Nonlocal competition and the speciation transition on random networks

Yosef E. Maruvka,[1] Tomer Kalisky,[2] and Nadav M. Shnerb[1]
[1]*Department of Physics, Bar-Ilan University, Ramat-Gan 52900, Israel*
[2]*Department of Bioengineering, Stanford University, Stanford, California 94305, USA*
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A simple model for competition-induced speciation is presented and analyzed. Logistic growth with nonlocal interaction is studied on regular and random networks, and the large scale structure of the emerging genomic frequencies is examined. The neutrality assumption is violated if the network is random and the competition is nonlocal. Instead, "hubs" in the sequence space are suppressed by the competition more than nodes of a lower degree. Thus speciation is unavoidable for large-scale free networks. The emerging genetic mixture depends strongly on the initial conditions. The frequency of hubs is much greater in a population that evolved from a single nucleation event than in a population that recovered from a catastrophe.

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I. INTRODUCTION

The origin of species, i.e., the mechanism that leads to speciation between individuals sharing the same ancestry, is one of the fundamental problems in the theory of evolution. The speciation mechanisms suggested so far are traditionally [1] classified according to the level of geographic separation between subpopulations. If the genetic divergence is attributed to the geographic isolation of a subpopulation, the mechanism is called allopatric. Parapatric speciation occurs when the mixing between geographic locations is partial, while sympatric speciation takes place in a well-mixed population. The most challenging task is to explain sympatric speciation, where the continuous genetic mixing tends to oppose divergence; it is difficult to explain how stochastic dynamics in the genomic landscape, induced by mutations, lead to substantial reproductive isolation.

A minimal model for evolutionary dynamics and speciation [2] regards any organism as a sequence of genes that has some fitness, where fitness is a measure of the organism's reproductive ability. If the set of all possible genotypes is regarded as the network vertices, and a (possibly directed) bond connects any pair of genotypes that may transform into each other by a single mutation, one gets the genotype (sequence) space. The evolutionary dynamics take place on this space, and are affected by its topology and the fitness associated with each genotype. This whole structure—the genotype network plus the fitness function defined on its vertices—is known as the adaptive (fitness) landscape [3].

The classic description of evolution and speciation is based on Wright's idea of local hill climbing, or adaptation, on the adaptive landscape [3]. If peaks in the fitness landscape are associated with highly reproductive genetic sequences, and "valleys" are sequences of low fitness, the population on the hills is growing faster; if the total population is fixed, the favored population takes over, and the "valley" inhabitants undergo extinction. Two species, according to this picture, correspond to two different hills in the adaptive landscape. Speciation is an event of "tunneling" between hills; it occurs when unfit hybrids (whose genotype lies in the adaptive valley) survive the competition with their highly fit ancestors until another set of mutations brings them to the next adaptive peak.

Theoretical studies [4,5] have pointed out many problems with this classic scenario. In particular, since a single peak is, dynamically speaking, an attractive fixed point, the characteristic time for a stochasticity-induced peak shift grows exponentially with the population size. Plugging typical numbers for the fitness variance and the size of the population, results in an unrealistically large time scale (of order 10^{30} generations) for speciation to occur. These results, together with other theoretical problems, led to a major shift in focus, and speciation is currently attributed to random genetic drift on neutral, or nearly neutral, adaptive landscapes. Basically, genotypes are assumed to be either viable or nonviable, and the fitness differences between viable sequences are negligible [6]. The sequence space is considered as an L dimensional hypercube, and one should "subtract" from this structure all the points that correspond to nonviable sequences. If the fraction of viable sequences is not too small, the high connectivity of the sequence space ensures that a single connected cluster of viable points exists and spans the sequence space (technically speaking, the system is above the site percolation threshold [5]). The resulting dynamics are simply stochastic motion (random walk due to mutations) on that spanning cluster. Note that since fitness and viability are correlated in the sequence space, one should not assume that the topology of the spanning cluster is determined by percolation theory; the actual topology of the viable mutations network is, as far as we know, unknown.

