Emergence of epidemics in rapidly varying networks

Vivek Kohar and Sudeshna Sinha
Indian Institute of Science Education and Research (IISER) Mohali,
Knowledge City, Sector 81, SAS Nagar, PO Manauli 140306, Punjab, India

We describe a simple model mimicking disease spreading on a network with dynamically varying connections, and investigate the dynamical consequences of switching links in the network. Our central observation is that the disease cycles get more synchronized, indicating the onset of epidemics, as the underlying network changes more rapidly. This behaviour is found for periodically switched links, as well as links that switch randomly in time. We find that the influence of changing links is more pronounced in networks where the nodes have lower degree, and the disease cycle has a longer infective stage. Further, when the switching of links is periodic we observe finer dynamical features, such as beating patterns in the emergent oscillations and resonant enhancement of synchronization, arising from the interplay between the time-scales of the connectivity changes and that of the epidemic outbreaks.

I. INTRODUCTION

Complex networks provide the framework in which to model a wide class of phenomena, ranging from engineered systems to biological systems [1-3]. Now, a prototypical dynamical network involves equations describing the time variation of state variables at the nodes or sites, and a set of links capturing the essence of the connection of a node with subsets of nodes in the system. Importantly, the links in the network could be *static* or *dynamic*. Static or annealed links imply that the connectivity is invariant or quenched throughout the evolution of the system [4]. Dynamic links on the other hand imply that the underlying connections may switch around, and so the nodes couple to changing environments. Studying the implications of such dynamic links is relevant, for example, in a socio-economic network, where the connectivity matrix generically changes over time.

In this work we will explore how changing the underlying web of connections at different rates influences the emergent spatiotemporal patterns in an extended interactive system. Specifically, we will focus on a problem of considerable relevance, namely, the nature of infection spreading in a population of individuals connected by links that vary over a large range of time scales.

At the level of individuals in the population, we consider the class of communicable diseases that progress as follows: at the outset an individual is *susceptible* to infection (a stage denoted by S); on infection through contact with other infected people, the individual moves to the *infectious* stage (I). In this stage of the disease, an individual may infect susceptible members of the population it comes into contact with. The infectious period is followed by a *refractory* stage (denoted by R), where the individual is immune to the disease and also does not infect others. The immunity in this class of disease is temporary and after a while the individual is again susceptible (S). So the temporal evolution of the stages of the disease at the nodes of the network will be modeled by the well known epidemiological model of disease progression: the SIRS cycle. This model is appropriate for diseases like small pox, tetanus, influenza, typhoid fever and cholera [7].

Now, we consider such individuals linked together in a web of connections. Various approaches have been employed to analyze such disease dynamics on networks. While some studies have focused on different network topologies [5, 8] others have analyzed using different rules for dynamics of diseases on the nodes. For example, Girvan et. al. studied how the variation in recovery times can result in different dynamical behaviours [9]. In [10], Nagy studied the impact of varying contagion scheme, time delay and infection probability on uncorrelated networks in the fast rewiring and annealed limits. Other studies have focussed on adaptive networks, where the evolution of the topology of the network depends on the dynamics on the nodes. Gross *et. al.* [12] observed assortative degree correlation, oscillations, hysteresis, and first order transitions in SIS model of epidemics employing an adaptive strategy in which susceptible are able to avoid contact with the infected by rewiring their network connections. Similar strategy leads to bistability of the endemic and disease free states in the SIRS model [14]. Segbroeck *et. al* considered disease spreading as a stochastic contact process embedded in a Markov chain and found that adaptive networks in which information about health status of others is available, can be considered as well mixed population with a rescaled effective disease infectiousness [15].

