Rare-event extinction on stochastic networks

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Abstract – We consider the problem of extinction processes on random networks with a given structure. For sufficiently large well-mixed populations, the process of extinction of one or more state variable components occurs in the tail of the quasi-stationary probability distribution, thereby making it a rare event. Here we show how to extend the theory of large deviations to random networks to predict extinction times. In particular, we use the theory to find the most probable path leading to extinction. We apply the methodology to epidemic models and discover how mean extinction times scale with epidemiological and network parameters in Erdős-Rényi networks. The results are shown to compare quite well with Monte Carlo simulations of the network in predicting both the most probable paths to extinction and mean extinction times.

In many models of finite populations, random fluctuations occur due to internal interactions between individuals or agents, and/or external stochastic forces. Such fluctuations are evident in the modeling of well-mixed populations that support epidemics of disease spread [1], as well as ecological bio-diversity of species [2,3], among others. Typically, the dynamics performs small random fluctuations about an attracting state. However, in almost all populations of finite size, fluctuations may organize in such a way to drive one or more components of the population to extinction. Mechanisms conjectured to play a role in extinction processes include small population size, low contact frequency for frequency-dependent transmission, competition for resources, and evolutionary pressure [4], as well as heterogeneity in populations and transmission in coupled population models [5,6].

In epidemic models where the population is well mixed, extinction of infectious individuals has been shown to be affected by noise intensity [7], peak infectious population size [8], and seasonal phase occurrence [9]. 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 [10]; i.e., the probability of extinction is a decreasing exponential function [11]. Vaccination and treatment programs have been studied to speed up the extinction of disease in well-mixed populations [10,12]. For example, although most vaccination schedules are designed to be administered periodically (deterministic) [13–15], Poisson-distributed scheduling was recently shown to be more efficient than regular treatment schedules [16].

In network populations, outbreak extinction probabilities have been predicted for early times when an infection has just been introduced [17,18]. Other studies of extinction on networks attempt to predict whether a persistent non-extinct state exists, such as for computer viruses in growing networks [19] and epidemics in various network geometries (e.g., [20]). This is equivalent to finding the bifurcation point where the extinct state becomes unstable and thus can frequently be predicted using deterministic approximations (e.g., [19]).

Extinction times for an endemic disease in a network have occasionally been studied, mostly numerically. In [21], extinction times were measured in simulations of an epidemic on an adaptive network, and the log of the extinction time was observed to increase with a power of the distance from a bifurcation point. In [22], lifetimes were measured in a similar model with the addition of pulsed vaccination. Similar lifetimes were obtained in simulations by adding stochastic pulsed vaccination to a deterministic mean-field model based on a pair approximation, suggesting that it is not necessary to model the full stochastic network dynamics in detail to capture the extinction time. Here we will present a method for finding the most probable path to extinction and the extinction time in networks that can be described using a pair approximation.
Here we consider the problem of epidemic extinction in stochastic 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.

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 $S$ as the number of susceptible individuals and $I$ as the number of infected individuals, such that $S + I = N$. The population is closed, and there are no births and deaths.

To quantify how the network topology compares with a well-mixed global, or all-to-all, coupling structure, we allow for two modes of disease spread. Specifically, global disease transmission occurs in a well-mixed population when the disease transmits from any infected person to any susceptible person. In contrast to global transmission models, local disease transmission occurs when a network of connections between individual persons is considered, and diseases can only spread along links between infected and susceptible individuals. In the case of transmission via only the network, we let $p$ be the infection rate per SI link. When transmission is only global, we define a global contact rate of $p2K/N$, selected so that the epidemic threshold occurs at $p^* = \frac{2N}{K\gamma}$ in both the globally coupled and network transmission cases. We will introduce a homotopy parameter $\epsilon$ which continuously transforms the system between the local and global transmission models. Finally, $r$ is a recovery rate.

We approximate the dynamics of the network by considering transitions in nodes and links, similar to the pair-based proxy model of [23]. We let $X$ denote the vector with components containing both the number of nodes and number of links of the network. Specifically, we let $X = [S, I, N_{SS}, N_{SI}, N_{II}]$, where $S, I$ denotes the numbers of susceptibles and infectives, respectively, and $N_{AB}$ denotes the number of connections between nodes of type $A$ and $B$. 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 $M$ transition events with transition rates $W(X, \nu_k)$ having increments $\nu_k$. The dynamics of the probability density, $\rho(X, t)$, can now be modeled as a master equation [11,24,25]:

$$\frac{\partial \rho}{\partial t}(X, t) = \sum_{k=1}^{M}[W(X - \nu_k, \nu_k)\rho(X - \nu_k, t) - W(X, \nu_k)\rho(X, t)],$$

where $\rho(X, t)$ represents the probability of finding the system in state $X$ at time $t$.

