18th International Conference on Information Fusion
Washington, DC - July 6-9, 2015

Adaptive Dynamics, Control, and Extinction in Networked Populations

Ira B. Schwartz
US Naval Research Laboratory
Code 6792
Nonlinear System Dynamics Section
Plasma Physics Division
Washington, DC 20375
Email: Ira.Schwartz@nrl.navy.mil

Brandon Lindley
Daniel H. Wagner Associates, Inc.
2 Eaton Street, Suite 500
Hampton, VA 23669-4054
Email:brandon.lindley@va.wagner.com

Leah B. Shaw
College of William and Mary
Department of Applied Science
Williamsburg, VA 23817-8795
Email:lbshaw@wm.edu

***Abstract*—Real networks consisting of social contacts do not possess static connections. That is, social connections may be time dependent due to a variety of individual behavioral decisions based on current network links between people. Examples of adaptive networks occur in epidemics, where information about infectious individuals may change the rewiring of healthy people, or in the recruitment of individuals to a cause or fad, where rewiring may optimize recruitment of susceptible individuals. In this talk, we will review some of the dynamical properties of adaptive and random networks, such as bifurcation structure and the size of fluctuations. We will also show how adaptive networks predict novel phenomena as well as yield insight into new controls.**

Applying a new transition rate approximation that incorporates link dynamics, we extend the theory of large deviations to stochastic network extinction to predict extinction times. In particular, we use the theory to find the most probable paths leading to extinction. We then apply the methodology to network models and discover how mean extinction times scale with network parameters in Erdos-Renyi networks. The results are shown to compare quite well with Monte Carlo simulations of the network in predicting both the most optimal paths to extinction and mean extinction times.

I. Introduction

Network models when coupled with individual social behavior have increased the understanding of the dynamics of populations. Hardware and technology coupling with populations have shown how society can understand network phenomena such as information spreading dynamics, epidemiology, and terrorist cell analysis [1], [2], [3], [4], [5], [6].

Much previous work on social dynamics assumed homogeneous populations, where real social structure was lacking. Most of these models were compartmental and were similar to mean field models of stochastic simulations. Modeling of social interaction that constantly arises is one of mass action, which accounts for one or social contacts between individuals. It is most evident in the modeling of epidemics, where infection spread in a population arises from direct contact between healthy and sick individuals. Another mass action modeling class of interest to defense is that of recruitment of susceptible individuals by the terrorist cell networks [6].

One current and future trend of network modeling is to consider adaptive behavior, or social response in the population to information about a current or future threat [7], [8], [9], [10]. Previously, we discussed some of the adaptive network models used in infectious disease and terrorist recruitment modeling, and how individual social adaptation may change the dynamics of the networks, which in turn alters the progression of disease or recruitment [11].

In epidemic models where the population is well-mixed (globally coupled individuals), extinction of infectious individuals has been shown to be affected by noise intensity and other factors [12], [13], [14]. Moreover, since the extinct state is typically unstable in the deterministic mean field and is an absorbing state of a stochastic process, time scales for extinction may be exponentially long [15]; i.e., the probability of extinction is a decreasing exponential function [16].

Vaccination and treatment programs have been studied to speed up the extinction of disease in well-mixed populations [17], [18], [19]. For example, although most vaccination schedules are designed to be administered periodically (deterministic) [20], [21], [22], Poisson distributed scheduling was recently shown to be more efficient than regular treatment schedules [23].

In network populations, outbreak extinction probabilities have been predicted for early times when an infection has just been introduced [24], [25]. Other studies of extinction on networks attempt to predict whether a persistent non-extinct state exists, such as for computer viruses in growing networks [26] and epidemics in various network geometries (e.g., [27]).

Here we consider the problem of epidemic extinction in stochastic and adaptive networks, and we find that the extinction process depends not just on the nodes of the network, but also on how the links change as the system evolves. That is, along the most probable path to extinction, we have derived a new approximate model showing that both nodes and links play an important role in the mean time to disease eradication. We have introduced a novel mathematical tool so that the path is derived constructively. (See [28] for details. In addition, we also show how some of the network properties in an adaptive network behave dynamically, such as time dependent link oscillations and degree.

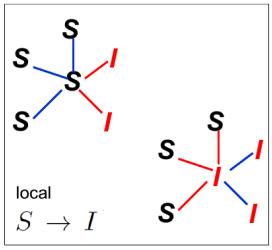

Fig. 1. An example of a local transition where $S \to I$. Notice that even though a single S node changes to I through a contact with an infectious node, more than one SS links convert to SI links.

