Fear in Networks

How social adaptation controls epidemic outbreaks

Ira B. Schwartz

Naval Research Laboratory
Nonlinear Systems Dynamics Section
Code 6792
Washington, DC 20375

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Friends and Collaborators

Jason Hindes

Naval Reearch Laboratory, Washington, DC

Leah Shaw

Applied Science
College of William and Mary, Williamsburg, VA

Brandon Lindley

Daniel H. Wagner Associates, Inc.

Lora Billings and Eric Forgoston

Department of Mathematical Sciences, Montclair State University, Montclair, NJ

Simone Bianco

IBM Almaden Research Center IBM Rock Star Employee

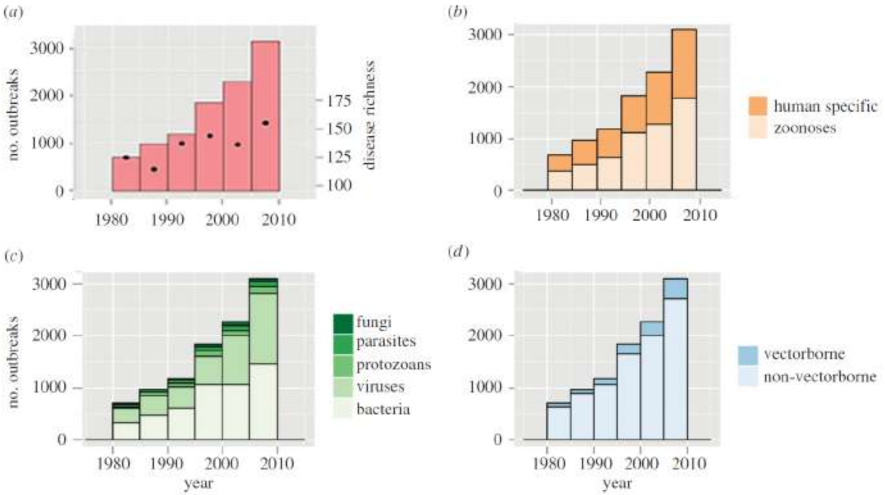
Luis Mier-y-Teran

Office of the Assistant Secretary for Preparedness and Response (ASPR)

Department of Health and Human Services (HHS)


COVID-19
EBOLA
ZIKA
DISEASES IN THE NEWS...
Novel Coronavirus Continues To Spread
by George Ochoa
May 2013
A novel coronavirus (nCoV) outbreak that began in Saudi Arabia has infected
More than 50 travelers just back in the United States from China who had flu-like symptoms have been tested for the H7N9 bird flu virus, federal health
The number of people infected with measles in South Wales reached 1,039 - 85 of whom have needed hospital treatment.
Europe's Embarrassing Problem
By Kai Kupferschmidt
222 cases, the highest number since 1996, and most importations come from Europe.
Health Dept: Measles outbreak hits Brooklyn
May 15, 2013
A measles outbreak has hit one Brooklyn community and may
can be deadly. Symptoms include rash, fever, cough, runny nose and sore throat.
The disease can be prevented with a vaccine. The new outbreak

Temporal Increase of Epidemic Outbreaks



Smith KF, 2014 J. R. Soc. Interface 11: 20140950. http://dx.doi.org/10.1098/rsif.2014.0950

Why Study Disease Extinction

- Control and eradication of infectious diseases are main and important public health goals.

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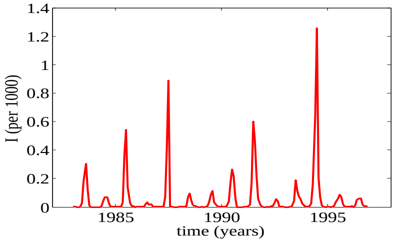
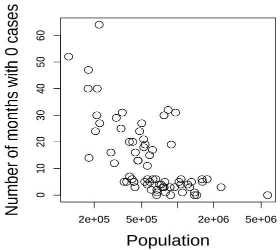
- Control and eradication of infectious diseases are main and important public health goals.
- Extinction is observed in networked populations.
  - Disease extinction occurs when infective population goes to zero.
  - Local extinction in connected patches but reintroduced
  - Global extinction is difficult and a rare event.