In contrast to earlier studies [5]-[15], we incorporate *changes in the underlying connectivity at varying timescales*, ranging from fast to slow vis-a-vis the nodal disease dynamics. Further, at a time, not all links get rewired; rather, a fraction of the regular contacts are replaced by random interactions. This is most relevant, as some of our connections change rarely, like family and close friends, whereas others change much more rapidly, such as strangers in our work place or in public spaces. The important consequence of disease spreading on time varying networks that we will demonstrate in the subsequent sections is the following: *quick changes in the connections enhance synchronization, as compared with slow network changes.* Namely, epidemic outbreaks emerge

in rapidly varying networks, while slowly changing links result in a low fluctuating state of endemic infection.

The organization of this paper is as follows: First, in section II, we describe our model of the disease cycle and infection spreading. In Section III we present the results obtained from extensive simulations of this dynamical network. In Section IV we present the phenomena arising in a probabilistic rewiring model, namely the scenario where the connections switch to random sites randomly in time. We conclude with discussions in Section V.

II. MODEL OF INFECTION SPREADING

We consider a network of N nodes on a ring, where each node (vertex) has $2K$ directed connections (edges). Consider first a completely regular network with each site i connected to sites $i \pm 1, i \pm 2, \ldots i \pm K$ on either side. On this regular network we incorporate random rewiring, with probability p. That is, when $p > 0$, we shuffle the connections with probability p, replacing some regular connections with a few random links. With probability p, site i will then be coupled to a randomly chosen site j on the ring. So large p implies that there will be many "short-cuts" connecting neighbourhoods, with parameter p interpolating between the regular lattice at $p = 0$ and a random network at $p = 1$ [2].

Now each node in this network represents an individual whose disease progression is described by a cellular automata model of the SIRS cycle [5]-[6]. The details of this model are as follows: each node i is assigned a value $\tau_i(t)$, which evolves over 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 susceptible at time t. If $\tau_I \geq \tau_i(t) \geq 1$, it is infected and if $\tau_i(t) > \tau_I$ it is in the refractory stage at time t. So τ_I is the time during which a node remains infected after inception of infection, and τ_0 is the total length of the full disease cycle. For sites which are not susceptible, i.e. $\tau_i(t) \neq 0$, dynamics is given as:

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

and

$$\tau_i(t+1) = 0 \quad \text{if} \quad \tau_i(t) = \tau_0 \tag{2}$$

The dynamics does not depend on the neighbours if the site is not susceptible. Neighbours come into play only while infecting the susceptible site. The model considers that only infected sites infect their neighbours. 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 the probability $q = k_{inf}/k_i$ where k_i are total number of neighbours of site i, of which k_{inf} are infected. With probability $1 - q$, the susceptible site does not change state.

$$\tau_i(t+1) = \begin{cases} 1 & \text{with pobability } q, \\ 0 & \text{with probability } 1-q \end{cases} \quad \text{if} \quad \tau_i(t) = 0$$

We also introduce a small quenched disorder in the system, namely ~ 1 % of the total number of sites is always kept in the infectious state i.e. $\tau_i(t) = \tau_i(0)$ for all these sites for all times and $\tau_i(0) = 1$. This prevents the system from falling into fully synchronized state, after which there can be no further evolution.

So this system has both deterministic and probabilistic features. The disease progression for the infected site is *deterministic*, with the infected site slowly becoming refractory and then eventually becoming susceptible again, thus going through a prescribed cycle. However, the inception of the disease cycle is a random event, as the infection of susceptible nodes occur with a certain probability that depends on the state of the neighbours.

Simulations of the above model on a small world lattice [5] showed that the fraction of infected sites at a given time t shows oscillations in time for a large random rewiring probability p. One can view the system as an union of many interacting clusters. When p is large these clusters get synchronized to each other, giving rise to large amplitude collective oscillations, which can be identified as *epidemic outbreaks*.

However, it is obvious that the connectivity of the individuals will most likely vary over time, even if the average number of connections and types of links remain the same. In order to investigate this issue, we consider the underlying connection matrix to switch between different realizations, having the same fraction of random links. We consider two types of varying networks: periodically rewired networks and probabilistic link switching.