II. SIS on a Random Network

The specific example we will consider here is a network of N nodes and K links, with an average degree of $2K/N$. The dynamics on the network is an **SIS** epidemic model, where susceptibles capable of acquiring the disease become infectious through a contact with an infective individual, and become susceptible again after a recovery period. Here we divide the population into two groups, with S denoting susceptible individuals and I infected individuals, such that the total population is fixed at N. The population is closed, and there are no births and deaths. We consider the state space of node and link numbers given by the vector $\mathbf{X} = [N_S, N_I, N_{SS}, N_{SI}, N_{II}]$, where N_A,N_{AB} denote the number of A nodes and AB links respectively.

We assume there are three state transitions: $S \to I$ along the network (local); $S \to I$ globally throughout the network; and $I \to S$ which represents recovery. These transitions all come with rates or probabilities of the event occurring. However, the transitions also require incremental values which designate how many nodes and links change. For global networks, both rates and transitions are known since the population is assumed to be well-mixed [18]. However, for networks having local structure, a change in a single node may result in a large change of link numbers, as shown in Fig. 1.

One of the problems in describing dynamics for stochastic networks is that it suffers from the curse of dimensionality which arises from exponential growth of memory and computational requirements. However, given that we have a known solution for the globally connected network supporting an SIS model, we will take a perturbative approach. In doing so, we introduce a homotopy parameter, ϵ, such that when $\epsilon = 0$, the solution is that of global network, and when $\epsilon = 1$, the solution corresponds to the network structure with local coupling having structure, such as average degree.

We approximate the dynamics of the network by considering transitions in links, similar to the pair-based proxy model of [29]. We assume large but finite population size, N, and we suppose the dynamics proceeds as a Markov process. To complete the formulation for the network dynamics, we suppose there exist $M = 3$ transition events with transition rates $W(\boldsymbol{X}, \boldsymbol{\nu}_k)$ having increments $\boldsymbol{\nu}_k$. To model the transition between nodes, we let p, r denote the infection and recovery rates.The transition rates as functions of ϵ are:

$$\begin{aligned} W(\boldsymbol{X}, \boldsymbol{\nu}_1) &= \epsilon p N_{SI} \\ W(\boldsymbol{X}, \boldsymbol{\nu}_2) &= (1-\epsilon) p \frac{2K}{N} \frac{N_S N_I}{N} \\ W(\boldsymbol{X}, \boldsymbol{\nu}_3) &= r N_I, \end{aligned} \tag{1}$$

where transmission is along the network, global transmission, and recovery, respectively. We have introduced the homotopy parameter ϵ which continuously transforms the system between the global and network transmission models.

Using the logic in [28] allows us to write the transition increments for transmission along the network, global transmission, and recovery, respectively:

$$\begin{aligned} \boldsymbol{\nu}_1 &= \left[-1, 1, -\frac{2N_{SS}}{S}, \frac{2N_{SS}}{S} - \left(1 + \frac{N_{SI}}{S}\right), \left(1 + \frac{N_{SI}}{S}\right)\right] \\ \boldsymbol{\nu}_2 &= \left[-1, 1, -\frac{2N_{SS}}{S}, \frac{2N_{SS}}{S} - \frac{N_{SI}}{S}, \frac{N_{SI}}{S}\right] \\ \boldsymbol{\nu}_2 &= \left[1, -1, \frac{2N_{SI}}{I}, -\frac{N_{SI}}{I} + \frac{2N_{II}}{I}, -\frac{2N_{II}}{I}\right]. \end{aligned} \tag{2}$$

III. Modeling the Optimal Path as a Rare Event

Given the specification of the transition rates and increments, the dynamics of the probability density, $\rho(\boldsymbol{X}, t)$, can now be modeled using a master equation [16], [30], [31]. The rare events are characterized by observing the extinction event in the tail of the probability distribution. Typically, when observing the times for the event to occur, one sees that the distribution of times is exponential [32]. When the population is sufficiently large, we may assume the distribution of times possesses such an exponential tail [15].

The rare events we are interested in are those of extinction where the number of infected nodes goes to zero. As in the globally coupled case ($\epsilon = 0$), the Monte Carlo dynamics of the network ($\epsilon = 1$) exhibits random fluctuations about an attracting endemic state, and then the internal fluctuations organize in such a way as to drive the infected nodes to extinction. We characterize the probability of an extinction event in the large population limit and compare it to the globally coupled case. In particular, we are interested in the most probable path and mean times to extinction. To understand the scaling of extinction times in terms of epidemiological parameters and network topology, we employ large deviation theory techniques for finite populations [33], [31].