The rare events are characterized by observing the targeted 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 [26]. When the population is sufficiently large, we may assume the distribution of times possesses such an exponential tail [10].

Making a homogeneity assumption that each node’s local neighborhood is identical to that of all other nodes of the same type, we can find its neighbors from the system average. For example, a node of type $S$ would have an expected degree $ds = (2N_{SS} + N_{SI})/S$ and has, on average, $2N_{SS}/S$ neighbors of type $S$.

We next specify the possible transitions and associated transition rates for the pair-based proxy model, so that the approximate stochastic process can be described by a master equation that captures fluctuations in the probability density. There are three types of state transitions to consider: $S \rightarrow I$ by transmission along the network, $S \rightarrow I$ by global disease transmission, and $I \rightarrow S$ by disease recovery.

To determine the increments for each transition, notice that whenever a node changes from one state to another, all of its associated links change. For example, if a susceptible node becomes infected via local disease transmission, then $S \rightarrow I$, and therefore all its $SS$ links become SI links. Further, $1 + N_{SI}/S$ links of type SI will be lost, creating as many II links. Using this logic, we can write the following transition increments for transmission along the network, global transmission, and recovery, respectively:

$$\nu_1 = \begin{bmatrix} -1, 1, -\frac{2N_{SS}}{S}, \frac{2N_{SS}}{S} \end{bmatrix},\begin{bmatrix} 1 + \frac{N_{SI}}{S} \end{bmatrix}, \begin{bmatrix} 1 + \frac{N_{SI}}{S} \end{bmatrix},$$

$$\nu_2 = \begin{bmatrix} -1, 1, -\frac{2N_{SS}}{S}, \frac{N_{SI}}{S} \end{bmatrix},\begin{bmatrix} 1 + \frac{N_{SI}}{S} \end{bmatrix}, \begin{bmatrix} 1 + \frac{N_{SI}}{S} \end{bmatrix},$$

$$\nu_3 = \begin{bmatrix} 1, -1, -\frac{2N_{SI}}{I}, \frac{N_{SI}}{I} \end{bmatrix},\begin{bmatrix} 2N_{II} \end{bmatrix}, \begin{bmatrix} 2N_{II} \end{bmatrix}.$$

For each transition $\nu_k$ we have an associated transition rate $W(X, \nu_k)$ given by

$$W(X, \nu_1) = \epsilon p N_{SI},$$

$$W(X, \nu_2) = (1 - \epsilon)p \frac{2K S I}{N},$$

$$W(X, \nu_3) = r I,$$  

for transmission along the network, global transmission, and recovery, respectively. We have introduced the homotopy parameter $\epsilon$ which continuously transforms the system between the global and network transmission models. Here $\epsilon = 0$ returns the standard equations for the SIS model in a globally coupled (well-mixed) population, and $\epsilon = 1$ is a locally coupled network SIS model. Even the pair-based proxy system is sufficiently high dimensional that finding the most probable path to extinction will be difficult a priori, but continuously varying $\epsilon$ will allow
us to track the path from the known case of global coupling to the network case. This approach to tracking the path to extinction by continuously varying a parameter is new. However, we note that far from the extinction bifurcation point, if the well-mixed population is constrained in fluctuation so that $S + I = N$, the limit of $I(\epsilon)$ as $\epsilon$ approaches zero may not exist since the SIS model has been shown to be fragile [27].

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 state, and then the fluctuations organize in such a way as to drive the infected nodes to extinction. We wish to 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 [25,28].

We introduce the scaled variables $x$ with components defined by $x_1 = I/N$, $x_2 = N_{SI}/N$, and $x_3 = N_{II}/N$, where the remaining variables $S$ and $N_{SS}$ can be eliminated by conservation of nodes and links. We define the scaled transition rates $w(x,\nu_k) = W(X,\nu_k)/N$ on these variables. Henceforth, we will refer to the fraction of infected and susceptibles as $P_I = I/N$ and $P_S = S/N$. The link variables will be given as $L_{AB} = N_{AB}/N$. Given that the probability current at the extinct state is sufficiently small for large enough $N$, and that the increments are sufficiently small with respect to $N$, there will exist a quasi-stationary probability distribution about a state with a non-zero number of infected individuals that decays into the stationary (extinct) solution over exponentially long times [16]. The extinction rate of infected individuals can be calculated from the tail of the quasi-stationary distribution. It has been demonstrated that a WKB approximation to the quasi-stationary distribution allows one to approximate the mean extinction time provided the population is sufficiently large when $\epsilon = 0$ [28].

