Chaos

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Cite as: Chaos **30**, 073103 (2020); https://doi.org/10.1063/5.0010057
Submitted: 06 April 2020 . Accepted: 12 June 2020 . Published Online: 02 July 2020

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Submitted: 6 April 2020 · Accepted: 12 June 2020 ·
Published Online: 2 July 2020

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ABSTRACT

The key to the survival of a species lies in understanding its evolution in an ever-changing environment. We report a theoretical model that integrates frequency-dependent selection, mutation, and asexual reproduction for understanding the biological evolution of a host species in the presence of parasites. We study the host–parasite coevolution in a one-dimensional genotypic space by considering a dynamic and heterogeneous environment modeled using a fitness landscape. It is observed that the presence of parasites facilitates a faster evolution of the host population toward its fitness maximum. We also find that the time required to reach the maximum fitness (optimization time) decreases with increased infection from the parasites. However, the overall fitness of the host population declines due to the parasitic infection. In the limit where parasites are considered to evolve much faster than the hosts, the optimization time reduces even further. Our findings indicate that parasites can play a crucial role in the survival of its host in a rapidly changing environment.

Understanding the mechanism that drives the biological evolution of a species plays a significant role in its survival under an extreme and fast-changing environment. In this manuscript, we consider the ecological interaction between the host and the parasite populations that often influence each other's evolution. We thus formulate a mathematical model that describes the key features of host–parasite coevolution in terms of the coupled partial differential equations. The role of selection, mutation, and asexual reproduction is studied on the dynamics of a host population, which evolves in the presence of parasites. A swift response to a rapidly changing environment is crucial for the survival of any species. The idealized mathematical model formulated in the paper indicates that the evolution of host species in the presence of parasites can speed up its evolution toward a global fitness peak, though at lower fitness values.

I. INTRODUCTION

Biological evolution of organisms is a complex phenomenon that usually proceeds toward increasing complexity over successive generations. Understanding the underlying processes responsible for promoting the complexity has been an active area of research since the last century.[1–6] A theory on this evolutionary dynamics was first proposed by Darwin and Wallace[7,8] and has been applied successfully in understanding a variety of biological phenomena. Darwinian dynamics formulates the theory, though qualitatively only, for the evolution of populations with time and is primarily based on selection, mutation, and reproduction.[9] However, using mathematical models and statistical techniques, more quantitative descriptions of the resulting evolution have been proposed.[10–20]

Smith described natural selection in evolutionary game theory, where the reproduction rate (or fitness) of an individual with a fixed strategy is considered dependent on the frequency of

strategies of others in the population.[14] Schuster and Sigmund incorporated replication and selection by using the replicator equation to describe the evolution of traits.[21] An immediate consequence of this model was the dominance of the individuals with larger fitness as compared to those having smaller fitness. For describing the effect of mutations and frequency-dependent selection, the replicator–mutator equation was proposed.[22] In an attempt to unify various formulations on evolution dynamics, Page and Nowak showed equivalence between the replicator–mutator equation and the Price equation describing any form of selection.[9,23] These equations find applications in population genetics, language evolution, ecological diversity, etc. There have also been attempts to correlate the stochastic nature of Darwin's evolutionary dynamics with thermodynamics and statistical mechanics.[15,16,24,25] This connection is due to the emergence of Fisher's fundamental theorem of natural selection[10] and the concept of adaptive landscape (also known as fitness landscape) in the genotypic space coined by Wright.[26] These two ideas include the randomness and the selection appearing in Darwin's theory. The landscape idea has also been applied to explain glass dynamics and protein folding dynamics.[27]

A key concept, namely, coevolution, has emerged in understanding the biodiversity through early works on the genetics of flax and rust;[28] Mode's mathematical model on short-term evolutionary dynamics of interacting species;[29] and the work of Ehrlich–Raven on mutual influences of butterfly species and host plants.[30] When one species exerts selective pressures on the other species, it feels selection in return. Due to this reciprocal selection, the species undergoes mutual evolutionary change known as coevolution.[31–33] Therefore, the term coevolution implies that the environment that stimulates variation in one species due to ecological feedback is itself responsive to that variation. Examples of coevolution usually include mutualism as in plant–pollinator, competition as in predator–prey, and antagonism as in host–parasite interactions between species and within species as well. How to measure the strength of selection in quantifying such coevolutionary dynamics is the key to understanding the selection itself.[34] Typically, the coevolutionary dynamics exhibited by mutualism, competition, and antagonism are quite different. Using modeling and computer simulations, it has been argued that such coevolution can produce biological diversity.[35,36]

The ecological interaction between host and parasite has been classically termed as host–parasite coevolution.[32] Here, hosts are under selection pressure to resist parasitic infection. Reciprocally, parasites are under selection stress to evade host defenses. This evolutionary arms race leads to a frequency-dependent selection, where hosts and parasites are coevolving through countless evolutionary cycles, a phenomenon known as *Red Queen effect*.[37] The mechanism of fluctuating selection has been considered a critical factor in driving the spatial and temporal diversity in nature.[11,38] The coevolution of a host or a parasite from one genotype to another is driven by a dynamic genotype-fitness mapping.[9] The natural selection drives a population from a genotype of lower fitness to genotypes with higher fitness.

Our focus in this work is to develop a mathematical model where we can observe the role of selection, mutation, and asexual reproduction on the dynamics of a finite host population, which evolves in the presence of a finite population of parasites. We explore the effect of the genotype-fitness map on the evolutionary dynamics of the host species when it evolves in the presence of parasites, which gives a dynamic component to the otherwise static fitness landscape of hosts. The parasite population also evolves under a dynamic fitness landscape due to evolving hosts. Thus, the genotypic alteration in host and parasite populations is due to mutation and a dynamic fitness landscape. Furthermore, we also consider an adiabatic limit where the timescales of the evolution of hosts are considered much slower than that of the parasites. The genotypic space usually is high-dimensional, and, thus, a complete understanding of the evolution of species considering the genotype-fitness mapping in higher dimensions is a complex task. We reduce the complexity of the problem by considering a one-dimensional genotypic space. The interaction between the hosts and the parasites is modeled by using a frequency-dependent selection. The strength of the interaction is tuned by introducing a coupling parameter, which we refer to as the degree of virulence. We discuss results on the evolution of fitness of the host population for different mutation rates and different degrees of virulence. We also compute the time required for the host population to reach the global peak of the fitness landscape.