Normalizing the state space of nodes and links with respect to N, $\mathbf{x} = \mathbf{X}/N$, we use a WKB approximation for the probability, $\rho(\boldsymbol{x}, t) = A(\boldsymbol{x})\exp(-NR(\boldsymbol{x}, t))$. To first order in $\mathcal{O}(N^{-1})$, we derive a Hamilton-Jacobi equation, with Hamiltonian H, and exponent R is defined as the action. The variable $\boldsymbol{p} = \partial_x R$ is the conjugate momenta of the Hamilton-Jacobi equation, and the Hamiltonian can be written as

$$H(\boldsymbol{x}, \boldsymbol{p}) = \sum_{k=1}^{3} w(\boldsymbol{x}, \boldsymbol{\nu}_k)(e^{\boldsymbol{p}\cdot\boldsymbol{\nu}_k} - 1). \tag{3}$$

We analyze the system by solving the characteristic equations $\dot{\boldsymbol{x}} = \partial_{\boldsymbol{p}} H(\boldsymbol{x}, \boldsymbol{p})$, $\dot{\boldsymbol{p}} = -\partial_{\boldsymbol{x}} H(\boldsymbol{x}, \boldsymbol{p})$ subject to appropriate boundary conditions. Since the action maximizes the probability of the rare event, we say the path to extinction is the most probable or optimal path to extinction given that the initial starting point is at the endemic state.

The boundary conditions for the characteristic equations are given as steady state solutions of from the equations of motion $\dot{\boldsymbol{x}} = \dot{\boldsymbol{p}} = 0$. For epidemic models in general, the distribution is quasi-stationary and peaks at an endemic state, where the number of new infections equals the number of recoveries per unit time, and corresponds to the zero conjugate momenta case. The other steady state is the extinct state which is saddle point. However, here we find that the conjugate momenta at this state is non-zero, since there is a non-zero probability current at that point. The steady states of the characteristic equation corresponding to zero conjugate momenta are those which satisfy the deterministic epidemic model. See Fig. 2 for a plot showing the quasi-stationarity and steady states.

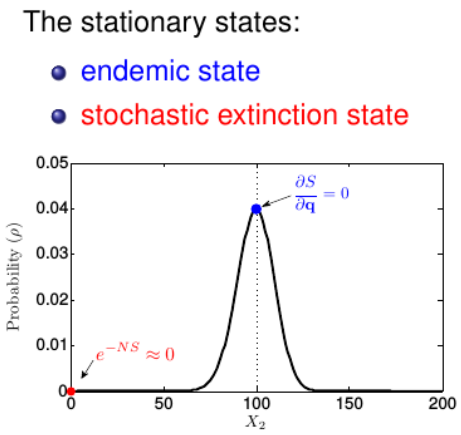

Fig. 2. An example of quasi-stationary density of states. X_2 on the x-axis denotes the number of infected nodes. The endemic steady state is attracting in the Hamiltonian equations of motion, while the extinct state where $X_2 = 0$ is a saddle point. The conjugate momenta corresponding to the endemic state is zero, while the momenta corresponding to the extinct state is non-zero due to a non-zero but small probability current.

To find the optimal path, we use the iterative action minimizing method (IAMM) [35] to perturb off the $\epsilon = 0$ solution, where the path can be analytically defined. For an all-to-all connected graph, the links depend quadratically on the nodes. We then use continuation as a function of ϵ to get to the locally-coupled $\epsilon = 1$ case. In Fig. 3, the computed most probable path is plotted for several values of ϵ. It is clear from Fig. 3a that the path predicted for the discretely coupled network ($\epsilon = 1$) is quite different from the globally coupled disease dynamics ($\epsilon = 0$). Note that as ϵ increases and infection spreads primarily along the network, II connections become more prominent because infected nodes arise next to other infected nodes.

