Dynamic transitions in small world networks: Approach to equilibrium limit

Prashant M. Gade*
Centre for Modelling and Simulation, University of Pune, Ganeshkhind, Pune, 411 007, India

Sudeshna Sinha†
The Institute of Mathematical Sciences, Taramani, Chennai 600 113, India
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We study the transition to phase synchronization in a model for the spread of infection defined in a small world network. It was shown [Phys. Rev. Lett. **86**, 2909 (2001)] that the transition occurs at a finite degree of disorder p, unlike equilibrium models where systems behave as random networks even at infinitesimal p in the infinite-size limit. We examine this system under variation of a parameter determining the driving rate and show that the transition point decreases as we drive the system more slowly. Thus it appears that the transition moves to $p=0$ in the very slow driving limit, just as in the equilibrium case.

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I. INTRODUCTION

The dynamics of spatially extended systems has been very well studied in the past two decades. On the other hand, in the recent past, the importance of studying networks, their structure, and properties has been realized, and researchers from fields ranging from neurodynamics and ecology to social sciences have been extensively working in this area [1–4]. In particular, small world networks [2] have been studied in many different contexts. This model is defined in the following way: One starts with a structure on a lattice—for instance, k regular nearest-neighbor connections. Each site is now linked with $2k$ of its nearest neighbors on either side. For nonzero p, we rewire all the links with probability p; i.e., the site is disconnected from the site within distance k and is connected to a randomly chosen lattice site which could be anywhere on the lattice. This model is proposed to mimic real life situations in which nonlocal connections exist along with predominantly local.

It has been observed in these systems that starting from a one-dimensional chain at $p=0$, one obtains long-range order at any finite rewiring probability with the same critical exponents as in the mean-field case. Moore and Newman recover critical exponents for percolation on small world lattices which are the same as for the Bethe lattice—i.e., an infinite-dimensional case [5]. For the XY model, Medvedyeva *et al.* conjecture that critical exponents are the same as for the mean-field case [6]. They have confirmed it for $p \geqslant 0.03$, and there is good reason to believe that it is true for any $p>0$. (The obvious difficulty is that one needs to simulate larger and larger lattices at small p.) Similar conclusions are reached for the Ising model in a small world network [7]. This strongly suggests that the behavior for any $p \neq 0$ is the same as the behavior for $p=1$ for these models.

Dynamical systems are nonequilibrium systems, and in general it would not be very surprising if they have different behavior [8,9]. In fact for dynamic transitions in nonequilibrium models there is evidence of transitions at finite p. For instance, the transition to self-sustained oscillations evident in a model of infection spreading occurred at finite p [8].

Here we will try to *identify the conditions under which we could expect the behavior of nonequilibrium or dynamical systems to be similar to that observed in equilibrium models*. As a case study we use the model of infection spreading showing finite-p transitions, mentioned above. First we discuss the model in detail in Sec. II. Then in Sec. III we study the model with respect to a parameter determining the driving rate of the system. We show how very slow driving leads to transitions at $p \to 0$, as in equilibrium models. We conclude in Sec. IV with a discussion.

II. MODEL OF INFECTION SPREADING

We consider the SIR-susceptible (SIRS) model (The labels S and I stand for susceptible and infectious state of individuals in population while R stands for the refractory state in which individuals are recovered with temporary immunity.) of infection spreading on a lattice. We take a graph of N vertices on a one-dimensional lattice. Each vertex has $2k$ connections. Each site i is connected to sites $i+1, i+2, \ldots, i+k$ on the right side and $i-1, i-2, \ldots, i-k$ on the left side when $p=0$. We assume periodic boundary conditions. For $p \neq 0$ we rewire these $2k$ connections with probability p and connect site i to a randomly chosen site j on the lattice. Each site i is assigned value $\tau_i(t)$ at time t. The variable $\tau_i(t)$ can take integer values from 0 to τ_0. If $\tau_i(t)=0$, the site i is considered susceptible at time t. If $\tau_I \geqslant \tau_i(t) \geqslant 1$, it is considered infected, and if $\tau_i(t) > \tau_I$, it is considered to be in the refractory stage at time t. For sites which are not susceptible—i.e., $\tau_i(t) \neq 0$—the dynamics is simple:

$$\tau_i(t+1) = \tau_i(t) + 1 \quad \text{if } 1 \leqslant \tau_i(t) \leqslant \tau_0 - 1$$

and

$$\tau_i(t+1) = 0 \text{ if } \tau_i(t) = \tau_0.$$

*Electronic address: gade@unipune.ernet.in
†Electronic address: sudeshna@imsc.res.in