This paper is organized as follows. In Sec. II, we present the formulation of the model in terms of two coupled partial differential equations corresponding to the coevolution of hosts and parasites. These coupled dynamical equations are obtained by incorporating selection, mutation, and asexual reproduction. In Sec. III, we numerically solve the proposed mathematical model for a few representative cases and discuss the implications. Finally, in Sec. IV, we summarize our conclusions.

II. MATHEMATICAL MODEL

In the model presented here, we consider that hosts and parasites evolve with different fitness goals. The fitness landscape for each is considered to be in one dimension, i.e., the genotypic space is taken to be one-dimensional. The host and parasite populations are modeled to evolve under selection, mutation, and asexual reproduction. As mutations are like a random walk in genotypic space, the equation for the evolution of a population under mutations with a mutation rate $\tilde{\mu}$ can be modeled using the master equation,[39–41]

$$M(i,N+1) = M(i,N) + \frac{\tilde{\mu}}{2}\left[M(i+1,N) + M(i-1,N) - 2M(i,N)\right]. \tag{1}$$

In Eq. (1), $M(i,N+1)$ represents the number of individuals having a genotype i in the $(N+1)$th generation. Equation (1) allows equal probability $\tilde{\mu}$ to an individual with genotype i, to either acquire a genotype $i-1$ or $i+1$ in the next generation. Similarly, individuals with either genotype $i+1$ or $i-1$ can be converted to genotype i in one generation with equal probability $\tilde{\mu}$. In the continuum limit, the generation N is replaced by time $t = Nh$ and the genotype i is replaced by a continuous genotypic space x such that $x = i\epsilon$, where $h \to 0$ and $\epsilon \to 0$. In the model, selection is taken into account by considering a fitness landscape $g(x)$, which is a distribution function representing the fitness of a genotype x. Therefore, in this limit, the evolution of a population $M(x)$ at time t can be written as

$$M(x,t+h) = g(x)\left[M(x,t) + \frac{\tilde{\mu}\epsilon^2}{2}\frac{\partial^2}{\partial x^2}M(x,t)\right]. \tag{2}$$

To keep the increment in the overall population size low in successive generations, $g(x)$ is considered to be very close to 1. Thus, we write

$$g(x) = 1 + hf(x), \tag{3}$$

where $f(x)$ is of $\mathcal{O}(1)$. Substituting this form of fitness $g(x)$ in Eq. (2),

$$\frac{\partial}{\partial t}M(x,t) \approx f(x)M(x,t) + \mu\frac{\partial^2}{\partial x^2}M(x,t), \tag{4}$$

where $\mu = \tilde{\mu}\epsilon^2/2h$. We transform the above equation involving population $M(x,t)$ to an equation involving frequencies $m(x,t)$ by normalizing the number of individuals having a genotype x with the total number of individuals in the population, $m(x,t) = M(x,t)/\int_{-\infty}^{\infty} M(y,t)dy$. Equation (4) then becomes

$$\frac{\partial}{\partial t}m(x,t) = f(x)m(x,t) + \mu\frac{\partial^2}{\partial x^2}m(x,t) - m(x,t)\int_{-\infty}^{\infty} f(y)m(y,t)dy. \tag{5}$$

The last term appearing on the right hand side of the above equation keeps the population constant. The above set of equations are derived generally for any population evolving under selection, mutation, and asexual reproduction.[25]

Next, we apply this formalism to coevolving host and parasite populations. In this model, we only consider inhibitory effects of parasites on hosts. By taking this into account, the fitness landscape of hosts is replaced by an effective fitness landscape f_{eff},

$$f_{\text{eff}}(x,t) = 1 + hf(x) - h\alpha f_H(x,t), \tag{6}$$

where α is the degree of virulence from parasites and $f_H(x,t)$ is a fitness function related to parasite population. Notice that the last term involving α in the above equation also appears, though in a different context, in the epidemiological model proposed by Anderson and May for studying the population dynamics of infectious diseases.[42,43] The form of $f_H(x,t)$ is assumed to be

$$f_H(x,t) = c_1\int_{-\infty}^{\infty} dy \exp[-\beta_1(x-y)^2]m_P(y,t), \tag{7}$$

where $m_P(y,t)$ is the frequency of parasites with genotype y. In writing down the above function, it is considered that the parasites can infect the hosts more effectively when they are closer to them in the genotypic space. The function also increases when the frequency of parasites closer to the hosts in genotypic space is higher. Therefore, a host, to be fit, will try to escape the most common parasites. For a given host genotype x, the pool of parasite genotypes y, which will infect that particular x, depends on the parameter β_1. For smaller values of β_1, a large number of parasite genotypes can infect the hosts and vice versa.

The fitness function for parasites $f_P(x,t)$ is written based on the fact that a parasite will evolve to match the most common host so that it can infect a host population more effectively.[35,38,44,45] Hence, the form of the function $f_P(x,t)$ is

$$f_P(x,t) = c_2\int_{-\infty}^{\infty} dy \exp[-\beta_2(x-y)^2]m_H(y,t). \tag{8}$$