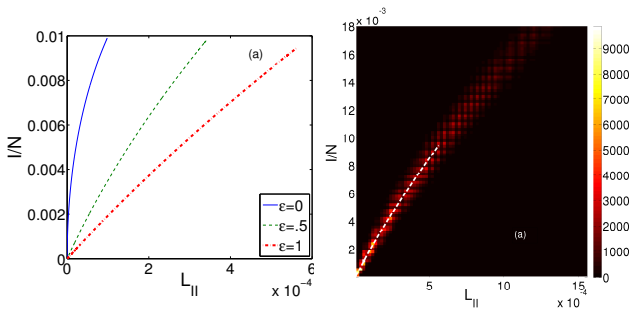

Fig. 3. (a) The most probable path projected onto the I/N versus the fraction of II links, L_{II}, for various ϵ values up to $\epsilon = 1$. Parameters: $p = 1.03 \times 10^{-4}$, $r = 0.002$ and $K/N = 10$. (b)The position density function of extinction paths computed from stochastic simulations on Erdős-Rényi random networks, projected onto the I/N versus L_{II} axis, and overlaid with the predicted most probable path (dashed curve). Reprinted from [28].

We compare the numerical results for our approximate system to Monte Carlo simulations of an **SIS** epidemic model on an Erdős-Rényi random network where the Monte Carlo simulations are completed using the Gillespie algorithm [36] with initial conditions at the endemic steady state. For the networks, 1000 trajectories are run to extinction, 100 for each of 10 randomly generated network geometries. From the pre-history of paths that go extinct, a density function is created from the prehistory of these paths, and a clear local maximum can be identified. This maximum corresponds to the most likely trajectory connecting the endemic and extinct points, and is shown in the density plots of Fig. 3b. Using the IAMM to compute the most probable path and comparing it to the prehistory of extinction events on stochastic networks, as shown by the dashed curve in, demonstrates that our model approximates well the path to extinction.

IV. Measuring Mean Extinction Times

From Monte-Carlo simulations, we can also approximate the mean lifetime of the disease from endemic state until extinction, where we assume that the mean lifetime τ is inversely proportional to the probability of the event; i.e., $\tau = B(\boldsymbol{x})e^{NR}$. The pre-factor may depend on all parameters for a given problem, but in general scales as $\frac{1}{\sqrt{N}}$ for sufficiently large N. For our purposes, the pre-factor can be found analytically for $\epsilon = 0$,

$$B = \frac{\sqrt{2\pi \frac{R_0^{\text{eff}}}{N}}}{r(R_0^{\text{eff}} - 1)^2}, \tag{4}$$

where the effective reproductive number is given as $R_0^{\text{eff}} = 2pK/(Nr)$ [37], [23].

In Fig. 4, we plot the log of the mean lifetimes of the disease compared to our numerical predictions of the action, incorporating the pre-factor in Eq. 4. Because it is analytically unavailable to find the pre-factor for $\epsilon > 0$, we assume that, since the action does not vary greatly with respect to ϵ, the change to the pre-factor will be negligible. Thus we can use the same pre-factor for the case when $\epsilon = 1$.

Figure 4a shows the log of the mean lifetime versus population for Monte Carlo simulations of the disease on discrete networks ($\epsilon = 1$) and the mean-field prediction generated by the IAMM, and scaled by the analytical pre-factor, i.e., $\ln \tau/N \approx R + \ln B/N$. Because the action R is invariant with respect to N, the scaling depends entirely on the pre-factor, and the good agreement between our analytical approximation and stochastic simulations shows that our approximation of the pre-factor is sufficient to capture the dynamics. Figure 4b varies the infection rate p, and shows the lifetime scaling predicted by our model and the stochastic simulations as the probability of disease propagation along the network is increased.

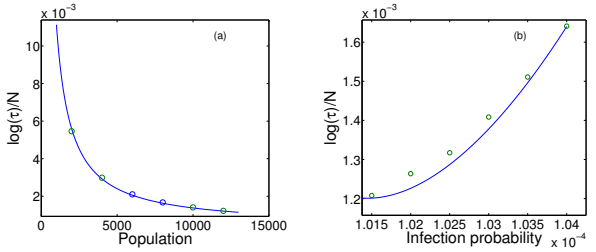

Fig. 4. a) $\ln(\tau)/N$ versus N predictions (solid curve) compared to mean over 1000 realizations of extinction on a random network (circles) for $r = 0.002$, $p = 1.03 \times 10^{-4}$, $K/N = 10$, and $\epsilon = 1$. b) $\ln(\tau)/N$ versus p predictions (solid curve) compared to mean over 1000 network extinction events (circles) for $r = 0.002$, $K = 10^5$, $N = 10^4$, and $\epsilon = 1$. Note there is no fitting parameter in the theoretical plots. Reprinted from [28].