So we simulate the SIRS disease cycle on networks of sizes upto $N = 10^5$, and we looked for the effects of switching links on the emergence of synchronized infection. First the system dynamics is investigated qualitatively, through inspection of the time series of the size of the infected set in the network. Then, we go on to characterize quantitatively the transition to large scale disease outbreak, through an order parameter reflecting the degree of synchronization of the individual disease cycles at the nodes, defined as [5]:

$$\sigma(t) = |\frac{1}{N}\Sigma_{j=1}^{N} \exp^{i\phi_j(t)}| \tag{3}$$

where $\phi_j = 2\pi(\tau_j - 1)/\tau_0$ is a geometrical phase corresponding to τ_j. Now, the occurrence of large oscillations corresponds to a spontaneous synchronization of a significant fraction of the elements in the system, implying that the phases $\tau_i(t)$ in the nodal disease cycles become synchronized and individuals progress through the disease together, becoming ill at the same time and recovering at the same time. Thus in this case σ will be large, with $\sigma = 1$ when all nodes are completely synchronized. On the other hand, when the system is not synchronized, the

phases are widely distributed. So the value of the complex numbers $\exp^{i\phi}$ will be spread widely over the unit circle, leading to small σ.

We present in the sections below, the infection patterns emerging from our extensive simulations on different kinds of rewired networks.

III. PERIODICALLY SWITCHED LINKS

First we consider the scenario where the network changes occur periodically, at some time period denoted by r. We study the influence of different rates of network change, ranging from links switched at every step in the disease cycle, to networks changing after several disease cycles. As we scan the full range of random rewiring probability p and time period of network change r, we look for the emergence of large oscillations in the number of infected nodes in the network, suggestive of epidemic outbreaks in the population. Our observations are presented in the subsections below.

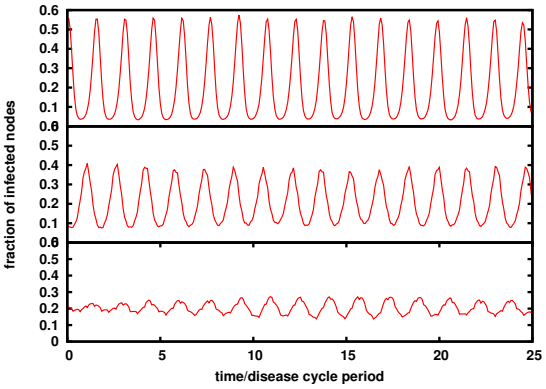

Figure 1. Evolution of the number of infected sites in a network of 10^4 nodes with $K = 1$, with network rewiring periods $r = 1$ (top), $r = 3$ (middle) and $r = 5$ (bottom). Here the random rewiring probability $p = 0.4$, and $\tau_I = 4$, $\tau_0 = 13$ in the SIRS disease cycle.

A. Enhancement of Synchronization in rapidly varying networks

The principal observation, from our extensive simulations, is the following: for any given fraction of random links, when the network varies fast, the oscillations in the total number of infected sites have large amplitude, indicative of more synchronized disease outbreaks. This is evident in the time evolution of the fraction of infected nodes in the population, shown for representative cases in Fig. 1.

Further, quantitatively, it is evident from inspection of this synchronization order parameter, that when the