The role of the parameter β_2 is similar to β_1, i.e., it controls the types of host genotype y that will be infected by the parasites of genotype x. Thus, according to the fitness functions $f_H(x,t)$ and $f_P(x,t)$, the hosts will evolve to escape the most common parasite species, while the parasites will evolve toward infecting the most common host genotypes. Finally, the equations for evolution of hosts and parasites are

$$\frac{\partial}{\partial t}m_H(x,t) = \tilde{f}_H(x,t)m_H(x,t) + \mu_H\frac{\partial^2}{\partial x^2}m_H(x,t) - m_H(x,t)\int_{-\infty}^{\infty} dy\tilde{f}_H(y,t)m_H(y,t), \tag{9}$$

$$\frac{\partial}{\partial t}m_P(x,t) = f_P(x,t)m_P(x,t) + \mu_P\frac{\partial^2}{\partial x^2}m_P(x,t) - m_P(x,t)\int_{-\infty}^{\infty} dyf_P(y,t)]m_P(y,t), \tag{10}$$

where in Eq. (9), $\tilde{f}_H(x,t) = f(x) - \alpha f_H(x,t)$. The average fitness of the host and parasite populations is given by

$$\mathcal{F}_H(t) = \int_{-\infty}^{\infty} dx\tilde{f}_H(x,t)\, m_H(x,t), \tag{11}$$

$$\mathcal{F}_P(t) = \int_{-\infty}^{\infty} dxf_P(x,t)\, m_P(x,t). \tag{12}$$

III. RESULTS AND DISCUSSION

In this section, we report a few representative cases for the mathematical model discussed in Sec. II. As discussed in Secs. I and II, the host population evolves under a dynamic fitness landscape, which has a static contribution $f(x)$ and a dynamic contribution $f_H(x,t)$ due to rapidly evolving parasites. The static part of the fitness landscape of hosts is taken to be a double Gaussian with one peak higher than the other peak, $f(x) = A_1\exp[-(x-x_1)^2/2\sigma_1{}^2] + A_2\exp[-(x-x_2)^2/2\sigma_2{}^2]$. In principle, there can be many more peaks in the fitness landscape. However, this work aims to observe the dynamics involved in the evolution of the host population from a lower (local) peak to a higher (global) peak in the absence and presence of parasites. Thus, we consider the simplest possible case with only one local and one global fitness peak in the landscape. The role of a suitable σ_1 and σ_2 in the fitness landscape $f(x)$ is to provide an environment that selects a more diverse pool of genes, thereby selecting at least few genes of an existing population. This represents the evolution of a species in response to environmental changes that are gradual and prevalent in nature and thus leads to a stable evolutionary dynamics. From Eq. (6), it can be seen that $f(x)$ has the unit of inverse time and so are the parameters A_1 and A_2 (related to the height of the peaks in the fitness landscape). The parameters σ_1 and σ_2 corresponding to the width of the peaks have dimensions of (genotypic) space. Therefore, for all the results presented here, the unit of time is taken as $\tau^* = 1/(2.5A_1)$ [A_1 is the amplitude of the smaller peak of $f(x)$] while the parameter σ_1 sets the scale of the genotypic space. The value of A_2 in units of τ^* is chosen as 0.6 and that of σ_2 in units of σ_1 is taken to be unity. The function $f(x)$ for which the results are presented in this section is shown in Fig. 1(a). The initial genotype frequencies of the host and the parasite populations are also provided in Fig. 1(a). The smaller and higher peaks