The dynamics does not depend on the neighbors if the site is not susceptible. Neighbors come into question only while infecting the susceptible site. The model assumes that only infected sites infect their neighbors. Thus a site susceptible at time t will be infected at time $t+1$ with probability proportional to the fraction of infected sites in its neighborhood. In other words, if $\tau_i(t)=0$, $\tau_i(t+1)=1$ with probability $p_i=k_{inf}/k_i$ where k_i are the total number of neighbors of site i, of which k_{inf} are infected. With probability $1-p_i$, the susceptible site does not change state. The dynamics for the infected sites is deterministic. The infected sites slowly become refractory and then eventually become susceptible again.

Kuperman and Abramson simulated the above model on a small world lattice [8]. They observe that the fraction of infected sites at a given time t shows oscillations in time for a large value of p. One can view the system as a sum of many interacting clusters, and at large values of p, these clusters get synchronized to each other, giving collective oscillations. It was reported in [8] that this transition to synchronization indeed occurs at a finite value of p and the transition becomes sharper in the thermodynamic limit.

We note the following fact about phase-synchronized oscillations. If all of them become truly synchronized, they will reach a value of zero at the same time, and since there are no infected sites in the lattice, infection will die down. If we assume that τ_0 is not fixed but has a distribution, it still does not guarantee that the absorbing state will be avoided. If the system that falls is a state in which $\tau_i(t)=0$ for all i, it stays in this state forever unless there is some external source of infection. To avoid this state, we make a small change in the model. We add *quenched disorder* or sources of infection [10,11]. We choose 1% of the total number of sites and keep them in the infectious state forever—i.e., $\tau_i(t)=\tau_i(0)$ for all these sites for all times and $\tau_i(0)=1$. This guards system against falling into a fully synchronized state where there is no further evolution [10]. The results in the section below are obtained from our modified SIRS model with quenched disorder.

III. RESULTS

Specifically we study the behavior of the infected sites with respect to $\tau_I+\tau_R\equiv\tau_0$, which determines the rate of driving in this model. We see pronounced fluctuations in the number of infected sites as a function of time. These fluctuations are periodic with the natural period τ_0, which is the time scale for a susceptible site, if infected, to become susceptible again.

Figure 1 shows the time evolution of the fraction of infected elements, for $p=0.1$, for a particular realization after discarding a long transient. The figure displays four cases with varying values of τ_0 (keeping the ratio τ_I/τ_R fixed). Time has been scaled by the natural time scale τ_0 so that results from different choices of τ_0 can be easily compared. It is clear that as τ_0 increases the collective oscillations become more pronounced. These oscillations essentially indicate the presence of cycles in the outbreak of disease.

For a small fraction of nonlocal connections p, these oscillations are seen only if τ_0—i.e., natural time scale for the disease—is fairly large. On the other hand, for a higher p, even smaller τ_0 yield collective oscillations in the number of infected individuals at a given time. An intuitive reasoning could be given as follows. For larger τ_0 the information that a given site is infected can propagate more, since the site stays infected for a longer time. A similar role is played by large p, as the information that a given site is infected spreads over several sites in a very small time if one has a lot of nonlocal connections. This sharing of information leads to collective phenomena like periodic excitations appearing spontaneously in the system. Since higher τ_0 and p play similar roles, one can expect that for higher τ_0, we will start seeing collective oscillations even at small p.