Employing the WKB approximation for the probability, $\rho(x,t) = A(x)\exp(-NR(x,t))$, from the first-order expansion with respect to $N^{-1}$, we can write the Hamilton-Jacobi equation,

$$\frac{\partial R}{\partial t} + H\left(x, \frac{\partial R}{\partial x}\right) = 0. \tag{4}$$

Here the function $H$ is called the Hamiltonian of the system, and $R$ is known as the action. The variable $p = \partial_x R$ is the conjugate momenta of the Hamilton-Jacobi equation, and the Hamiltonian can be written as

$$H(x, p) = \sum_{k=1}^{3} w(x, \nu_k)(e^{p\nu_k} - 1). \tag{5}$$

We analyze the system by solving the characteristic equations

$$\dot{x} = \partial_p H(x, p), \quad \dot{p} = -\partial_x H(x, p).$$

Since we are interested in the probability of extinction events, we must compute the action $R$ that maximizes this probability. This will, naturally, be the one that satisfies the Hamilton-Jacobi equation (4). This implies that there will be some trajectory along which $R$ is minimized, which represents the maximal probability of such an extinction event, and that this trajectory, which we will call the “most probable path”, satisfies the characteristic equations for the Hamiltonian plus boundary conditions [25,28].

The boundary conditions for the characteristic equations are given as steady-state solutions of $\dot{x} = \dot{p} = 0$. In fact, for epidemic models, the quasi-stationary probability distribution 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, i.e., the steady state is at $(x, p) = (x_e, 0)$. The other steady state is the extinct state, $P_S = 1$, $P_I = 0$, which is a saddle point. However, here we find that the conjugate momenta at this state, $p_0$, are non-zero, since there is a non-zero probability current at that point. We label the endemic state as $(x_e, 0)$, and the non-trivial extinct state as $(x_0, p_0)$. The steady states of the characteristic equation corresponding to zero conjugate momenta are those which satisfy the deterministic epidemic model.

For the case $\epsilon = 0$, analytic solutions are available for the most probable path to extinction [29], and hence the action may be computed directly. However, for $\epsilon = 1$, the additional equations for the mean link fractions complicate the analysis, and the most probable path must be computed numerically. We therefore employ the iterative action minimizing method (IAMM) [30] to compute the most probable path between $x_0$ and $x_e$, and the action will be given by the path integral $\int_{x_0}^{x_e} p \cdot dx$ along this path since $H(x, p) = 0$ along the path.

Using the IAMM, we perturb off the $\epsilon = 0$ solution, where the path can be analytically defined. For an all-to-all connected graph, $L_{SI} = 2P_S P_I$ and $L_{II} = P_I^2$ and momenta conjugate to the link variables are 0. We then use continuation as a function of $\epsilon$ to get to the locally coupled $\epsilon = 1$ case. We demonstrate the robustness of this approach in fig. 1, where the computed most probable path is plotted for several values of $\epsilon$. It is clear from fig. 1(a) 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 $\epsilon$ increases and infection spreads primarily along the network, $II$ connections become more prominent because infected nodes arise next to other infected nodes. Also, as shown in fig. 1(b), the action decreases, corresponding to a decrease in the extinction time, presumably due to stochastic effects in local neighborhoods of nodes.

To validate the predicted paths and extinction times, we compare the numerical results for our approximate system
to Monte Carlo simulations of an SIS epidemic model on an Erdős-Rényi random network. Here, the Monte Carlo simulations are completed using the Gillespie algorithm [31] with an initial condition 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 set 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. 2. 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 fig. 2, demonstrates that our proxy model approximates well the path to extinction.

From Monte Carlo simulations, we can also approximate the mean lifetime of the disease from endemic state until extinction. We assume that the mean lifetime $\tau$ is inversely proportional to the probability of the event, and thus can be related to our action by the formula $\tau = B(x)e^{NR}$. Note the appearance of the prefactor $B(x)$ in this calculation. The prefactor may depend on all parameters for a given problem, but in general scales as $B \approx \frac{1}{\epsilon}$ for sufficiently large $N$. For our purposes, the prefactor can be found analytically for $\epsilon = 0$,

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

where the effective reproductive number is given as $R_0^{\text{eff}} = 2pK/(Nr)$ [16,32].

In fig. 3, we plot the log of the mean lifetimes of the disease compared to our numerical predictions of the action, incorporating the prefactor in eq. (6). Because it is analytically intractible to find the prefactor for $\epsilon > 0$, we assume that, since the action does not vary greatly with respect to $\epsilon$, the change to the prefactor will be negligible, and thus we can use the same prefactor for the cases in which $\epsilon = 1$.

Figure 3(a) shows the log of the mean lifetime vs. 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 prefactor, 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 prefactor, and the good agreement between our analytical approximation and stochastic simulations shows that our approximation of the prefactor is sufficient to capture the dynamics. Figure 3(b) varies a different parameter, 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.