While the above model does allow for increasing genotypic diversity within a population, the phenomenon of speciation is still to be explained. Indeed, a mechanism for disruptive selection, i.e., selection against phenotypically intermediate individuals, should be incorporated into the model [7]. Many authors have suggested that the disruptive selection happens due to frequency-dependent competition for a resource; while similar phenotypes (close nodes in the sequence space) interact strongly, dissimilar individuals are not competing for the same resource. As an example, birds with different beak sizes consume different types of seeds.

Dieckmann and Doebelli [8] have suggested that frequency-dependent competition may induce speciation even in the presence of a strong adaptive peak. They consider a one-dimensional sequence space, and associate sequence with trait, such that proximity in the sequence space

reflects similarity in traits. The growth rate is higher for a specific trait and becomes smaller further away from it, such that this trait is the "fittest" in the Darwinian sense. If the global carrying capacity is fixed, the population is localized on the optimal trait. However, if the carrying capacity of a point in the sequence space depends on the population around it (since animals with similar traits compete with each other), the nonlinear interaction may induce bifurcation that leads to bimodal distribution with a *minimum* at the fittest trait, such that the population splits into two different species. The specific model used by [8], with a Gaussian kernel for the competition along the trait axis (i.e., the impact of the population with a given trait on the carrying capacity of populations with different traits decays with the distance like a Gaussian), was subject to criticism, as its steady state solution fails to support speciation [9]. However, non-Gaussian kernels may lead to speciation [10], and even for Gaussian kernels, recent works suggest that speciation may result from environmental [11] or demographic [12] stochasticity.

The emerging picture, thus, is that sympatric speciation may be modeled on the viable sequence network as a simple growth process with *nonlocal competition*, i.e., with competition between genotypes that are neighbors in the sequence space. The crucial assumption underlying disruptive selection is the dependence of the strength of the competition on the genetic distance. We stress that to achieve speciation, competition should be neither local nor global. Local competition refers to the scenario in which only individuals with the same genotype compete for resources. In such a case, all viable sequences will be populated. On the other hand, if *global* competition is assumed, i.e., if the mutual competition between genotypes is independent of their genetic distance (like in the Eigen-Schuster quasispecies model [13]), the system either allows a unimodal steady state (when the system is below the error threshold), or fails to adapt (when the system is above the error threshold). Only nonlocal (yet not global) competition may induce speciation. For a certain range of parameters population peaks are "disconnected," and the chance to find an individual with an intermediate genotype in the region between peaks is negligible.

In recent years, many works discussed the conditions under which segregation appears in a model of logistic growth with nonlocal competition [14–21]. The only difference between these models and the speciation model described above is the topology of the underlying network. While [14–21] dealt with Euclidian geometry, the viable mutations landscape is most likely some sort of random network. The aim of this work is to generalize [14–21] to the case of random networks of different types, and to understand the connection between the surviving genotypes and the local topology of the network. We restrict our discussion to the effect of frequency-dependent competition, and regard the population as asexual, thus avoiding the discussion of assortative mating and related processes.

It should be stressed that once the competition is nonlocal, random networks of viable sequences are not neutral anymore, even if the fitness of all vertices is the same. The reason is that the number of links is now a factor that affects the fitness. If a site has a large number of links, this implies strong competition and a lower carrying capacity. Thus "hubs" of the network are suppressed by the competition, while low-degree nodes admit larger carrying capacity. Here we intend to show the consequences of this competition-induced bias on the genetic diversity of the population.

In the following section, we review the known results about logistic growth with nonlocal competition on a lattice and generalize them to the case of graphs and trees with a constant number of links per node. In the third section, we consider the growth process on the two prototypes of random networks, namely the Erdős Rényi and the scale-free topologies. In the last section, we discuss the relevance of our findings for the study of speciation.