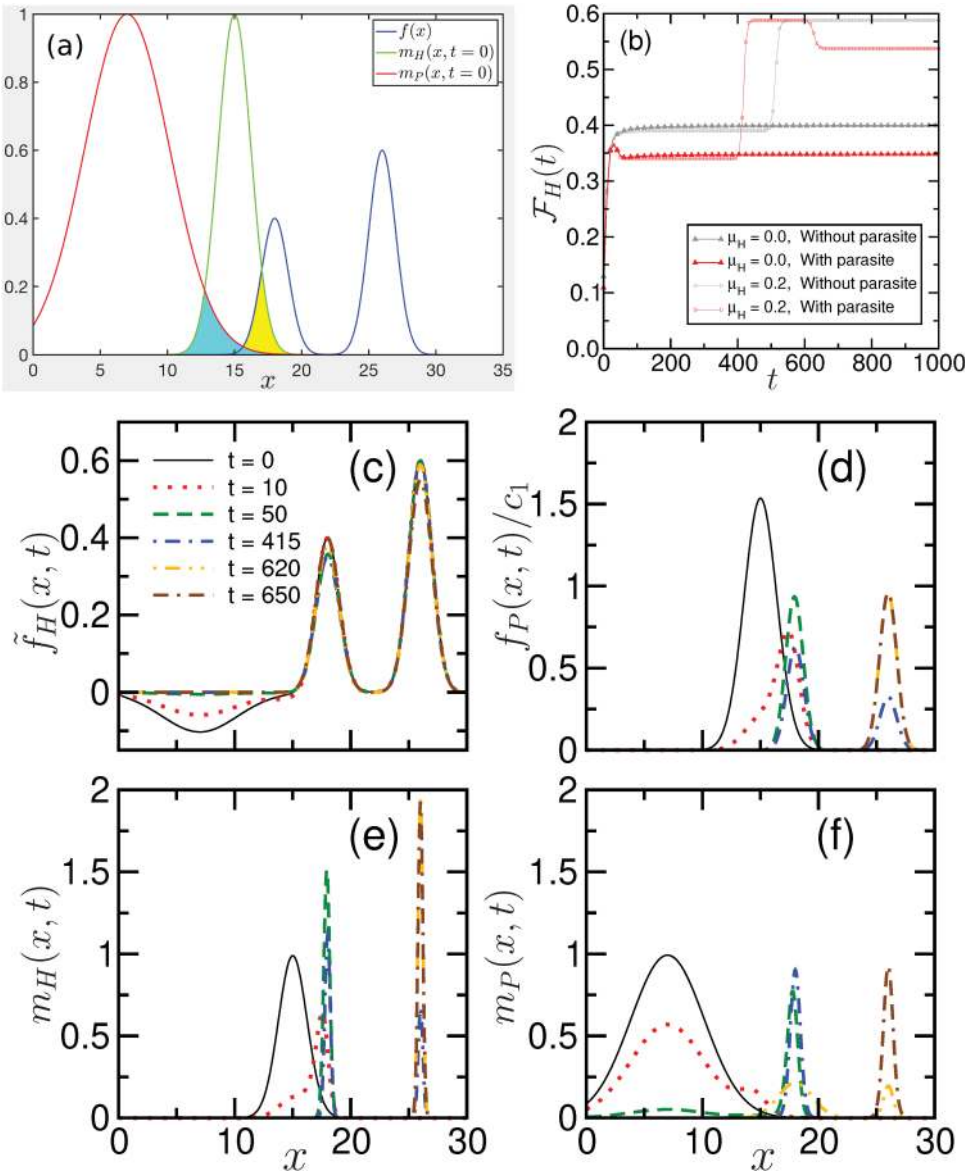

FIG. 1. (a) Static fitness landscape $f(x)$ driving the evolution of hosts vs genotype x (blue curve). The plots for the initial distribution of host (green) and parasite (red) frequencies are also shown. The finite overlap of the population distribution of the hosts with fitness landscape is shown in the yellow-filled region, whereas the region of overlap of the parasite with the host is shown in cyan color. (b) Evolution of hosts with time in the absence and presence of parasites is shown for $\mu_H = 0$ and $\mu_H = 0.2$. The value of the degree of virulence α is taken as 0.06, and $\mu_P = 0.9$ is considered for the evolution of parasites. The time evolution of (c) fitness landscape of hosts $\tilde{f}_H(x,t)$, (d) fitness landscape of parasites $f_P(x,t)$, (e) distribution of the host population $m_H(x,t)$, and (f) distribution of the parasite population $m_P(x,t)$.

of $f(x)$ are located at $x = 18$ and $x = 26$, respectively, while the host frequencies $m_H(x,t)$ and parasite frequencies $m_P(x,t)$ are both considered to have a Gaussian distribution around $x = 15$ and $x = 7$ with widths $\sqrt{3/2}$ and $\sqrt{10}$, respectively.

The choice of the above parameters does not change the results qualitatively but influences the timescales related to the dynamics. We choose these parameters to consider a finite overlap between parasite and host population as well as a finite overlap between the host population and the static part of the fitness landscape. In general, any population evolves with a finite mutation rate, and thus even if it starts with a finite pool of genes, the population will explore all the genotypes in subsequent generations depending on its mutation rate. This will, however, require diffusive timescales $[\sim\langle x^2\rangle/(2\mu_H)]$, which can be large if mutation rates are small. Thus,

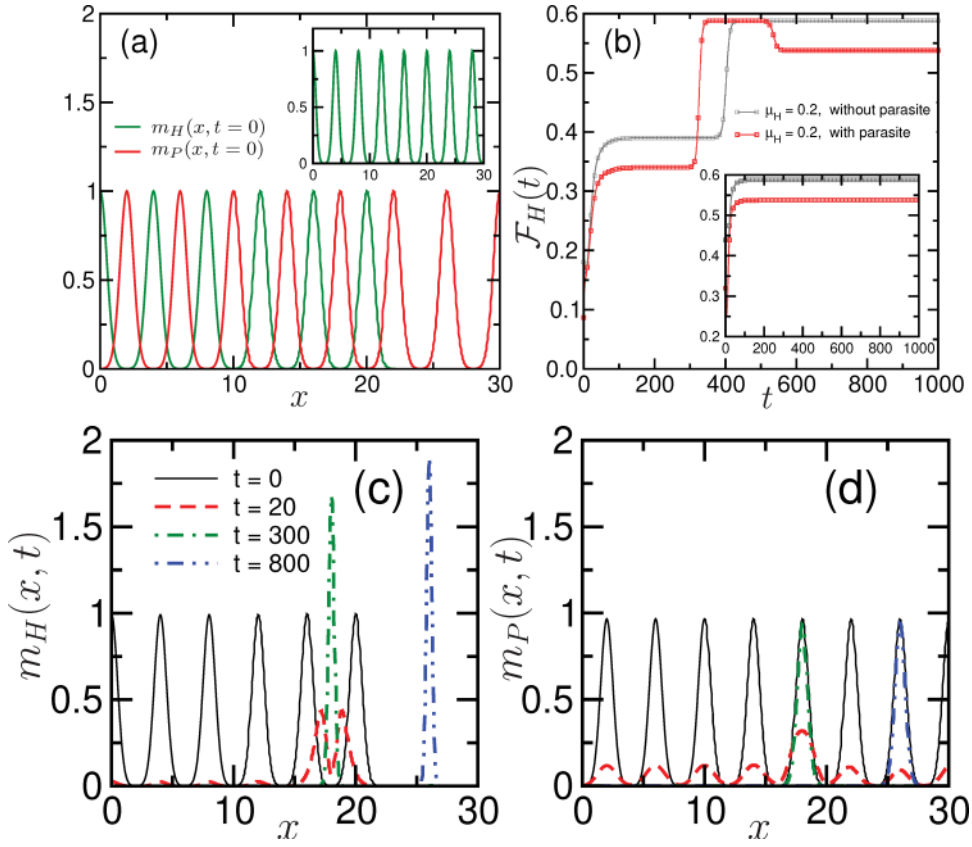

FIG. 2. (a) Initial distribution of the host and the parasite populations with increased diversity. Inset: the initial distribution of the host population covering the complete genotypic space. (b) Evolution of fitness of the host population at $\alpha = 0.06$ and $\mu_P = 0.9$. Inset: $\mathcal{F}_H(t)$ vs t is plotted for the distribution of the host population shown in the inset of Fig. 2(a). The corresponding evolution of (c) host population distribution and (d) parasite population distribution.

we start by considering a finite overlap between the populations and thereby reducing the simulation time. To implement that the host and parasite populations have a finite pool of genes at $t = 0$, genotype frequencies of hosts and parasites smaller than 10^{-8} are taken to be zero.

In Fig. 1(b), the evolution of fitness of host population $\mathcal{F}_H(t)$ [defined in Eq. (11)] in the absence and presence of parasites are compared for cases with zero and finite host mutation rate ($\mu_H = 0.2$). For $\mu_H = 0$, when the host population evolves in the absence of parasites, it can only detect the local peak at $x = 18$ with which it had a finite overlap at $t = 0$. It is to be noted that the population genetics models are often constructed using deterministic equations with an infinite population or using stochastic equations with a finite population. Here, the case with $\mu_H = 0$ implies a deterministic dynamics, which in principle must be considered with an infinite population. Nevertheless, we consider a zero mutation rate for reference. For $\mu_H = 0.2$, a two-step relaxation for fitness $\mathcal{F}_H(t)$ is observed. At first, the host population detects the local peak and evolves in time to get dispersed around it (at $t \approx 50$). The host population stays around the local peak for a duration given by the length of the first plateau $[\sim\langle x^2\rangle/(2\mu_H)]$ and then it reaches the global peak (at $t \approx 530$), which is evident by the second plateau in the plot.