We have presented a method for predicting extinction in stochastic network systems by analyzing a pair-based proxy model. Both extinction times and paths to extinction were obtained. Tracking the path to extinction was aided by perturbing from the known path in a well-mixed system. In the future, the pair-based proxy method will be extended to systems such as epidemics on adaptive networks (e.g., [33]) by adding network adaptation to the list of transitions (eqs. (2) and (3)).
can also be added, as in [22]. 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., [34,35]). 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 [36], and pattern switching along paths in continuous systems [37].

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REFERENCES

[1] Anderson R. M. and May R. M., Infectious Diseases of Humans (Oxford University Press) 1991.
[2] Banavar J. R. and Maritan A., Nature, 460 (2009) 334.
[3] Azaele S., Pigolotti S., Banavar J. R. and Maritan A., Nature, 444 (2006) 926.
[4] de Castro F. and Bolker B., Ecol. Lett., 8 (2005) 117.
[5] Lloyd A. L., Zhang J. and Root A. M., J. R. Soc. Interface, 4 (2007) 851.
[6] Eriksson A., Elías-Wolff F. and Mehlig B., Theor. Popul. Biol., 83 (2013) 101.
[7] Melbourne B. A. and Hastings A., Nature, 454 (2008) 100.
[8] Alonso D., McKane A. and Pascual M., J. R. Soc. Interface, (2006) 575.
[9] Stone L., Olinky R. and Huppert A., Nature, 446 (2007) 533.
[10] Dykman M. I., Schwartz I. B. and Landsman A. S., Phys. Rev. Lett., 101 (2008) 078101.
[11] Kuro R., J. Math. Phys., 4 (1963) 174.
[12] Nasef I., J. R. Stat. Soc. B, 61 (1999) 309.
[13] Bolker B. and Grenfell B., Proc. Natl. Acad. Sci. U.S.A., 93 (1996) 12648.
[14] Schwartz I. B., Billings L. and Bollt E. M., Phys. Rev. E, 70 (2004).
[15] Khasin M. and Dykman M. I., Phys. Rev. E, 83 (2011) 031917.
[16] Billings L., Mier-y Teran-Romero L., Lindley B. and Schwartz I. B., PLoS ONE, 8 (2013) e70211.
[17] Keeling M., Theor. Popul. Biol., 67 (2005) 1.
[18] Trapman P., Theor. Popul. Biol., 71 (2007) 160, http://www.ncbi.nlm.nih.gov/pubmed/17222879.
[19] Hayashi Y., Minoura M. and Matsukubo J., Phys. Rev. E, 69 (2004) 016112.
[20] Silva S., Ferreira J. and Martins M., Physica A: Stat. Mech. Appl., 377 (2007) 689.
[21] Shaw L. B. and Schwartz I. B., Phys. Rev. E, 77 (2008) 066101.
[22] Shaw L. B. and Schwartz I. B., Phys. Rev. E, 81 (2010) 046120.
[23] Rogers T., Clifford-Brown W., Mills C. and Galla T., J. Stat. Mech.: Theory Exp. (2012) P08018.
[24] Van Kampen N. G., Stochastic Processes in Physics and Chemistry, 3rd edition (Elsevier, Amsterdam) 2007.
[25] Gang H., Phys. Rev. A, 36 (1987) 5782.
[26] Schwartz I. B., Billings L., Dykman M. and Landsman A. J., Stat. Mech.: Theory Exp. (2009) P01005.
[27] Khasin M. and Dykman M. I., Phys. Rev. Lett., 103 (2009) 068101.
[28] Dykman M. I., Schwartz I. B. and Landsman A. S., Phys. Rev. Lett., 101 (2008) 078101.
[29] Kamenev A. and Meerson B., Phys. Rev. E, 77 (2008) 061107.
[30] Lindley B. S. and Schwartz I. B., Physica D, 255 (2013) 22.
[31] Gillespie D. T., J. Comput. Phys., 22 (1976) 403.
[32] Assaf M. and Meerson B., Phys. Rev. E, 81 (2010) 021116.
[33] Gross T., Dongmar D’Lima C. J. and Blasius B., Phys. Rev. Lett., 96 (2006) 208701.
[34] Rand D. A., CWI Q., 12 (1999) 329.
[35] Ohtsuki H., Hauert C., Lieberman E. and Nowak M. A., Nature, 441 (2006) 502.
[36] Bunin G., Kafri Y. and Podolsky D., EPL, 99 (2012) 20002.
[37] Weinan E., Weiqing R. and Vanden-Eijnden E., Commun. Pure Appl. Math., 57 (2004) 0001.