II. LATTICES, TREES, AND GRAPHS

Let us first introduce the results previously found for the lattice version of our model [14–21]. The simplest, one-dimensional realization is described by

$$\frac{dc_n}{dt} = ac_n - c_n \sum_j \gamma_j c_{n+j} - \sum_j \mu_j (c_n - c_{n+j}), \tag{1}$$

where c_n is the population density on the nth site (this corresponds to the abundance of individuals with a certain sequence denoted by n), γ_j corresponds to the strength of the competition with the population j sites away (where j takes both positive and negative values, and its absolute value is the genetic distance), and μ_j corresponds to the rate of mutations, such that a replication of sequence n results in genome $n+j$. Note that the mutation rate is much smaller than the growth rate, indicating that the loss of population due to mutations to nonviable genotypes is negligible. If $\gamma_j = \delta_{j,0}$, the system admits only local competition. In such a case, local initiation results in Fisher-Kolmogorov-Petrowskii-Piscounov (FKPP) invasion [22], and the steady state is uniform, $c_n = a$. If the competition is nonlocal, the system may undergo speciation transition where the steady state allows for filled and empty sites. In cases of nearest-neighbor competition ($\gamma_0 = 1$, $\gamma_{\pm 1} \equiv \gamma$, all other competition terms vanish) the critical value of γ may be calculated [not only for Eq. (1) but also for its d-dimensional generalization]:

$$\gamma_c = \frac{1 + 4\mu d}{2d(1 - 2d\mu)}. \tag{2}$$

Below this value the steady state remains uniform, while above this value local initiation yields a checkerboard ($0a0a0a0a\cdots$) stable solution, i.e., the speciation transition takes place at γ_c.

An important distinction should be made between local and global initiation [20]. If the system is initiated locally by a small group of founders with the same genome, or at least with limited genetic diversity (which may occur, e.g., if the first establishment of a living colony on a certain island happens due to immigration of a small number of individuals with minimal genetic diversity, or, in a petri-dish experiment, when a colony emerges from a single bacterium), its dynamics yield a perfect checkerboard configuration. If, on the other hand, the system initiation is global (as in the case of

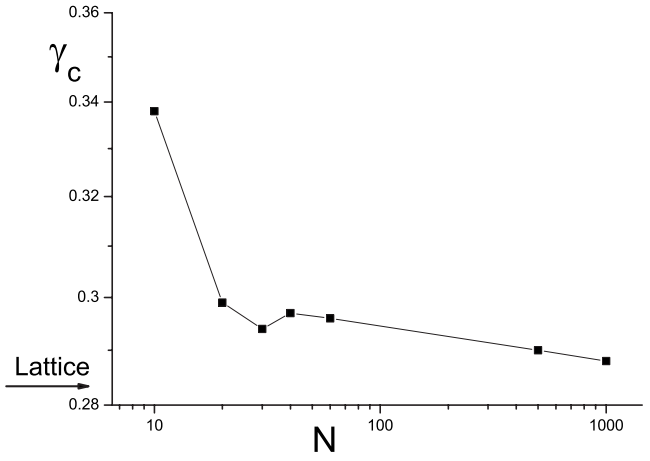

FIG. 1. Speciation transition point in a regular graph (with degree $k=4$) vs network size, averaged over five configurations. The model parameters are $a=\gamma_0=1$, $\mu=0$.

immigration of a diverse community to an island, or recovery of a colony after a catastrophe), the dynamics may lead to *frustration*. In the latter case, the checkerboard configuration may admit different phases (e.g., empty odd sites vs empty even sites) on different regions of the lattice, and the steady state supports different domains connected by domain walls, or even glasslike structures [20].

