average of f_C over the population. In addition to the insight into evolutionary dynamics given by Eq. (6), the mapping can thus also be used as a practical way to study large deviations of any observable F in a Markov jump process by simulating a Moran process at large but fixed population size. One can then estimate $|\hat{P}_s(t)\rangle$ from $|x(t)\rangle$ and the cumulant-generating function from $\langle -|\mathbb{f}|x(t)\rangle$ at large times. This approach could solve issues present in other population dynamics algorithms used to simulate large deviations [15, 35, 36]. In the rest of this Letter, we focus instead on the implications for evolutionary dynamics, especially in phenomena akin to speciation. In particular, large-deviation theory tells us that varying the parameter s can induce dynamical phase transitions. We show here how this can be recast into a speciation transition for the evolutionary dynamics (4).

Dynamical phase transition and speciation. To do so, we study the fate of a population when the relative strength s of selection with respect to mutation is varied. We follow [17, 37] and consider a “ladder-model” illustrated in Fig. 1(a), in which genotypes are indexed by an integer $-M_0 \leq M \leq M_0$, which we rescale as $m = M/M_0 \in [-1, 1]$. We assume that a mutation landscape favors the $m = 0$ genotype via the transition rates $W(m \rightarrow m \pm M_0^{-1}) = \gamma(1 \mp m)$. On the contrary, the selection process favors genotypes away from $m = 0$, via a reproduction rate $b(m) = f(m) = s f^0(m)$ that increases with $|m|$ (and a death rate $d(m) = 0$).

Simulations of the Moran process show that, as s increases, the population experiences a variety of transitions that appear continuous or discontinuous depending on the symmetry and shape of $f(m)$. When $f(m)$ is symmetric, Figs. 1(c-d) show that the system undergoes a continuous “speciation” transition: For $s < s_c$, the genotype distribution is unimodal and individuals are localized close to the same optimal $m = 0$ genotype; For $s > s_c$, the genotype distribution becomes bimodal and two populations coexist with markedly distinct genotypic

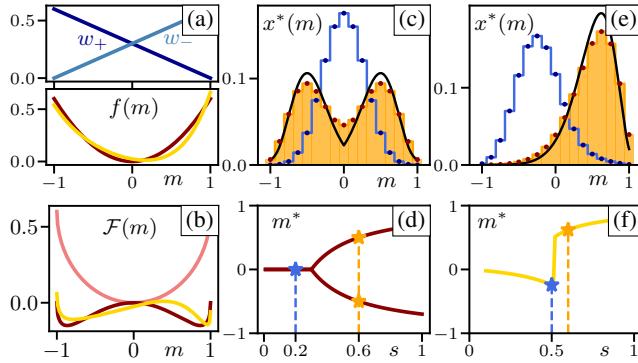


FIG. 1. (a): Mutation rates $w^\pm(m) = \gamma(1 \mp m)$ and selection landscape $f(m) = s [m + \alpha(m-1)(1+m)^2 + \delta m]$ represented for $s = 1$ in the symmetric case (red, $\alpha = \delta = 0$) and asymmetric case (yellow, $\alpha = 0.2$ and $\delta = 0.1$). (b): Landau free energy Eq. (8) in the symmetric case ($s = 0$, pink and $s = 0.6$, red) and asymmetric case (for $s = 0.6$, yellow). (c-d) Distribution $x_s^*(m)$ and its maxima m^* in the symmetric case of panel (b). The theory predicts the sympatric speciation transition at $s_c = 0.3$. Histograms from simulations of the Moran model are shown for $s = 0.2$ (blue star & histogram) and $s = 0.6$ (orange star & histogram). The black line corresponds to the WKB prediction ($M_0 \gg 1$) and the dots correspond to the right eigenvector $|R_s\rangle$ obtained from the exact diagonalization of $\hat{\mathbb{W}}_s$ for $M_0 = 8$. (e-f): Same as (c-d) for the asymmetric free energy in panel (b). The theory predicts a first-order transition at $s \simeq 0.52$ and we show data from numerical simulations of the Moran model for $s = 0.5$ (blue) and $s = 0.6$ (orange). All simulations are performed for $N = 6 \times 10^5$, $M_0 = 8$ and $\gamma = 0.3$.

distributions. Instead, when $f(m)$ is asymmetric, with a thin and sharp maximum at $m = 1$ and a broader—but less fit—maximum at $m = -1$, Figs. 1(e-f) show that the transition becomes discontinuous: $m < 0$ is favored at small selection pressure before the population genotype suddenly jumps at a critical s_c to favor $m > 0$. To account for these behaviors, we study the variations of the genotype distribution $|x_s^*\rangle$ as s changes using the framework of large deviations and dynamical phase transitions [18–20, 38].