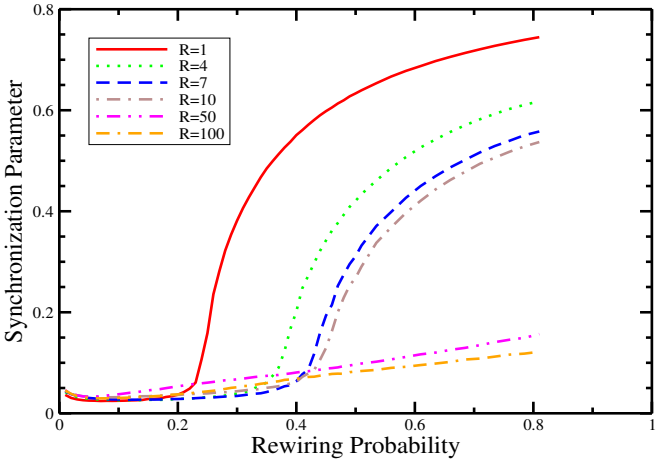

Figure 2. Variation of the synchronization order parameter with rewiring probability p, for a network of size $N = 10^4$, and $K = 1$, for different network rewiring periods. Here $\tau_I = 4$, $\tau_0 = 13$ in the SIRS disease cycle.

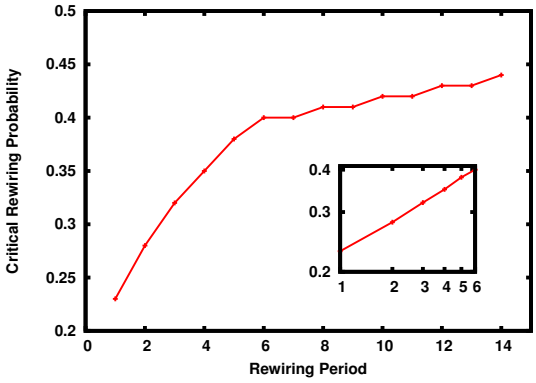

Figure 3. Variation of critical rewiring probability p_c, with respect to network rewiring time period r, for a network of size $N = 10^4$, and $K = 1$. Here $\tau_I = 4$, $\tau_0 = 13$ in the SIRS disease cycle. Inset shows the curve on a log-log scale, for low r values, indicating power-law scaling at small r.

frequency of network change is low, the rewiring probability at which transition to large oscillatory behaviour occurs, increases. For instance, in Fig. 2, the transition to large-scale synchronization occurs around $p = 0.2$ when the network varies with time period $r \sim 1$. In contrast, when the network changes slowly ($r \sim 10$) then the transition to synchronized disease outbreak occurs only for much larger random rewiring probabilities ($p > 0.4$). For very slow rewiring ($r \sim 50$) the transition doesn't occur at all.

For small r there is a clearly defined transition to the synchronized state as the fraction of random links increases. We obtain the transition point from the sharpest change in the slope of the curve of the synchronization order parameter as a function of rewiring probability p.

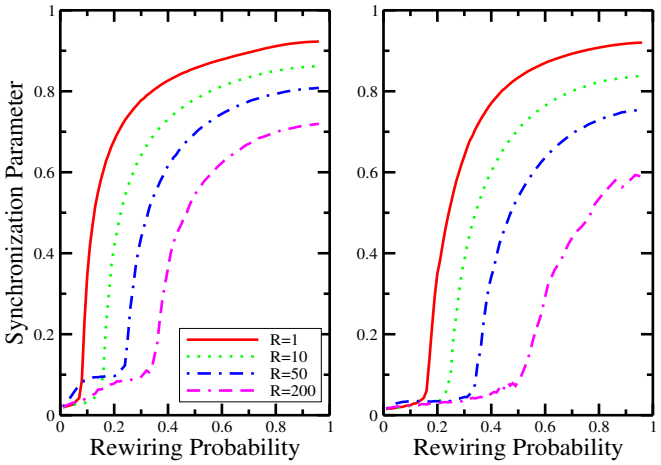

Figure 4. Variation of the synchronization order parameter with rewiring probability p for a network of size $N = 10^4$, $K = 1$, $\tau_0 = 26$, and infectious period $\tau_I = 8$ (left), $\tau_I = 16$ (right) for network different rewiring periods.