The lattice dynamics of Eq. (1) may be generalized to an arbitrary topology of the viable mutations network:

$$\frac{dc_n}{dt} = ac_n - c_n \sum_{j\in\Omega_\gamma^n} \gamma(n,j)c_j - \sum_{j\in\Omega_\mu^n} \mu(n,j)(c_n - c_j), \quad (3)$$

where Ω_μ^n and Ω_γ^n are the sets of genotypes to which species n can mutate, or with which they compete for resources, correspondingly. In our simple model, genotypes mutate to nearest sites on the network and competition also takes place only between nearest neighbors, such that the sets Ω_μ^n and Ω_γ^n reflect the local topology of the network.

The simplest modification of the lattice model is a viable sequence space with a topology of a Cayley tree. In a Cayley tree, every node has the same number of neighbors, k, and there are no loops. For weak competition, a homogenous stable solution exists, and Eq. (3) becomes $\dot{c}=ac-c^2-k\gamma c^2$, with the solution $c=a/(1+k\gamma)$. As γ grows, this homogenous solution becomes unstable, and the system reaches the "up-down" state, where the tree is segregated into an alternating structure of empty and filled layers. This occurs at $\gamma_c=(1+2\mu k)/[k(1-k\mu)]$, which is the same result we obtained for a lattice with dimensions $d=k/2$.

It is well-known that large graphs with random connections are very similar to trees, as the number of small loops is negligible [23]. Thus, for fixed k graphs, one should expect that as N becomes large, the critical value of γ will approach the corresponding value for a tree, with k branches for each node. This feature is demonstrated in Fig. 1. Note that the convergence of the regular graph with the tree behavior is very slow, since only loops smaller than the diameter of the network $[\mathcal{O}(\log N)]$ disappear as the network size grows. The convergence in Fig. 1 is thus logarithmic, and for small networks, the deviation from the tree/lattice value is still large.

III. RANDOM NETWORKS

Now let us proceed to the more realistic case, where k is not fixed, i.e., where the number of viable mutations for a certain sequence is fluctuating. Two generic examples of such networks are the Erdős Rényi (ER) and the scale-free (SF) networks [24]. For an ER network the degree of connectivity k is taken from a Poissonian distribution with average k_0, while for a scale-free network the probability $P(k) \sim k^{-\alpha}$ decays like a power law at large k.

Random networks differ qualitatively from regular networks. In random networks the homogenous state may exist only without any nonlocal competition. As soon as $\gamma>0$, no homogenous solution can satisfy the corresponding set of differential equations. Assuming $c_j=c_0$, the time dependence of the population with sequence j is given by

$$c_j = ac_0 - c_0^2 - k\gamma c_0, \quad (4)$$

where k is the degree of the jth node. Obviously, this equation may have a nonzero solution only for $\gamma=0$; for any nonlocal competition the solution should admit some sort of dependence on k, the degree of the node. This is the mathematical manifestation of the fact discussed above, namely, that for networks with nonuniform degree distribution and nonlocal competition, the neutrality assumption breaks down.

Still, weak competition implies that all the nodes with the same degree will admit almost the same population. In fact, one can formulate a mean-field theory, based on this assumption, and solve for the steady-state population as a function of the degree k. This mean-field theory is presented in the Appendix, and for low γ the results show very good agreement with the simulations, as emphasized in Fig. 2, for both ER and SF networks.

Technically, the region of parameters for which the mean-field theory works should be viewed as the random network analog of the uniform solution for regular networks. As explained above, it is impossible to attain a homogenous solution on a random network; the mean-field calculation assumes that nodes with the same degree have the same environment and yields the same c_k for all the k nodes. As demonstrated in Fig. 2, population size decays continuously with k, so only hubs with a very large k maintain a population below a reasonable threshold (since the real number of animals is an integer, a population below some threshold should not be considered). For Erdős Rényi and small scale-free networks this implies no speciation; all nodes are connected and no empty regions separate between two quasispecies. For large SF networks, on the other hand, the removal of hubs will produce a disconnected network, as in the case of an intentional attack considered by [25].