In practice, solving the Moran Eq. (4) is a complex non-linear problem, but progress can be made thanks to the toolbox of large-deviation theory. First, the mapping in Eqs. (5)–(6) effectively linearizes the problem. Then, while the evolution operator $\hat{\mathbb{W}}_s$ entering Eq. (5) is not symmetric, it can be brought into a symmetric form $\hat{\mathbb{W}}_s^{\text{sym}}$ thanks to a suitable change of basis [39]. In the large M_0 limit, we can then determine the population fitness ψ_s using a variational principle [38]:

$$\psi_s = \max_v \frac{\langle v | \hat{\mathbb{W}}_s^{\text{sym}} | v \rangle}{\langle v | v \rangle} \underset{M_0 \gg 1}{\sim} -\min \mathcal{F}(m), \quad (7)$$

where $\mathcal{F}(m)$ plays the role of a Landau free energy. Introducing $r(m) = w^+(m) + w^-(m)$, \mathcal{F} is given by

$$\mathcal{F}(m) = r(m) - f(m) - 2\sqrt{w^+(m)w^-(m)}. \quad (8)$$

The population distribution is then found from a WKB ansatz

$x_s^*(m) \propto e^{-M_0 I(m)}$ with a rate function $I(m)$ that solves

$$w^+(m)e^{I'(m)} + w^-(m)e^{-I'(m)} = r(m) - f(m) + \psi_s \quad (9)$$

with ψ_s given by Eq. (7). As usual in WKB asymptotic analysis of phase transitions [40], Eq. (9) accepts two solutions for $I'(m)$ which have to be compared, as detailed in [39]. We now discuss the solutions for the symmetric and asymmetric $f(m)$ considered in Fig. 1(a).

When the selection rates are symmetric under $m \rightarrow -m$, the Landau free energy undergoes a second-order transition akin to a ϕ^4 phase transition, see Fig. 1(b). For $s < s_c = \gamma$, $\mathcal{F}(m)$ is convex and its minimum is located at $m = 0$. Mutations dominate the dynamics and selection simply amplifies the population diversity by broadening the genotype distribution. For $s > s_c$, $\mathcal{F}(m)$ develops a double-well structure with minima at $m = \pm m_{\text{opt}}$: selection overcomes mutations, favoring genotypes away from $m = 0$. While this scenario is reminiscent of a ferromagnetic transition, the steady-state solution for $s > s_c$ is the coexistence of two sub-populations with genotypes centered around $\pm m^*(s)$ (see Fig. 1(c-d)), and not an ergodicity-broken phase in which the system would pick one of the two solutions. This can be seen by determining the distribution $x_s^*(m)$, which transitions at $s = s_c$ from a unimodal distribution to a bimodal distribution. A further difference with the ferromagnetic transition is that the most represented genotypes in $x_s^*(m)$ are *not* located at the free energy minima $\pm m_{\text{opt}}$. The latter indeed correspond to the most represented genotypes among the ancestors of the surviving population, and need not equal the most represented genotypes of the survivors. Mathematically, this is because $\pm m^*$ correspond to the maxima of $R_s(m)$ while $\pm m_{\text{opt}}$ maximize $L_s(m)R_s(m)$, which is the distribution of the ancestors of the surviving population. This feature, surprising if one tries to understand the speciation transition as a static phase transition, is a standard feature of dynamical phase transitions in large-deviation theory [2, 14, 41].