V. Extinction on Adaptive Networks

As another future network application, we can consider the extinction dynamics of coupled systems which are adaptive. (See [8] for details and further results regarding the effects of fluctuations.) In real networks nodes and links change in time, in that node dynamics affects network geometry, and in turn geometry affects node dynamics. This leads to a feedback mechanism since links depend on nodes and vice versa. For example, in a network in which the population consists now of suseptible, infectious and recovered individuals, there exists adaptation in which those non-infected nodes coming in contact with infectious nodes rewire away to a non-infectious node with rate w. The rewiring rules specifying transitions between states and parameter definitions are shown in Fig. 5.

The effect of rewiring adaptively has a significant effect not just on the node dynamics, but the entire bifurcation structure. In Fig. 6, the left panels show that when the rewiring rate is non-zero, new states are created, as well as a bi-stable region where both endemic and extinct states are attracting. The right panel shows how the degree distributions change from the non-adaptive case when rewiring is turned on.

In terms of node and link dynamics, the degree appears to have strong oscillations. For a given node chosen at random, we can see how the adaptivity affects the network as a function of time, as in Fig. 7. There are strong oscillations in the degree on average, and it is easy to see infected node lose connections rapidly, while the recovered and susceptible nodes gain links as

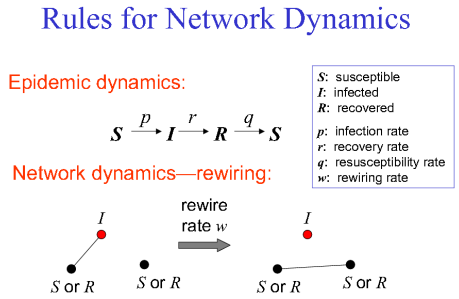

Fig. 5. Adaptive network dynamical rules for an SIRS model. Here we introduce a recovery variable R. The new transition rate for $R \to S$ is the re-susceptibility rate q. The rewiring rate w acts to change IS or IR to SS, SR or RR links.

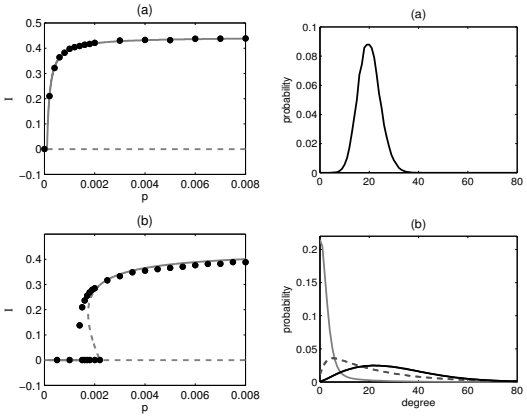

Fig. 6. The effect of rewiring in an adaptive network using avoidance behavior. The left panels show the fraction of infectious individuals I in a finite population as a function of the infection rate p. (a) Fixed contact network (no rewiring). (b) Rewiring is turned on, causing the emergence of bistable behavior. Solid (dashed) lines are stable (unstable) mean field predictions, and dots correspond to averaged Monte Carlo runs. The right panels show the degree distribution (a) without rewiring and (b) with rewiring. Light grey denotes infectious, black susceptible, and dashed recovered individual fractions. Reprinted from [8].

time evolves. The oscillatory nature of the degree is probably responsible for the existence of oscillatory behavior for certain regions of parameters [38].

It has been observed that when a network going to extinction is adaptive, vaccination controls work synergistically with adaptivity to improve the elimination of infected nodes [39]. A model of an adaptive SIVS network is shown schematically in Fig. 8. It is similar to the previous model, except that the recovered class is replaced with a vaccinated class. The other change is in vaccine control where the parameter to vaccinate a percentage of the susceptible population is applied as a Poisson process. That is, control is discontinuous in time, and

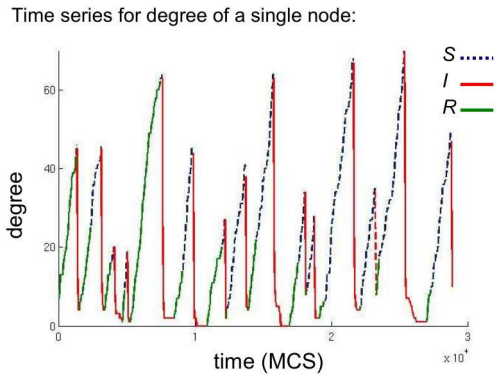

Fig. 7. Node connections are shown when the network is adaptive. An example of the degree of a single node chosen at random is shown as a function of time. Due to rewiring, infected nodes lose links over time, while recovered and susceptible nodes gain links. Infected nodes thus become more isolated.