We note that the host population gets dispersed around the local fitness peak in a short time, which is almost the same as the $\mu_H = 0$ case. This is because the host population already had the genotypes corresponding to the local fitness distribution, and, thus, its population is drifted toward those genotypes (activated dynamics) due to selection. The evolution of hosts from the local peak at $x = 18$ to the global peak at $x = 26$ is slower as it requires the host population to diffuse in genotypic space for which the timescale depends on the corresponding mutation rate as $\sim\langle x^2\rangle/(2\mu_H)$. Once the host population acquires the genotypes corresponding to the global fitness peak, the dynamics becomes activated again (a sharp increase in average fitness). We refer to the time required to reach the global fitness peak as optimization time τ. Similar behavior is observed for all other mutation rates (results not shown) with the optimization time depending inversely on the mutation rates.

As parasites are introduced to coevolve with the host population, the fitness landscape $f(x)$ for hosts gets modified and is replaced by the fitness function $f_{\text{eff}}(x,t)$ given by Eq. (6). The evolution of fitness of the host population with a fixed degree of virulence or

parasitic infection $\alpha = 0.06$ is also reported in Fig. 1(b). For this, we set the parameters c_1, c_2, β_1, and β_2 to be 1, 0.1, 1, and 1, respectively, in Eqs. (7) and (8). These parameters are required to calculate the dynamic fitness landscape of hosts and parasites. The value of parasite mutation rate μ_P in Eq. (10) is fixed at 0.9 for all the cases investigated hereafter. We emphasize here that the choice of parameters c_1, c_2, and μ_P influence the timescales only while the qualitative trends reported in the manuscript are independent of these particular parameters. As discussed in Sec. II, the parameters β_1 and β_2 control the extent of genotypic interaction between hosts and parasites. For zero mutation rate, even in the presence of parasites (which mutate with a finite rate), the host population is unable to reach the global fitness peak. The reduction in fitness by an amount of approximately 0.06 for the hosts is due to exposure to parasites with $\alpha = 0.06$.

For further discussion, we refer to the evolution of hosts in the absence and presence of parasites at $\mu_H = 0.2$ as in cases I and II, respectively. The dynamics in case II is observed to be significantly faster as compared to case I. It is seen that for both cases, the host population finds the local peak in almost the same duration, while for the global peak, the host population in case II arrives around it earlier. Furthermore, the value of average fitness achieved by the host population in case II also stays the same as the case I for some time interval (that depends on the mutation rate of parasite μ_P) after which it decays to a fixed value depending on the degree of virulence of the parasites. The decrease in fitness of hosts is due to the evolution of parasites toward infecting it. Therefore, when the host population gets localized around the global peak, the parasite population follows it. This leads to the dispersion of the parasite population around the same fitness peak as the hosts, which reduces the host fitness. Thus, we see that the asymptotic fitness achieved by the host population that evolves in the presence of parasites is lower as compared to the hosts that evolve in the absence of parasites with non-zero mutation rates. Therefore, we conclude that the faster optimization of host fitness in the presence of parasites comes with the cost of having a lower fitness. Moreover, we also observe that the average fitness of the host population $\mathscr{F}_H(t)$ decreases monotonically with increasing mutation rate μ_H (results not shown), except for the case with $\mu_H = 0$. As higher mutation rates give higher dispersion of a population around a fitness peak, the average fitness of a population decreases. However, we also observe that the asymptotic fitness of the host population for zero mutation rate is the lowest. This implies that finite mutation rates are essential for the optimization of fitness when the global fitness peak for a finite population exists far from its existing location in the genotypic space.

To further elucidate the observations made in Fig. 1(b), we analyze the temporal evolution of the fitness landscape of hosts $\tilde{f}_H(x,t)$, the fitness landscape of parasites $f_P(x,t)$, the population distribution of hosts $m_H(x,t)$, and the population distribution of parasites $m_P(x,t)$ in Figs. 1(c), 1(d), 1(e), and 1(f), respectively. At $t = 0$, the dip in $\tilde{f}_H(x,t)$ for $0 < x < 15$ is due to the negative contribution coming from the parasites located around $x = 7$ as shown in Fig. 1(c). However, $f_P(x,t)$, which is solely determined using the population distribution of hosts [Eq. (8)] is located around the peak at $x = 15$ [Fig. 1(d)]. In response to the fitness peak $\tilde{f}_H(x,t)$, $m_H(x,t)$ at $t = 10$ is observed to be drifting toward the local fitness peak of $\tilde{f}_H(x,t)$ at $x = 18$ as can be seen in Fig. 1(e). Subsequently, at $t = 50$, $m_H(x,t)$ is localized around the local peak. Furthermore, at $t = 415$, the host population is in the transient state [as is evident from Fig. 1(b)] and is gradually drifting toward the global fitness peak. Finally, the host population distribution reaches a steady state by dispersing around the global maximum as is seen for $t = 620$ and 650. By definition, $f_P(x,t)/c_1$ plotted in Fig. 1(d) shows the same time dependence. As a result, the parasite population distribution is seen to be drifting toward the host population at all times. It is observed that at $t = 620$, $m_P(x,t)$ is starting to build up around the global fitness peak of the hosts, which leads to a decrease in the average fitness of the hosts [see Fig. 1(b)]. Once $m_P(x,t)$ is completely localized ($t = 650$), $\tilde{f}_H(x,t)$ attains a constant value.

In Fig. 2(a), we investigate the evolution of a more diverse host and parasite populations by considering the initial distributions so that the role of mutations can be minimized in the host–parasite coevolutionary dynamics. We consider two types of distribution for the host population. In the first case, the host population does not contain the genes corresponding to the global fitness peak, whereas in the second case, we consider its distribution in the full range of genotypic space (inset). However, for both the cases, the dynamics of the host–parasite coevolution is obtained by taking the parasite population distribution in the whole genotypic space. Corresponding to these two cases, we present the temporal evolution of the fitness of the host population in the absence and presence of parasites in Fig. 2(b). It can be observed that the qualitative behavior of $\mathscr{F}_H(t)$ remains unchanged for the first case, while for the second case, a different behavior is observed (inset). It is to be noted that in the latter, the optimization time for the case with parasites is marginally higher than the case without the parasites. This is in contrast to the other cases considered so far and indicates that if the genes corresponding to a higher fitness peak is already present in a population, the presence of parasites hinders the evolution of the hosts toward the fitness maximum. Moreover, a single-step relaxation is observed instead of a two-step relaxation, which is expected due to the presence of fittest genes already at $t = 0$.