Let us now turn to the case of the asymmetric $f(m)$ depicted in Fig. 1(a). Because of its asymmetric shape, $f(m)$ has a sharp peak at $m = 1$ and a broader but less pronounced peak at $m = -1$. At small s , the genotype distribution first crosses over from a peak at $m = 0$ toward a peak at $m < 0$. At a critical value s_c that can be determined analytically from Eq. (7), the system discontinuously jumps to a distribution peaked at $m > 0$, approaching $m = 1$ at large s . As s increases, the population thus transitions from favoring the broadest fitness peak, which amplifies a broad set of genotypes, to the fittest peak, whose maximal fitness eventually takes over. This can be understood by the fact that, when s is small and mutations occur at a faster rate than selection, broad fitness peaks offer a stronger robustness against detrimental mutations, an effect that has been named “the survival of the flattest” [42–45]. On the contrary, at large s , maximizing fitness is the best strategy. Mathematically, this transition can be inferred from the evolution of the Landau free energy $\mathcal{F}(m)$, whose minima are degenerate at s_c , as typical in first-order phase transitions.

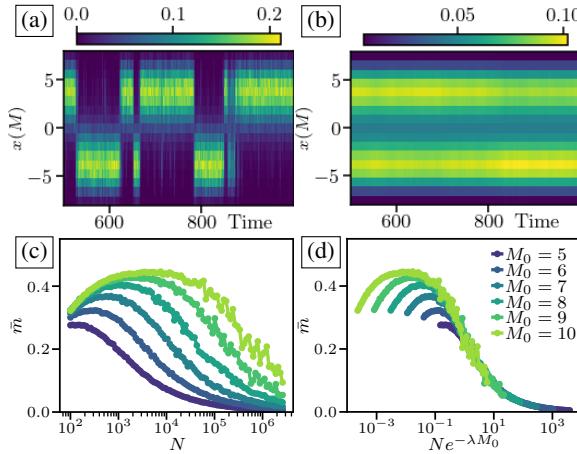
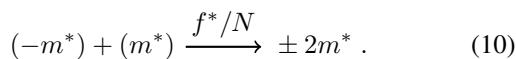


FIG. 2. (a-b): Evolution of the genotype distribution from Moran simulations for $M_0 = 8$ and $N = 3.189 \times 10^3$ (a) or $N = 1 \times 10^7$ (b). (c-d): Order parameter $\bar{m} = \sum_{i=1}^N \frac{|m_i|}{N}$ as a function of N (left) that distinguishes coexistence ($\bar{m} = 0$) and stochastic switching ($\bar{m} \sim O(1)$), for different values of M_0 . The curves can be collapsed around the transition by rescaling N with a characteristic population size $N_c \sim e^{\lambda M_0}$. We found a good collapse for $\lambda \simeq 1.3$ which compares surprisingly well with the predicted value $\lambda = 1.31$.

Here also, the minima of $\mathcal{F}(m)$, which dominate the population at intermediate times, differ from the most represented genotypes among the descendants.

Finite populations. The results discussed so far, derived using the toolbox of large-deviation theory, hold for the case of infinite populations. It is well known in the literature on large-deviation simulations that using finite populations has important consequences [35, 36], and one may wonder what is the fate of the transitions discussed above for large but finite populations. The most interesting case is that of sympatric speciation in which we predict the coexistence of two different genotypes in the surviving population. As shown in Fig. 1(c), the sympatric coexistence survives at large but finite populations, and the genotype distribution of the Moran process reproduces the bimodal distribution predicted by the theory. For smaller populations, however, coexistence is replaced by a stochastic switch between genotype distributions centered at $\pm m^*$, as shown in Fig 2(a). Furthermore, the size N_c beyond which coexistence is restored is shown in Fig. 2(b) to sharply increase with M_0 . This effect is akin to well-known results on Wright–Fisher models with 2 possible genotypes (or alleles) in which the population size strongly affects the genotype diversity [46].

To adapt this framework to our problem, we consider a simpler “Wright–Fisher” version of the ladder model, in which individuals can have only two genotypes, $\pm m^*$. Selection then occurs through the reactions



In the absence of mutations, the dynamics admits two absorb-

ing states, in which the fraction ρ of the population that has a genotype m^* satisfies $\rho = 0$ and $\rho = 1$, respectively. In the large- N limit, the system evolves according to the Fokker–Planck equation [39, 46] $N \partial_t P(\rho) = f^* \partial_\rho^2 [\rho(1 - \rho)P(\rho)]$, which is an example of Kimura diffusion. Fixation thus occurs in a time $\sim O(N)$. The situation changes, however, if a mutation rate $\tilde{\gamma}$ allows individuals to switch between the two genotypes, leading to the steady state [39, 46]

$$P_{\text{st}}(\rho) \propto [\tilde{\gamma} + 2f^*\rho(1 - \rho)]^{N/N_c - 1} \text{ with } N_c = \frac{f^*}{\tilde{\gamma}}. \quad (11)$$