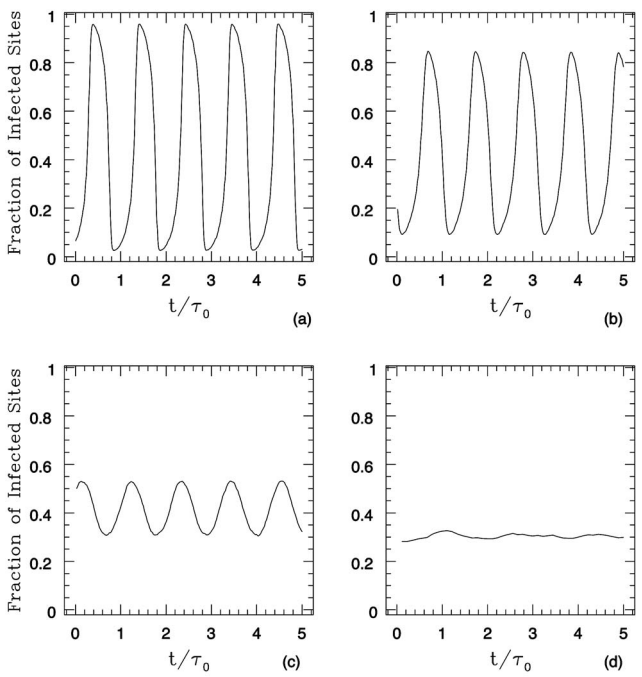

FIG. 1. Fraction of infected sites vs t/τ_0 for a system of size 10 000, for (a) $\tau_R=144$, $\tau_I=64$, $\tau_0=208$, (b) $\tau_R=72$, $\tau_I=32$, $\tau_0=104$, (c) $\tau_R=36$, $\tau_I=16$, $\tau_0=52$, and (d) $\tau_R=9$, $\tau_I=4$, $\tau_0=13$. The value of p is 0.1.

As an illustrative example of the similar roles played by high p and high τ_0 consider the following: the number of nonlocal connections are certainly important in the spread of disease, as the outbreaks can affect locations far apart geographically, but time scales also play a role. For instance, Ebola is far more deadly virus than HIV and kills the host much faster as it has a much shorter incubation period. However, due to the very fact that it kills so swiftly, Ebola outbreaks are contained very soon. The people infected by Ebola die very quickly, and so the virus has less time to jump to a new host and spread the disease. If no new victims come in contact with the body fluids of infected people in their short lifetime, the epidemic stops. On the other hand, HIV remains a problem worldwide since victim lives longer and has longer time to infect others [12].

To see this quantitatively, we study the synchronization parameter, which is the relevant order parameter here. This is defined as

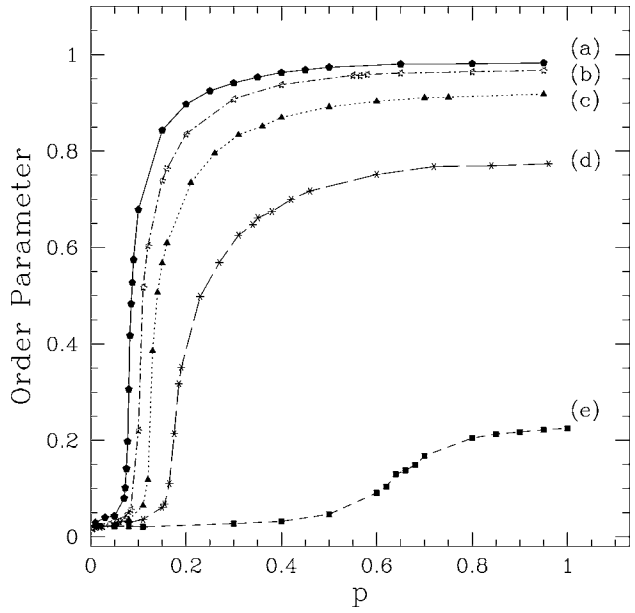

FIG. 2. Order parameter [defined in Eq. (1)] vs p for a system of size 10 000, for (a) $\tau_R=144$, $\tau_I=64$, $\tau_0=208$, (b) $\tau_R=72$, $\tau_I=32$, $\tau_0=104$, (c) $\tau_R=36$, $\tau_I=16$, $\tau_0=52$, (d) $\tau_R=18$, $\tau_I=8$, $\tau_0=26$, and (e) $\tau_R=9$, $\tau_I=4$, $\tau_0=13$.