As the competition coefficient increases, the frequency variance for nodes of the *same* degree grows, and eventually nodes with the same k are segregated into a group of surviving (full) nodes and a group of extinct (empty) nodes, as

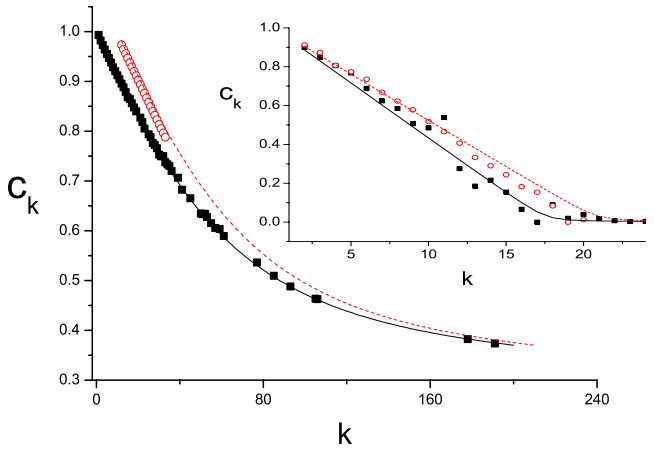

FIG. 2. (Color online) Mean-field predictions (lines) vs simulation results for scale-free (solid line: theory, squares: simulation) and Erdős Rényi (dashed line and circles) networks. The number of nodes is $N=2000$; other parameters are $a=1$, $\gamma_0=1$, $\mu=0.0041$, and $\gamma=0.01$. The ER results are shifted (by 10) to the right to avoid overlap with the SF results. The inset shows the mean-field predictions vs the simulation results for a network with $N=800$ nodes, both for the SF [solid line (squares)] and for the ER [dashed line (circles)]. Parameters are $a=1$, $\gamma_0=1$, $\mu=0.0001$, and $\gamma=0.1$.

exemplified in Fig. 3. The mean-field approximation clearly fails in that parameter regime. This segregation transition within nodes of the same degree affects first the hubs, but as competition increases, the speciation occurs for lower and lower degrees. This is the random network state that corresponds to the "spiky phase" on a lattice [20].

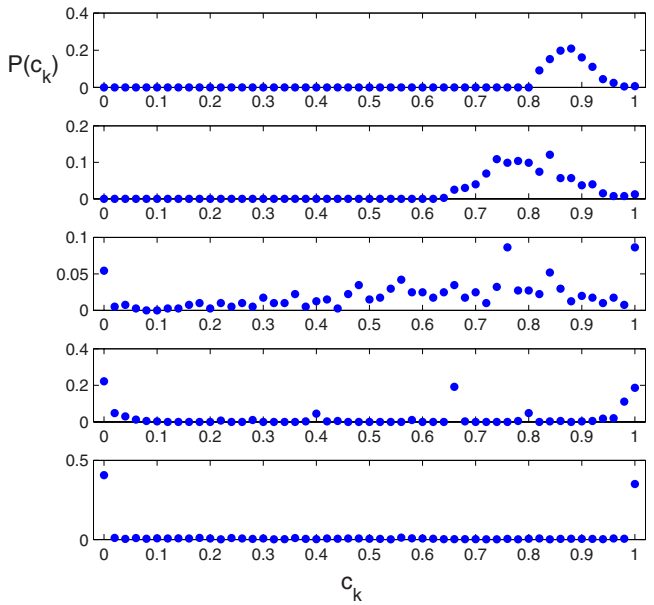

FIG. 3. (Color online) A series of histograms showing the occupation distribution of nodes with degree $k=4$ (on the same network) with different competition coefficients. From top to bottom, the fraction of nodes with population c, $P(c_k)$, is shown for $\gamma=0.05$, 0.1, 0.3, 0.5, and 1. For weak competition, the histogram allows a peak around the value predicted by the mean-field theory. For strong competition, the nodes are either "alive" or admit no population at all.