When $N > N_c$, the distribution is peaked around $\rho = 1/2$: the most probable state is a population split between the two genotypes. On the contrary, $P_{\text{st}}(\rho)$ is peaked at $\rho = 0$ and $\rho = 1$ when $N < N_c$. In that situation, the system jumps randomly between two states in which most individuals have the same phenotype. In this two-genotype model, speciation is thus replaced by metastability at small population sizes.

The goal is then to relate this simplified picture to the full ladder model with $2M_0 + 1$ genotypes. In the ladder model, the mutation-induced population switch is realized by individuals that manage to mutate from, say, $-m^*$ to m^* , before they reset to $m = -m^*$ due to a selection event. Assuming that the other $N - 1$ individuals remain at $-m^*$, resetting occurs with rate $(N - 1)f^*/N \simeq f^*$. We thus estimate the effective mutation rate $\tilde{\gamma}$ of the simplified model as the inverse of the mean-first passage time from $-m^*$ to m^* of a single individual undergoing a mutation-induced random walk in the ladder model, and resetting to $-m^*$ with rate f^* . Using methods developed to study stochastic resetting [47, 48], detailed in [39], we find $\tilde{\gamma} \simeq f^* \exp[-M_0 \lambda]$, where $\lambda = \max_m \{J_{f(m)}(m) - I(m)\}$ with $I(m)$ obtained from Eq. (9) and

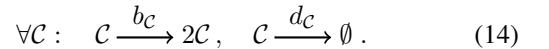
$$J_\sigma(m) = \int_{m^*}^m dm' \log \frac{2\gamma + \sigma - \sqrt{4m'^2\gamma^2 + 4\gamma\sigma + \sigma^2}}{2\gamma(1 - m')}. \quad (12)$$

We thus predict a transition from sympatric speciation to metastability when the population and genome sizes satisfy

$$N < N_c \equiv \exp[M_0 \lambda], \quad (13)$$

which agrees well with the numerics shown in Fig. 2.

Beyond Moran processes. So far, we have shown that the non-linear selection entering the Moran process can be seen as realizing a large deviation of the fitness of the mutation-induced random walk in genotype space. A natural question is how general this appealing image of evolutionary dynamics is. A first step to address this is to relax the constant-population constraint of the Moran process. To do so, we consider a model in which the mutation-induced random walk in genome space at rates W is complemented by one-body birth & death events occurring with rates:



By itself, this population dynamics leads to either exponential growth or extinction of the population. When the density is

large enough, competition between individuals occupying a common volume V should set in. We thus consider additional mutualistic and antagonistic interactions in the form of

$$\forall \mathcal{C}, \mathcal{C}' : \mathcal{C} + \mathcal{C}' \xrightarrow{mc'/V} 2\mathcal{C} + \mathcal{C}', \quad \mathcal{C} + \mathcal{C}' \xrightarrow{ac'/V} \mathcal{C}' . \quad (15)$$

In the dynamics (15), any individual with genome \mathcal{C}' can either help an individual with genome \mathcal{C} to reproduce or kill it. Starting from a large population, the mean-field evolution of the abundance $x_{\mathcal{C}} = n_{\mathcal{C}}/V$ is given by

$$\partial_t x_{\mathcal{C}} = \sum_{\mathcal{C}'} \mathbb{W}_{\mathcal{C}\mathcal{C}'} x_{\mathcal{C}'} + \sum_{\mathcal{C}} f_{\mathcal{C}} x_{\mathcal{C}} - \sum_{\mathcal{C}'} \varphi_{\mathcal{C}'} x_{\mathcal{C}} x_{\mathcal{C}'} \quad (16)$$