The time dependence of $m_H(x,t)$ and $m_P(x,t)$ for the case shown in Fig. 2(a) is presented in Figs. 2(c) and 2(d), respectively. In Fig. 2(c), we observe that most of the peaks for $m_H(x,t)$ are already vanishing at $t = 20$, except for the peak near the local fitness maximum. At $t = 300$, the host population is completely localized around the local peak, while at $t = 800$, $m_H(x,t)$ is found to be concentrated around the global peak. A similar behavior is observed for $m_P(x,t)$ as seen in Fig. 2(d).

Next, we investigate the dependence of optimization time τ on host mutation rate μ_H and degree of virulence α, as shown in Figs. 3(a) and 3(b), respectively. From Fig. 3(a), we observe that the time τ is inversely related to μ_H for different α values. The optimization time shows a nonlinear decrease with the increase in μ_H with similar qualitative behavior for all the cases. The time $\ln(\tau)$ is found to be linearly related with $\ln(\mu_H)$ until $\mu_H = 0.9$. This suggests that a decrease in τ with the increase in μ_H follows a power-law behavior $\mu_H^{-\delta}$, where the exponent δ depends on α. The fitting to these plots in the range $\mu_H \in [0, 0.9]$ gives the following relation among τ, μ_H, and α: $\tau = (-710\,\alpha + 285)\,\mu_H^{-(0.37-0.18\,\alpha)}$. We note that the proposed fitting relation is valid only for weak coupling $\alpha \leq 0.1$.

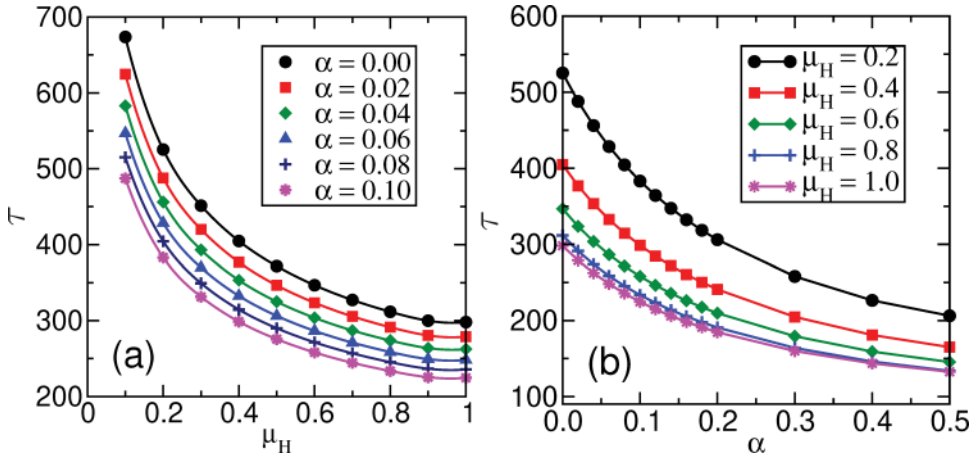

FIG. 3. (a) Optimization time τ vs the host mutation rate μ_H for different degrees of virulence $\alpha = 0$, 0.02, 0.04, 0.06, 0.08, and 0.1 at $\mu_P = 0.9$. (b) Plot of τ vs degree of virulence α at $\mu_H = 0.2$, 0.4, 0.6, 0.8, 1.0, and $\mu_P = 0.9$.

For $\alpha = 0$, the optimization time $\tau \propto \mu_H^{-0.37}$. However, for non-zero α, the exponent is modified by an additional factor dependent on α, though this contribution is negligible for this regime. The origin of linear part of the fitting form is due to the linear decrease in τ with the virulence, i.e., $\tau \propto -\alpha$. The inverse power-law dependence on μ_H is due to the diffusive nature of mutations. The value of power-law exponent for μ_H is determined by the curve fitting.

The dependence of τ on the degree of virulence α for a fixed mutation rate is shown in Fig. 3(b). From this plot, it can be inferred that as the parasitic infection is increased, the time τ required by the host population to reach the global fitness peak decreases. It is also found that the decrease in τ with increasing α is nonlinear. The fitting relation obtained from Fig. 3(a) can be further verified from Fig. 3(b) in the weak coupling regime $\alpha \leq 0.1$. To this end, we investigate the dependence of τ on α by fitting the plots in Fig. 3(b). As a result, we get a general fitting form $\tau = k_0\alpha + k_1 \exp(-k_2\alpha)$, where k_1, k_2, k_3 are fitting parameters (all positive) dependent on μ_H and μ_P. From this, we can access the small α behavior readily and recover $\tau \propto -\alpha$ as obtained previously. We analyze the effect of virulence on the asymptotic fitness of the host population $\mathcal{F}_H(t)$ for different μ_H values in Fig. 4. The findings are consistent with the expectation that the increased parasitic infection will decrease the fitness of the host population. The asymptotic fitness of hosts is found to decrease linearly with the increase in the degree of virulence.