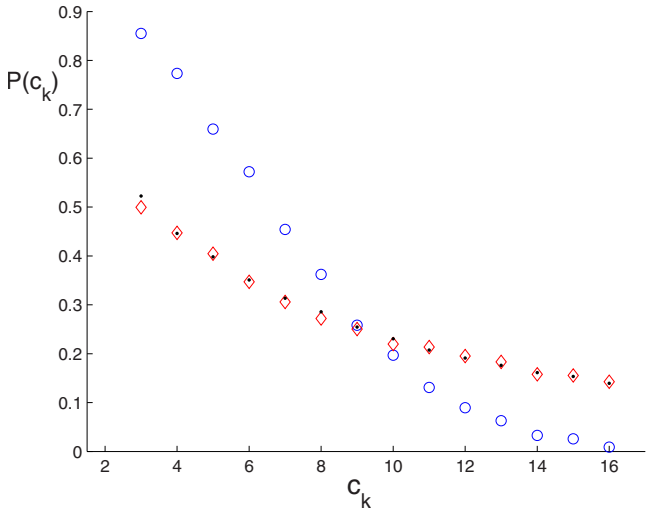

FIG. 4. (Color online) The average population per degree for ER networks with global (circles) and local initiations (diamonds for random degree, dots for largest hub initiation). Results presented are an average of 20 different realizations, with five different initial conditions for global initiation, 20 for local initiation, and one for the largest hub. The parameters are $N=800$, $\langle k\rangle=12$, $a=1$, $\gamma_0=1$, $\mu_j=0.0041$, and $\gamma=0.01$.

In that spiky phase, there is a surprising distinction between local initiation (i.e., one node with its maximal carrying capacity and all the others empty) and global initiation (i.e., all the nodes with a small random population). It turns out that for global initiation the probability of a large node to survive is much smaller than in the case of local initiation, as demonstrated in Figs. 4 and 5. This feature is independent of the degree of the node around which the initial condition is localized. Starting with a populated hub, or with a populated node of a lower degree, the same dependence of the population on the degree of the node is obtained.

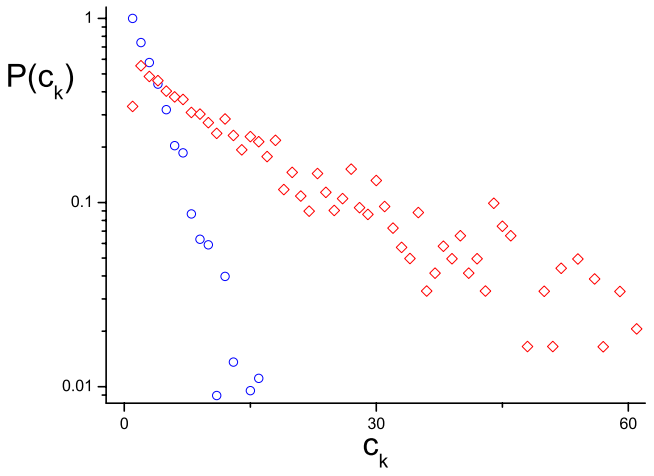

FIG. 5. (Color online) The average population per degree for SF networks, with global (circles) and local (diamonds) initiations. The average was taken over six different realizations, with five different initial conditions for global initiation, and 60 initial conditions for random degree local initiation. The parameters are $N=800$, $P(k)\sim k^{-2.5}$, $a=1$, $\gamma_0=1$, $\mu_j=0.0041$, and $\gamma=0.01$.

IV. DISCUSSION

The concept of fitness in the theory of evolution has to do with the performance (survival and reproduction) of an individual (i.e., a phenotype) in a given environment. The environment of a single organism is determined by extrinsic factors, such as the availability of food, air, sunlight, and other necessities. The neutral theory of evolution [26] in its various forms assumes that the differences between viable individuals are negligible, and that the main limitation on the growth of a population is its competition with other, equally fitted, populations. Within this framework, sympatric speciation takes place when the genetic drift is accompanied by strong frequency-dependent competition, where the "strength" is measured with respect to the mutation rate. In this paper, we have tried to illustrate the large scale structure of the genetic polymorphism in the emerging population.