where $f_{\mathcal{C}} = b_{\mathcal{C}} - d_{\mathcal{C}}$ is the one-body fitness, $\varphi_{\mathcal{C}'} = ac' - mc'$ represents the impact of genome \mathcal{C}' on the rest of the population, and $n_{\mathcal{C}}$ is the number of individuals in \mathcal{C} . Interestingly, we find that the solution $|x(t)\rangle$ of Eq. (16) still obeys Eq. (6), albeit with the factor $\mathcal{Z}_s(t)$ now given by

$$\mathcal{Z}_s(t) = 1 + \int_0^t dt' \langle \varphi | \hat{P}_s(t') \rangle , \quad (17)$$

where we have introduced the vector $|\varphi\rangle$ whose components in the basis $\{\mathcal{C}\}$ are given by $\varphi_{\mathcal{C}}$. Already, Eqs. (6) and (17) yield the surprising result that the relative abundances of the individuals in the population are entirely determined by their one-body linear dynamics. In contrast, the interactions (15) play a crucial role in controlling the factor $\mathcal{Z}_s(t)$.

The fate of the population can then be predicted by analyzing Eqs. (6) and (17). Consider first the case where, in the absence of interactions, the population grows exponentially ($\psi_s > 0$). Once mutualism and antagonism set in, they either turn the exponential increase of the population into a finite-time blow up, or, instead, damp the population growth and lead to a steady-state ecosystem with a finite population size. The outcome is decided by whether $\varphi_{\infty} = \langle \varphi | R_s \rangle$ is positive or negative. When $\varphi_{\infty} > 0$, the one-body birth-death dynamics (14) leads to a population distribution where antagonism dominates mutualism, which mitigates the population growth and stabilizes a finite-size ecosystem with stationary abundance $|x_s^*\rangle = \frac{\psi_s}{\varphi_{\infty}} |R_s\rangle$. On the contrary, when $\varphi_{\infty} < 0$, mutualism dominates and the interactions amplify the population growth, leading to a blow-up at a finite time t_b , determined by $\mathcal{Z}_s(t_b) = 0$ in Eq. (17). Instead, if $\psi_s < 0$, the fate of the population is governed by the amplitude of the initial abundances, through the quantity $\xi_0 = \langle \varphi | \hat{W}^{-1} |x(0)\rangle$. The population vanishes exponentially fast if $\xi_0 < 1$, i.e. if the initial population is small, or blows up at a finite time if $\xi_0 > 1$, when mutualism overcompensates the population decay. A stationary state is reached in the special case $\xi_0 = 1$. This, notably, occurs when one falls back on the model defined by Eq. (4), for which $|\varphi\rangle = |f\rangle$ and $\langle -|x(0)\rangle = 1$. Interestingly, a population that decays due to the one-body population dynamics Eq. (14) cannot be stabilized by interactions when $m_{\mathcal{C}}$ and $a_{\mathcal{C}}$ are time independent. Either $\varphi_{\infty} > 0$ and the decay is accelerated or $\varphi_{\infty} < 0$ and, generically, the mutualism will

overshoot and lead to diverging population at finite time. It would be interesting to check whether this can be mitigated by considering mutualistic and antagonistic interactions that depend on the population size in Eq. (15). All in all, this section shows that the mapping to large-deviation theory thus extends to evolutionary dynamics with non-constant populations.

Conclusion. Fitness and the impact of evolution on the traits of populations are fascinating aspects of biological systems, without counterparts in the world of soft-matter physics. By establishing a mapping between the large deviations of stochastic processes and evolutionary dynamics, our work bridges these two worlds and offers a new perspective on how selection interplays with mutations. From a statistical mechanics perspective, this mapping is a first route towards the realization of large deviations and the study of dynamical phase transitions in experiments. A relevant class of biological systems where this could apply are viral populations, where mutations are particularly frequent [45, 49]. Furthermore, we have shown how dynamical phase transitions offer a new framework and toolbox to study transitions occurring in evolutionary dynamics. In particular, our formalism opens up a route to identify the key mechanism that induces a first-order transition from the flattest to the fittest. It would then be interesting to see if such a criterion could be related to the canonical equation of adaptive dynamics introduced in [50]. Finally, we have established the mapping in the context of Moran processes, with and without fixed-population constraints. These models have attracted a lot of attention in the mathematical biology and evolutionary dynamics literature, and we should now explore how far the mapping can be generalized beyond the case discussed in this Letter.

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