$$\sigma(t) = \left| \frac{1}{N} \sum_{j=1}^{N} \exp^{i\phi_j(t)} \right|, \tag{1}$$

where $\phi_j = 2\pi(\tau_j - 1)/\tau_0$ is a geometrical phase corresponding to τ_j. The states $\tau=0$ are left out of the sum [8]. As mentioned above, 1% of the sites are quenched in the infectious state during the time evolution. We choose initial conditions in which 10% of the total sites are in the infected state. The sites which are not quenched evolve according to the rule mentioned above. We average over 120 configurations for $N=10^4$ and compute the above order parameter after waiting for 2.5×10^4 time steps.

When the system is not synchronized, the phases are widely distributed and the value of $\exp^{i\phi}$ is spread widely over the unit circle. This leads to small σ. On the other hand, when the elements are synchronized, σ is large. If all elements are strictly synchronized, σ will be 1.

Figure 2 shows the synchronization parameter σ obtained as a time average of $\sigma(t)$ over 1000 time steps. Subsequently we also average over different realizations of the system. The different curves are obtained for different values of τ_0. A transition in synchronization can be observed as p runs from 0 to 1. This transition occurs at values closer to 0 as τ_0 increases. We must mention that we also carried out the same calculation for $N=10^5$ where we averaged over 20 configurations and waited for 9×10^4 time steps. As in the case of the original system, we observe that there is no qualitative change as we do simulations for larger system size, except that the transition becomes sharper.

The original authors postulated that the transition to collective oscillations in infected individuals could be related to the dependence of clusterization in the network on the probability p of nonlocal connections. Clusterization $C(p)$ is a quantifier of the number of closed triangles in the network—i.e., how many neighbors of a given site i are also neighbors of each other. It is averaged value over all sites and normalized to its maximum possible value. (See footnote 12 of [8].) Unlike the average path length, the $C(p)$ decreases slowly as a function of p and there is an intermediate regime where there is a low average clusterization $C(p)$ for a given p though the distribution of clusterization at element level $c_i(p)$ is rather broad. They find that this is precisely the regime when the onset of collective oscillations occurs. However, in this work, we studied the dynamics of the system for different values of τ_0. This change does not alter topology of underlying network and hence does not affect $C(p)$ or dispersion around it. But the transition is certainly affected. The fact that the transition can be seen without changing topology of underlying network suggests that *time scales also play an important role in this transition apart from structure of the network on which the dynamics is taking place*.

IV. CONCLUSIONS

We studied the transition to phase synchronization in a model for the spread of infection defined on a small world network. It was shown in [8] that the transition occurs at a finite degree of disorder p, unlike equilibrium models where systems behave as random networks even at infinitesimal p in the infinite-size limit. We examined this system under variation of a parameter determining the driving rate and showed that the transition point decreases as we drive the system more slowly. Thus it appears that the transition moves to $p=0$ in the very slow driving limit, just as one expects in the equilibrium case.

Some earlier studies may also be interpreted in this light. For instance, it was observed that the transition point of the finite p transitions to synchronization in coupled chaotic maps decreases to $p=0$ as the chaoticity of the local map (which determines the time scales of information loss) decreases [9]. This can be seen to reflect the fact that a transition at $p\to0$ is obtained when the rate of Lyapunov exponent $1/\lambda$ tends to zero. The characteristic time scale for information loss in a chaotic system varies as $1/\lambda$. So as the time scale reaches infinity, the transition point goes to zero. We also note Fig. 2 in our previous paper [13]. There we have plotted the power spectra of the collective field for small world lattices at different values of p. We note that high-frequency (i.e., short-time-scale) peaks are seen only at large values of p, while low-frequency (long-time-scale) peaks are seen even at small p. Thus there is a clear interplay between the probability of nonlocal connections p and the time scales in the system. This suggests that in an extended parameter space one can find dynamic transitions at infinitesimal p, as in the equilibrium case, in the very slow driving parameter limit.

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