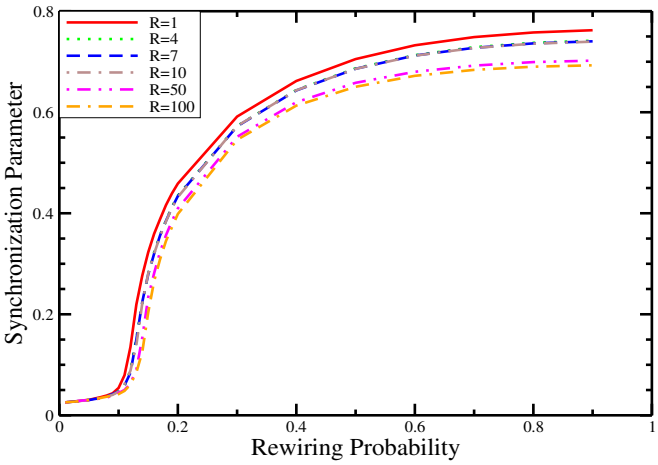

Figure 6. Variation of the synchronization order parameter with rewiring probability for a network of size $N = 10^4$ and $K = 5$, for various network rewiring periods. Here $\tau_I = 4$, $\tau_0 = 13$ in the nodal SIRS disease cycle.

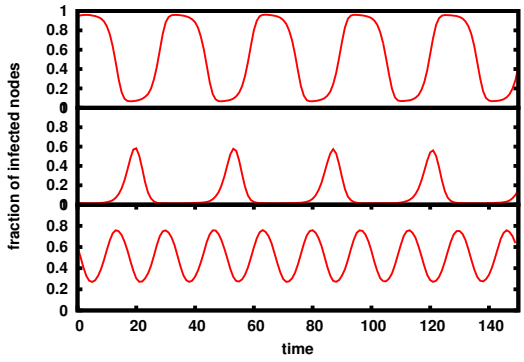

Figure 5. Evolution of the number of infected sites in a network of 10^4 nodes with $K = 1$, $p = 0.4$ and network rewiring period $r = 1$, for nodal SIRS cycles with: $\tau_I = 16$, $\tau_0 = 26$ (top), $\tau_I = 4$, $\tau_0 = 26$ (middle) and $\tau_I = 8$, $\tau_0 = 13$ (bottom).

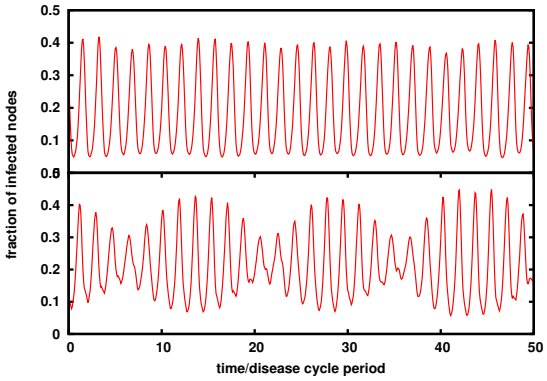

Figure 7. Evolution of the number of infected sites in a network of 10^4 nodes, with $K = 1$, for network rewiring period $r = 23$ and rewiring probability $p = 0.5$ (top), $p = 0.7$ (bottom). Here $\tau_I = 4$, $\tau_0 = 13$ in the SIRS disease cycle.

The critical rewiring probability p_c thus obtained, with respect to the network rewiring time period r, is displayed in Fig.3. It is evident that p_c increases with increasing network rewiring period r. Namely, for rapidly changing networks, i.e. with small r, we obtain synchronization at smaller values of rewiring probability p.

B. Interplay of nodal dynamics and network rewiring

The significant nodal time-scale here is τ_I, namely the time over which a node can infect others. If the network connections change rapidly compared to τ_I, it strongly aids synchronization. However, if the underlying web of links changes slower than τ_I, the effect on the emergence of synchronized cycles is much lower. So while a node is in the infective stage, if the network changes often, shortcuts are provided to many different non-local nodes in the network. Thus the infection can spread much faster, assisting the emergence of large-scale synchronization.