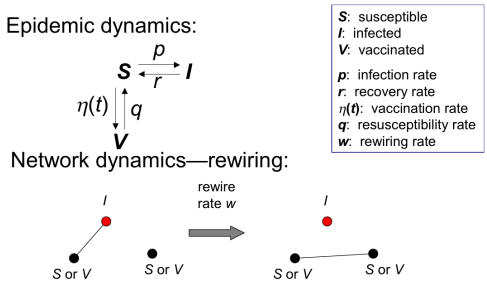

Fig. 8. An SIVS model of an adaptive network with vaccination. The new variable here is V, which represents the vaccination class. Parameters are shown in the box. Vaccination is assumed not to be permanent, so a return to the susceptible class may occur at rate q. The vaccine control is time dependent, and is a Poisson process having mean frequency ν. A fixed fraction, A, of the population of the susceptibles is vaccinated. The rewiring rate w is defined as before moving links between infected and non-infected nodes to links between non-infected nodes.

the percentage A of the susceptible population is vaccinated with mean frequency ν. The effects of combining vaccination with rewiring behavior were shown to be highly effective in that a reduction of between two and three orders of magnitude of vaccine resources were necessary to achieve the same mean extinction times for the non-adaptive case.

In terms of the degree dynamics for both nodes and links, and example is shown when the vaccination rate is applied using the Poisson process in Fig. 9. Here 10 per cent of the susceptible population was vaccinated with a mean frequency of $\nu = 0.0005$. Finite vaccine pulses are shown by green $x's$. One can see a dramatic change in both infected node fraction and its degree on the path to extinction.

VI. Conclusions

We have presented a method for predicting extinction in stochastic network systems by analyzing a pair-based proxy model. The optimal paths to extinction were found, allowing the prediction of mean extinction times. Tracking the path to

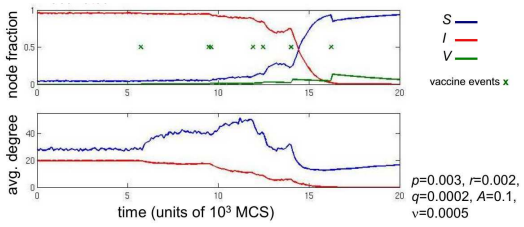

Fig. 9. Node and degree changes in an adaptive network with vaccine controls in an SIVS mode along the path to extinction of I nodes. Poisson distributed vaccine pulses are designated by green crosses. The top panel shows the evolution of node fraction for each of the nodes for S, I, V. The bottom panel shows the degree shedding of the I nodes as the disease goes extinct in red. The blue curve is the total of S and V nodes. The parameters are as shown in the lower right part of the figure.

extinction was aided by perturbing from the known path in a well-mixed system of individuals in a large population.

We also considered the dynamic network structure of adaptive networks. We showed how a strong oscillatory degree dynamic arises due to the rewiring away from infective nodes. In the presence of vaccine controls, the degree was also shown to evolve in a special way when the vaccination is a Poisson process. Here we applied vaccine to susceptible nodes, which tend to have a higher degree due to the rewiring away from infected nodes. In general, vaccination of high degree nodes provides better protection. In contrast to the adaptive network, a static network has higher degree infected nodes which are not vaccinated.

In the future, the pair-based proxy method will be extended to systems such as epidemics on adaptive networks (e.g., [38]) by adding network adaptation to the list of transitions (Eqs. 2 and 1). Vaccination transitions can also be added, as in [39]. More generally, this pair-based proxy method will be applicable to predict extinction in any network system that is well described by a pair-based approximation for dynamics of nodes and links, including games on networks (e.g., [40], [41]).

Further, we expect that our method of continuously varying a parameter while tracking the path to extinction will be useful in other contexts where finding the path a priori is difficult due to high dimensionality, as in finite mode projection of partial differential equations [42], and pattern switching along paths in continuous systems [43].

Acknowledgment

BL was a National Research Council post doctoral fellow when this research was done. IBS was supported by NRL base funding (N0001414WX00023) and Office of Naval Research (N0001414WX20610). LBS was supported by the Army Research Office, Air Force Office of Scientific Research, and by Award Number R01GM090204 from the National Institute of General Medical Sciences. We also acknowledge useful discussions on quasi-stationarity with Lora Billings.