One should bear in mind that even if the assumption of neutrality is wrong in general, it perhaps still holds for certain parts of the genome. An implementation of the theory presented here does not require a global sequencing of the whole genome, and is even inadequate in that case, as no loops appear in that limit. A sequence of $\mathcal{O}(10)$ nucleotides or regions in the genome is more than sufficient for the analysis suggested here. Moreover, we have examined only the steady-state properties of the population. If the whole genome is the subject of the study, the system never equilibrates; small sequences, on the other hand, may reach equilibrium within a reasonable number of generations.

The present work suggests three qualitative insights regarding examination of polymorphism data collected from a population at a steady state.

(1) The criteria for speciation is generally not sharp, and depends on both the network's topology and history.

(2) The population density on the "hubs" is smaller than the population on the "dead ends." The more links a point admits, i.e., the more viable mutations it possesses, the more it suffers from competition.

(3) The chance to find an existing "hub" (to sample individuals with the corresponding genotype) is much greater for the case of local initiation than for the case of global initiation. This last criterion may be utilized to recover the history of a colony. Specifically, it may help one to distinguish between a colony that grew monotonically from a founder and a colony that recovered from a catastrophe.

The extent to which one can quantify these qualitative considerations depends on the resolution of certain technical obstacles. The main problem seems to be the reconstruction of the viable mutations network.

If all possible genotypes appear with, say, Poissonian fluctuations, one should assume that part of the sequence is irrelevant for single organism fitness and that the competition is more or less global. If the population is localized around a few genotypes, implementation of our results requires a knowledge of the structure of the viable mutations network. Distances should be measured along that network. The Hamming distance is meaningless since the shortest path may pass through a nonviable genotype.

If the number of sequenced individuals is large enough though, one may use the fluctuations induced by demographic stochasticity in order to make a distinction between nonviable mutations and the genetic configurations prohibited by competition. In that case, the qualitative power of our results may be extracted. With the current vast growth of genetic data, one may hope that such an implementation will be possible in the near future.

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APPENDIX

In this appendix, we use the mean-field approximation to solve the steady state of the population, as a function of the degree on a network. Assuming that all the nodes with the same degree admit the same population, and that all the nodes of the same degree "see" the same environment on average, Eq. (1) may be written as

$$c_k = c_k - bc_k^2 - k\gamma c_k \Theta_k + \mu k(\Theta_k - c_k), \tag{A1}$$

where Θ_k (this notation has been used also by [27,28]) is the average population of the nearest neighbors of a node with degree k. If the network is uncorrelated, i.e., the degree distribution of the neighbor profile of a node with degree k is independent of node degree, on average all the nodes see the same surroundings, and one obtains $\Theta_k = \Theta$ for all the possible k's. The explicit expression of Θ is

$$\Theta = \frac{\sum_{k_{min}}^{\infty} (kP_k c_k)}{\langle k \rangle}, \tag{A2}$$

where kP_k is the probability that an arbitrary link leads to a node with degree k. c_k is the average population on a node with degree k, and $\langle k \rangle$ serves as the normalization factor.

With that, Eq. (A1) becomes

$$c_k = ak_n - bc_k^2 - k\gamma c_k \Theta + \mu k(\Theta - c_k). \tag{A3}$$

In a steady state where the left-hand side of that equation vanishes, one can solve for c_k at a given

$$c_k = \frac{a - \gamma k\Theta - k\mu + \sqrt{(a - \gamma k\Theta - k\mu)^2 + 4bk\mu\Theta}}{2b}. \tag{A4}$$

The two equations (A3) and (A4) are then solved self-consistently for Θ to yield c_k for all the k's.

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