The above also implies that the effect of rewiring is more evident when the infective stage is longer. This is demonstrated in Fig. 4, which displays the synchronization order parameter for networks that rewire at different frequencies, with respect to the probability of random rewiring p. It is clear that for the larger τ_I the onset of large scale synchronization is most affected by the rate of change of the underlying connections.

Interestingly, we observe that the time period of the emergent oscillations in infected nodes, $T_{epidemic}$, is larger than the length of the disease cycle τ_0. For in-

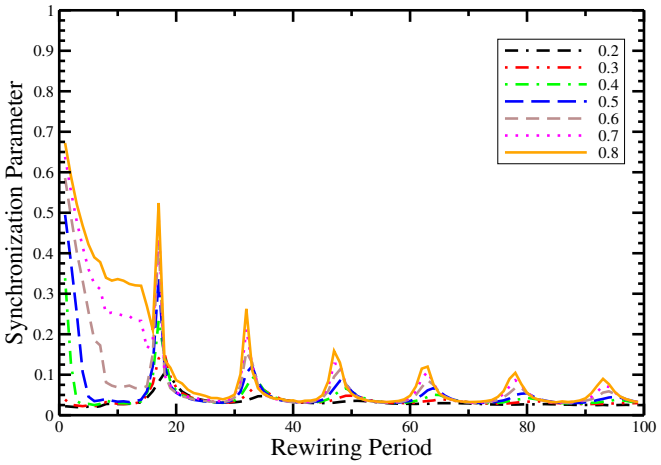

Figure 8. Variation of the synchronization order parameter with network rewiring period r, for a network of size $N = 10^4$, with $K = 1$, $\tau_I = 8$, $\tau_0 = 13$ and different rewiring probabilities.

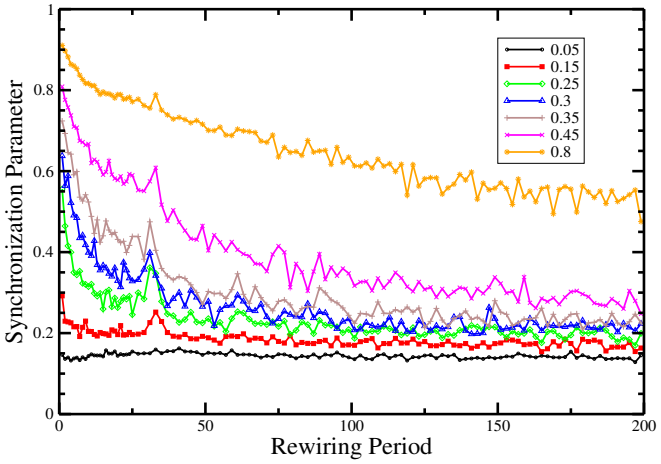

Figure 9. Variation of the synchronization order parameter with network rewiring period r, for a network of size $N = 10^4$, with $K = 1$, $\tau_I = 16$ and $\tau_0 = 26$ for different rewiring probabilities.

stance, for $r = 1$, if τ_0 is 13, then the time period of the collective infection is around 18 (as evident from Fig. 5), and when τ_0 is 26, $T_{epidemic}$ is around 32.

Further, if the length of the infective stage τ_I increases, $T_{epidemic}$ decreases slightly and the magnitude of oscillations increases, indicating the emergence of faster and more pronounced oscillatory infection outbreaks (see Fig. 5). $T_{epidemic}$ also decreases slightly with increase in rewiring probability p. Lastly, the time period of the collective infection oscillations is weakly dependent on the size of the network. For instance, for a network with 100 nodes the period is approximately 18, while for a network of 10^4 nodes $T_{epidemic} \sim 23$.

We simulated the system for various possible combinations of τ_I and τ_0, by varying τ_I from 2 to 40, and τ_0 from 10 to 50. The results were found to be qualitatively similar over the entire spectrum.