REFERENCES

[1] M. Barthelemy, A. Barrat, R. Pastor-Satorras, and A. Vespignani, "Velocity and hierarchical spread of epidemic outbreaks in scale-free networks," *Phys. Rev. Lett.*, vol. 92, no. 17, APR 30 2004.

[2] M. Girvan and M. E. J. Newman, "Community structure in social and biological networks," *Proceedings of the National Academy of Science*, vol. 99, pp. 7821–7826, 2002.

[3] T. Gross and B. Blasius, "Adaptive coevolutionary networks: a review," *J. Roy. Soc. Interface*, vol. 5, no. 20, pp. 259–271, MAR 6 2008.

[4] F. Wu, B. A. Huberman, L. A. Adamic, and J. R. Tyler, "Information flow in social groups," *Physica A-Statistical Mechanics And Its Applications*, vol. 337, pp. 327–335, 2004.

[5] D. H. Zanette and S. Risau-Gusmán, "Infection spreading in a population with evolving contacts," *J. Biol. Phys.*, vol. 34, pp. 135–148, April 2008.

[6] M. S. Shkarayev, I. B. Schwartz, and L. B. Shaw, "Recruitment dynamics in adaptive social networks," *Journal of Physics A: Mathematical and Theoretical*, vol. 46, no. 24, p. 245003, 2013.

[7] T. Gross, C. J. D. D'Lima, and B. Blasius, "Epidemic dynamics on an adaptive network," *Phys. Rev. Lett.*, vol. 96, p. 208701, 2006.

[8] L. B. Shaw and I. B. Schwartz, "Fluctuating epidemics on adaptive networks," *Phys. Rev. E*, vol. 77, JUN 2008.

[9] L. Shaw and I. B. Schwartz, "Enhanced vaccine control in adaptive networks," *arXiv:0907.5539*, 2009.

[10] I. B. Schwartz and L. B. Shaw, "Rewiring for adaptation," *Physics*, vol. 3, p. 17, Feb 2010.

[11] I. B. Schwartz, L. B. Shaw, and M. S. Shkarayev, "Adaptive network dynamicsmodeling and control of time-dependent social contacts," in *Information Fusion (FUSION), 2011 Proceedings of the 14th International Conference on*. IEEE, 2011, pp. 1–7.

[12] B. A. Melbourne and A. Hastings, "Extinction risk depends strongly on factors contributing to stochasticity," *Nature*, vol. 454, pp. 100–103, 2008.

[13] D. Alonso, A. McKane, and M. Pascual, "Stochastic amplification in epidemics," *J. R. Soc. Interface*, pp. 575–582, 2006.

[14] L. Stone, R. Olinky, and A. Huppert, "Seasonal dynamics of recurrent epidemics," *Nature*, vol. 446, pp. 533–536, 2007.

[15] M. I. Dykman, I. B. Schwartz, and A. S. Landsman, "Disease extinction in the presence of random vaccination," *Phys. Rev. Lett.*, vol. 101, p. 078101, 2008.

[16] R. Kubo, "Stochastic Liouville equations," *J. Math. Phys.*, vol. 4, pp. 174–183, 1963.

[17] I. Nasell, "On the time to extinction in recurrent epidemics," *J. R. Statist. Soc. B*, vol. 61, pp. 309–330, 1999.

[18] I. B. Schwartz, E. Forgoston, S. Bianco, and L. B. Shaw, "Converging towards the optimal path to extinction," *Journal of The Royal Society Interface*, p. rsif20110159, 2011.

[19] E. Forgoston, S. Bianco, L. B. Shaw, and I. B. Schwartz, "Maximal sensitive dependence and the optimal path to epidemic extinction," *Bulletin of mathematical biology*, vol. 73, no. 3, pp. 495–514, 2011.

[20] B. Bolker and B. Grenfell, "Impact of vaccination on the spatial correlation and persistence of measles dynamics," *Proceedings of the National Academy of Sciences*, vol. 93, no. 22, p. 12648, 1996.

[21] I. B. Schwartz, L. Billings, and E. M. Bollt, "Dynamical epidemic suppression using stochastic prediction and control," *Phys. Rev. E*, vol. 70, no. 4, Part 2, 2004.