In Fig. 5(a), the fitness of host population $\mathcal{F}_H(t)$ and the fitness of parasite population $\mathcal{F}_P(t)$ are plotted with time t for very high infection obtained by setting $\alpha = 0.4$. For such a high parasitic infection, the fitness of the host population does not reach a fixed value within the simulation time and exhibits an oscillatory behavior. In this case, the host population reaches the global peak of the fitness landscape very fast. However, the parasite population follows the host population to its global peak, and once the parasites reach the peak, the fitness of the host population gets reduced. Due to a high degree of infection from parasites, the effective fitness landscape of hosts changes in a way that the local peak becomes higher than the global peak at that instant. Thus, the host population starts evolving toward the local peak again. Once the host population gets localized around the local peak, the parasites again follow and decrease the fitness of hosts in the local peak. This again triggers the evolution of hosts toward the global peak. Hence, a time-dependent oscillatory fitness for host and parasite populations is observed. We also note that the time interval of oscillation between two successive peaks as well as the amplitude of oscillation decay with time. This only happens beyond a critical value of the virulence parameter α_c for which

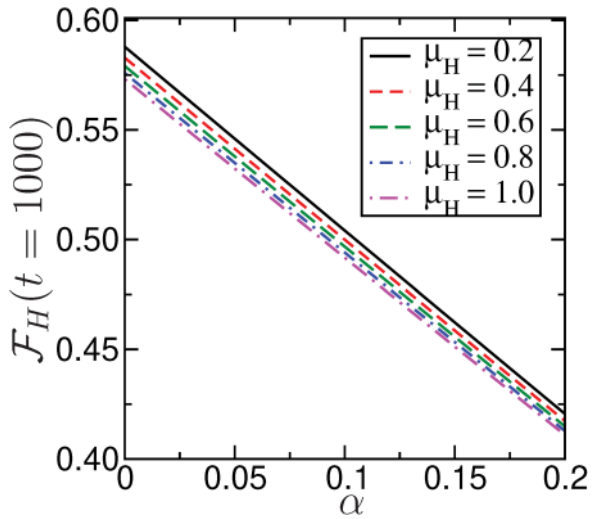

FIG. 4. The fitness of host population $\mathcal{F}_H(t)$ at $t = 1000$ is plotted for α range 0–0.2 at host mutation rates $\mu_H = 0.2$, 0.4, 0.6, 0.8, 1.0, and $\mu_P = 0.9$.

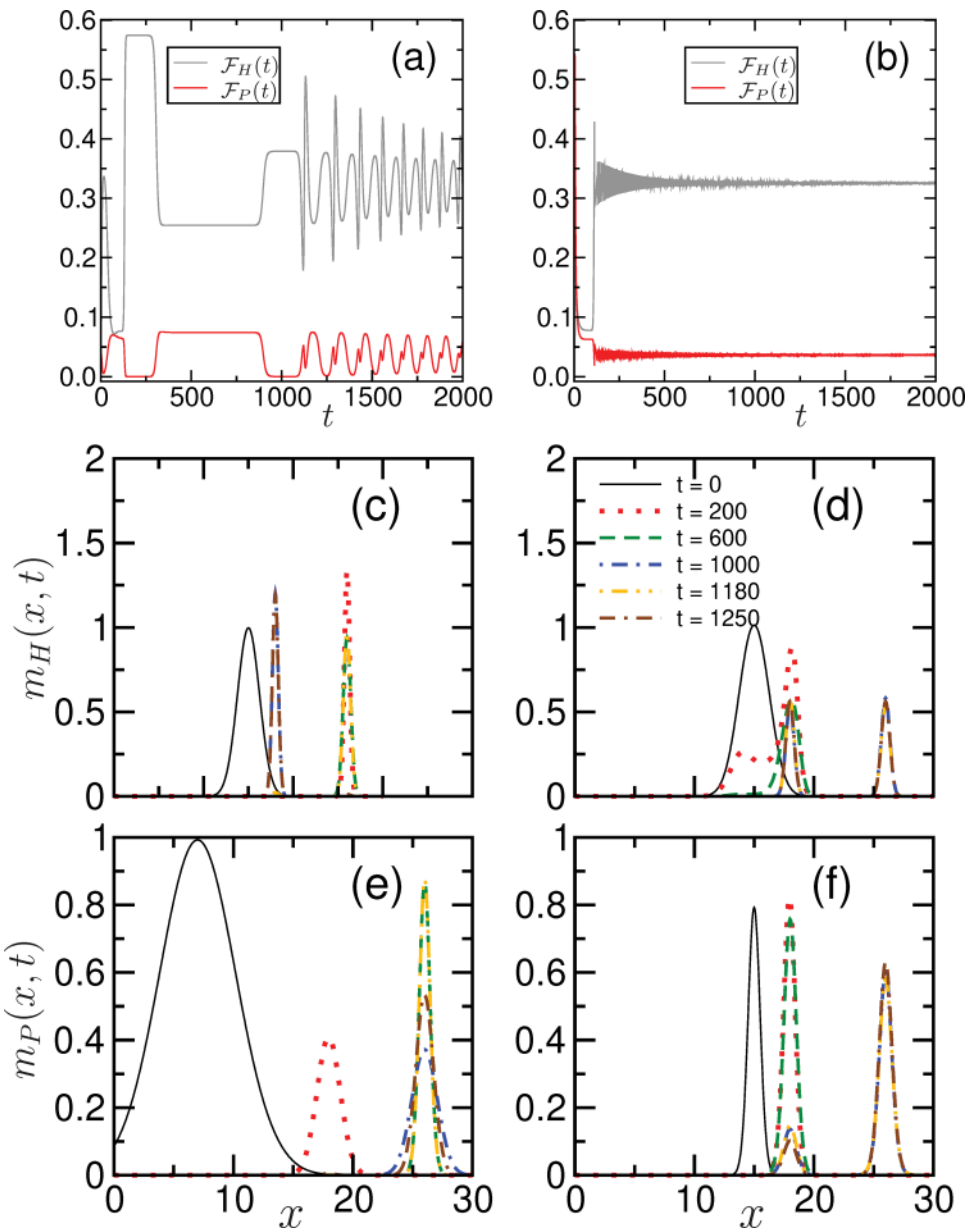

FIG. 5. Fitness of host population $\mathcal{F}_H(t)$ and fitness of parasite population $\mathcal{F}_P(t)$ are shown for parameters $\alpha = 0.4$, $\mu_H = 0.9$, and $\mu_P = 0.9$. (a) Corresponding to the oscillatory regime. (b) Corresponding to the adiabatic limit. The time evolution of distribution of the host population $m_H(x,t)$ is plotted for (c) oscillatory dynamics shown in Figs. 5(a) and 5(d) adiabatic limit shown in Fig. 5(b). Evolution of distribution of parasite population $m_P(x,t)$ for (e) oscillatory regime and (f) adiabatic limit.

the average fitness of the host population around the global maximum is less than the average fitness of the host population around the local peak. In our simulation, we find $\alpha_c \approx 0.24$, which roughly corresponds to the difference in heights of the global and the local peaks.