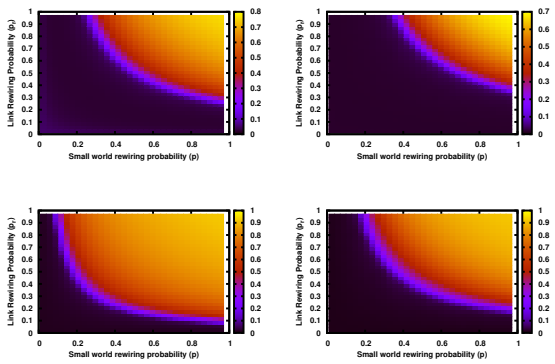

Figure 10. Variation of the synchronization order parameter in the parameter space of small world rewiring probablity (p) and link rewiring probablity (p_r) for a network of size $N = 10^4$, with $K = 1$ and $\tau_I = 4$, $\tau_0 = 13$ (top left), $\tau_I = 8$, $\tau_0 = 13$ (top right), $\tau_I = 8$, $\tau_0 = 26$ (bottom left) and $\tau_I = 16$, $\tau_0 = 26$ (bottom right). Again notice that the enhanced synchronization due to rewiring is more evident when the infective stage is longer.

C. Influence of neighbours

Now we investigate the effect of increasing number of neighbours on disease outbreaks. If the number of neighbours is large, namely K is large, then the effect of changing links on synchronization is less significant. This is evident in representative examples displayed in Fig. 6, from where it is clear that the *system with larger number of neighbours is less sensitive to network changes.*

One can argue that this arises from the fact that the loss in information spreading speed due to slower rewiring has been compensated by large number of links. One can also rationalize this by considering the limit of very large number of neighbours ($K \to N/2$), where the connectivity matrix has no effect on the dynamics, as the coupling is all-to-all.

At the level of nodal dynamics note that the time period decreases as the number of neighbours increases, or if the fraction of random links increases. This trend is anticipated, as more random neighbours will increase the probability of infection.

D. Fine structure in emergent oscillations

Interestingly, over and above the broad trends mentioned above, we also observe some *fine structure in the oscillations.* These arise from the complex interplay of the changing the random links and the emergent epidemic cycles $T_{epidemic}$. The competition and cooperation between the underlying processes of infection and interaction gives rise to "resonances" in the system.

For instance, these different simultaneous periodic influences lead to *beating patterns* in the oscillations in infected population. This is clearly observable in the representative example displayed in Fig. 7, where the frequency of the envelope of the amplitude modulation is proportional to the difference in the emergent infection outbreak frequency and the network rewiring frequency. At specific values of p, the two periodic influences become comparable in strength, and so the beating patterns are most pronounced at these values. Such emergent beating patterns have bearing on the phenomenology of dynamically changing networks in general.

Further we find quantitative evidence of resonant increase in synchronization occurring when the network rewiring time period is multiples of the oscillatory epidemic outbreak time period $T_{epidemic}$ (see Fig. 8 and 9). So, while we may generally expect that as we keep on decreasing the frequency of switching links, the epidemic outbreak will reduce, there may be a *sudden increase in the number of infected individuals when the network rewiring period r comes close to the frequency of emergent oscillations $T_{epidemic}$.*

To demonstrate this explicitly we took two different disease cycle lengths τ_0: 13 and 26. For the case of $\tau_0 = 13$, an increase in the amplitude of oscillations was observed around network rewiring period 17 (Fig. 8), which is close to the time period of the emergent epidemic oscillations $T_{epidemic}$ (~ 17). For the case of the longer disease cycle $\tau_0 = 26$ (Fig. 9), where $T_{epidemic}$ is larger (~ 30), increased synchronization occurred at multiples of a larger network rewiring period (namely $r \sim 30, 60, 90$). Further, we checked the generality of these qualitative features over a range of network sizes and varying lengths of the complete disease cycle and the infective stage of the disease cycle.