[22] M. Khasin and M. I. Dykman, "Control of rare events in reaction and population systems by deterministically imposed transitions," *Phys. Rev. E*, vol. 83, p. 031917, 2011.

[23] L. Billings, L. Mier-y Teran-Romero, B. Lindley, and I. B. Schwartz, "Intervention-based stochastic disease eradication," *PloS one*, vol. 8, no. 8, p. e70211, 2013.

[24] M. Keeling, "The implications of network structure for epidemic dynamics." *Theoretical population biology*, vol. 67, no. 1, pp. 1–8, 2005.

[25] P. Trapman, "On analytical approaches to epidemics on networks." *Theoretical population biology*, vol. 71, no. 2, pp. 160–73, Mar. 2007. [Online]. Available: http://www.ncbi.nlm.nih.gov/pubmed/17222879

[26] Y. Hayashi, M. Minoura, and J. Matsukubo, "Oscillatory epidemic prevalence in growing scale-free networks," *Physical Review E*, vol. 69, no. 1, p. 016112, Jan. 2004.

[27] S. Silva, J. Ferreira, and M. Martins, "Epidemic spreading in a scale-free network of regular lattices," *Physica A: Statistical Mechanics and its Applications*, vol. 377, no. 2, pp. 689–697, Apr. 2007.

[28] B. S. Lindley, L. B. Shaw, and I. B. Schwartz, "Rare-event extinction on stochastic networks," *EPL*, vol. 108, p. 58008, 2014.

[29] T. Rogers, W. Clifford-Brown, C. Mills, and T. Galla, "Stochastic oscillations of adaptive networks: application to epidemic modelling," *Journal of Statistical Mechanics: Theory and Experiment*, vol. 2012, no. 08, p. P08018, Aug. 2012.

[30] N. G. Van Kampen, *Stochastic Processes in Physics and Chemistry*, 3rd ed. Amsterdam: Elsevier, 2007.

[31] H. Gang, "Stationary solution of master equations in the large-system-size limit," *Phys. Rev. A*, vol. 36, no. 12, pp. 5782–5790, 1987.

[32] I. B. Schwartz, L. Billings, M. Dykman, and A. Landsman, "Predicting extinction rates in stochastic epidemic models," *J. Stat. Mech.-Theory E.*, p. P01005, 2009.

[33] M. I. Dykman, I. B. Schwartz, and A. S. Landsman, "Disease extinction in the presence of random vaccination," *Phys. Rev. Lett.*, vol. 101, p. 078101, Aug 2008.

[34] A. Kamenev and B. Meerson, "Extinction of an infectious disease: A large fluctuation in a nonequilibrium system," *Phys. Rev. E*, vol. 77, p. 061107, Jun 2008.

[35] B. S. Lindley and I. B. Schwartz, "An iterative action minimizing method for computing optimal paths in stochastic dynamical systems," *Physica D*, vol. 255, pp. 22–30, 2013.

[36] D. T and Gillespie, "A general method for numerically simulating the stochastic time evolution of coupled chemical reactions," *Journal of Computational Physics*, vol. 22, no. 4, pp. 403 – 434, 1976.

[37] M. Assaf and B. Meerson, "Extinction of metastable stochastic populations," *Phys. Rev. E*, vol. 81, p. 021116, Feb 2010.

[38] T. Gross, C. DLima, and B. Blasius, "Epidemic dynamics on an adaptive network," *Physical review letters*, vol. 96, no. 20, p. 208701, 2006.

[39] L. B. Shaw and I. B. Schwartz, "Enhanced vaccine control of epidemics in adaptive networks," *Phys. Rev. E.*, vol. 81, p. 046120, 2010.

[40] D. A. Rand, "Correlation Equations and Pair Approximations for Spatial Ecologies," *CWI Quarterly*, vol. 12, pp. 329–368, 1999.

[41] H. Ohtsuki, C. Hauert, E. Lieberman, and M. a. Nowak, "A simple rule for the evolution of cooperation on graphs and social networks." *Nature*, vol. 441, no. 7092, pp. 502–5, May 2006.

[42] G. Bunin, Y. Kafri, and D. Podolsky, "Large deviations in boundary-driven systems: Numerical evaluation and effective large-scale behavior," *EPL (Europhysics Letters)*, vol. 99, p. 20002, 2012.

[43] E. Weinan, R. Weiqing, and E. Vanden-Eijnden, "Minimum action method for the study of rare events," *Comm. on Pure and Appl. Math.*, vol. 57, pp. 0001–0020, 2004.