So far, we have assumed that the hosts and parasites evolve on the same timescales. However, timescales for the evolution of parasites in nature are often found to be faster than their respective hosts, and, hence, in many cases, parasites are found to reach a steady state in between successive generations of hosts. We implement this in our model and refer to this as the adiabatic limit. To achieve this limit in our simulation, we solve the dynamical equation [Eq. (10)] of the parasites for 5000 time units for the evolution of the hosts in one time unit. This essentially means that between each

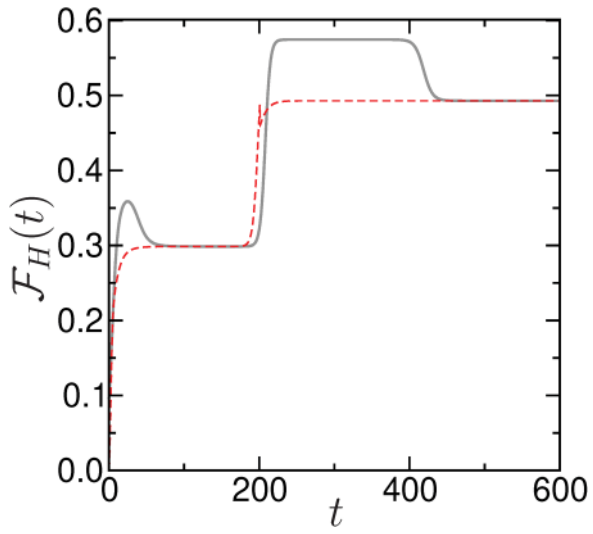

FIG. 6. Evolution of fitness of the host population from the model where hosts and parasites evolve in the same timescale (solid curve) is compared with the adiabatic limit (dashed curve) at $\alpha = 0.1$, $\mu_H = 0.9$, and $\mu_P = 0.9$.

generation of hosts, the parasites have evolved for 5000 generations. The fitness of the host population $\mathcal{F}_H(t)$ and the parasite population $\mathcal{F}_P$(t), in the adiabatic limit, is plotted in Fig. 5(b). The set of parameters chosen for this plot is the same as that in Fig. 5(a). It is observed that when parasites reach a stationary state in between the successive generations of hosts, the fitness of the host population relaxes much faster as compared to the non-adiabatic case [see Fig. 5(a)].

The time dependence of $m_H(x,t)$ for the oscillatory regime and the adiabatic limit is presented in Figs. 5(c) and 5(d), respectively. In Figs. 5(e) and 5(f), we plot the corresponding $m_P(x,t)$. In Fig. 5(c), at $t = 200$, the host population is seen to be already distributed around the global peak. However, the parasite population is still around the local peak [see Fig. 5(e)]. The parasite population $m_P(x,t)$ at $t = 600$ is observed to be completely concentrated around the global fitness maximum. As discussed earlier, this reduces the average fitness of the host population around the global peak. The reduced value of $\mathcal{F}_H(t)$ is less than the fitness attained by the hosts at the local peak in the absence of parasites. Hence, it triggers the evolution of $m_H(x,t)$ toward the local peak as can be seen at $t = 1000$. The parasite population again starts to drift toward the hosts and thus the peak of its distribution around the global maximum reduces (for instance, at $t = 1000$). This again triggers the evolution of $m_H(x,t)$ toward the global peak at $t = 1180$. As expected, the parasites follow [$m_P(x,t)$ at $t = 1180$], pushing $m_H(x,t)$ toward the local peak again [$m_H(x,t)$ at $t = 1250$]. This continues resulting in an oscillatory behavior for $\mathcal{F}_H(t)$ and $\mathcal{F}_P(t)$ as shown in Fig. 5(a). In Figs. 5(d) and 5(f), at $t = 0$, $m_H(x,t)$ and $m_P(x,t)$ are both located around $x = 15$. This is because $m_P(x,t)$ reaches the steady state between the successive generations of the hosts in the adiabatic limit. Similarly, for all other cases, the spatial location of $m_P(x,t)$ is found to be synchronized with that of $m_H(x,t)$.

We also check whether considering parasites in the adiabatic limit further reduces the optimization time τ in Fig. 6. We observe that, indeed, the optimization time τ decreases for the adiabatic case as compared to the non-adiabatic case. Nevertheless, the decrease is not very significant in this parameter regime.

IV. CONCLUSION

In this paper, we have developed a mathematical model to investigate the coevolution of hosts and parasites by incorporating selection, mutation, and asexual reproduction. The dynamical fitness landscapes for the hosts and parasites are constructed in terms of their genotypic distance and frequencies, which are evolving with time. These considerations for a fluctuating selection are in agreement with the existing literature.[35,38,44] It is observed that when the location of a population in genotypic space lies far from its global peak, the role of mutations becomes critical in optimizing its fitness. Next, we studied the evolution of host fitness in the presence of parasites for different mutation rates and varying strengths of parasitic infection. It was found that after the introduction of parasites, the host population can detect the global peak of the fitness landscape more quickly. However, the parasites follow the host population and reduce its fitness, once the latter gets localized around the global maximum. Therefore, faster optimization of the host fitness in the presence of parasites results in lower fitness. We also observe a decrease in the time required by the host population to reach its global peak as the parasitic infection is increased. For a high degree of virulence, the fitness of the host population exhibits an oscillatory behavior. Finally, we discuss the adiabatic limit in which the parasites evolve much more quickly as compared to the hosts. We find that in this limit, the optimization time is reduced even further.

ACKNOWLEDGMENTS

M.P. acknowledges funding from the Department of Science and Technology-Science and Engineering Research Board (DST-SERB), New Delhi, India (Grant No. ECR/2017/003091). We thank Ajay Vikram Singh for fruitful suggestions and discussion. We also thank the anonymous reviewer for constructive comments and suggestions.

DATA AVAILABILITY

The data that support the findings of this study are available within the article.

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