IV. PROBABILISTIC SWITCHING OF LINKS

Now, we expect to see the resonance-like fine structure *only* in scenarios where the links are switched together at regular time intervals, for instance in a situation where the connections are determined by a global external periodic influence. However this is not always the most realistic scenario for disease spreading, as the interaction patterns usually don't change periodically in time. Rather we must consider a probabilistic model of link switching, such as in [18]- [19]. So in this section we study such randomly switched networks in order to determine which emergent features are robust to the manner in which links change, and which phenomena are specific to periodically switched links. Namely, we verify the generality of our observations above by investigating the SIRS dynamics on a network whose underlying links switch randomly asynchronously in time.

Specifically now, at each instant of time, a node has the probability p_r (the "link rewiring probability") of its connections being rewired. Further, as above, a node rewires to its nearest neighbours with probability $1 - p$ and to some random neighbour with probability p (the "small world rewiring probability").

We observe transitions to synchronized epidemic cycles here as well. Further, when the link rewiring probability is increased, namely when the links change more frequently we obtain greater synchronization amongst the disease cycles in the system (see figure 10). This indicates the generality of the central observation: changing links induces stronger synchronization in the SIRS disease progession, leading to the emergence of epidemics.

The only significant difference between connections varying periodically and probabilistically, is the absence of resonance-like features in the synchronization order parameter. This is expected, as there is no time scale in the random switching case that may interplay with the periodicity of the disease cycle to create "resonances".

So we conclude that the enhancement of synchronization under varying links is a robust and general phenomena. However, resonances may be observed only when there is regularity in the link switching, perhaps driven by external periodic influences.

V. DISCUSSIONS

Here we discuss some broad, and possibly speculative, potential applications of these results. Our observations above suggest an adaptive strategy where individuals need not be quarantined for long periods, nor isolated till they fully recover. Rather, as is intuitively obvious, infected individuals just need to be quarantined during their infective phase, namely over the time during which they can infect others. More importantly, our study suggests that large scale epidemics in the population can be prevented by simply ensuring that the infected do not swap their links for some time i.e. the infected set can retain the small set of contacts they already have, and just not be allowed to *change* their contacts.

Such a strategy is in contrast to most prevalent strategies which entail full quarantine and complete deactivation of links with the infected, which in real networks is difficult to implement and prone to failure due to inaccurate real time information on the infection status of the individuals [21]. On the other hand, if the infected can retain their set of existing contacts, it will have a positive effect on their well being, as they will be able to maintain some interactions, such as with close family, while diseased. Such a scenario will have positive implications for the psychological health of the infected, which in turn is likely to have a positive effect on their physical health, as suggested by many studies on the close interaction between the two. Also, it is easier to simply not change existing links, rather than obtain real time information of other individuals and deactivate links accordingly.

Notice that small local neighbourhoods that change rapidly, as modeled by fast switching networks with nodes of low degree, may also mimic real life scenarios

better than large fixed neighbourhoods. The reason for this is that generically we come in close proximity to only few individuals at a time, though the set of individuals we interact with may change quite frequently. Further, all interactions with neighbours may not be capable of transferring an infection. It is particularly relevant in case of sexually transmitted diseases where the number of neighbours is low for vast majority of population and only contacts involving sexual partnership can be counted as edges [16]. This has analogues in other scenarios, such as spread of computer virus, as well.

Further, the analysis here can also be used to identify which groups are more vulnerable to an outbreak. For example children tend to change their connections more rapidly compared to adults [3]. Thus it may happen that an epidemic occurs only among the sub-population of children and not among adults.

In conclusion, we have described a simple model mimicking disease spreading on a network with dynamically varying connections, and we have investigated the dynamical consequences of switching links in the network. Our central observation is that the disease cycles get more synchronized, indicating the onset of epidemics, as the underlying network changes more rapidly. Further, for periodic switching of links, we observed dynamical features arising from the interplay of the time-scales of the network changes and that of the emergent synchronized infection oscillations. Lastly, we discuss some possible implications of our results on potential epidemic management strategies.

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