Dengue Incidence for
Chiang Mai province (1/72), Thailand.



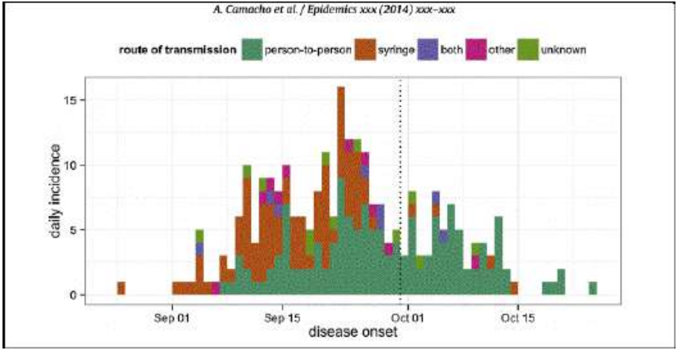
Measles Incidence by
Thailand province (1980-2001).



***Data provided by Derek Cummings (JHU).*

Human Behavior Modifies Disease Fade Out

- Strong evidence hospital and person-to-person transmission declined over the course of the outbreak.
- Community stopped coming to the outpatient department as they associated the epidemic with the hospital.
- Suspicions governed the people who did not touch the corpses.



EPIDEMICS, v. 9, pp. 70-78, 2014, Zaire, 1976 Ebola

Outline

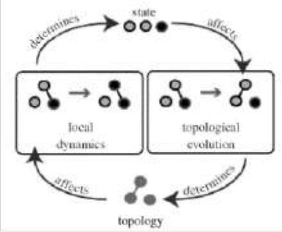
- Dynamics of Stochastic Adaptive Networks
  - Epidemics on homogeneous networks
  - Human behavior of avoidance
- Extinction and control
  - Extinction in an adaptive network
  - Vaccination and extinction enhancement
- Analyzing fluctuations to extinction
  - All-to-all networked coupled populations
  - Using a dynamical system framework to solve the problem
- Extending fluctuation analysis to networks
  - Homogeneous networks
  - General theory of heterogeneous networks
  - Optimal control on heterogeneous networks
- Conclusions

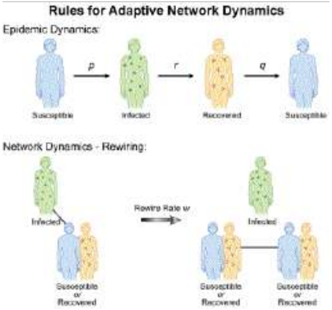
Dynamics of Stochastic Adaptive Networks

- In real networks nodes and links change in time-Dynamic networks

Dynamics of Stochastic Adaptive Networks

- In real networks nodes and links change in time-Dynamic networks
- Node dynamics affects network geometry; Network geometry affects node dynamics
- Feedback loop interaction
- Adaptive networks applications
  - Human social networks
  - Fads, terrorist networks
  - Self healing networks
  - Swarming of autonomous agents
  - Immune system networks
  - Biological networks (e.g., food webs)





Epidemics on Adaptive Social Networks

Epidemic dynamics: Avoidance Behavior

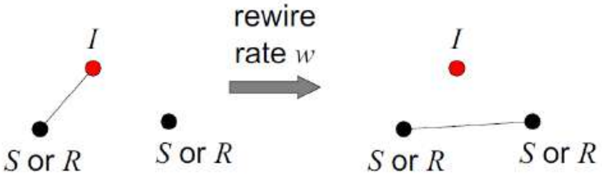
$$S \xrightarrow{p} I \xrightarrow{r} R \xrightarrow{q} S$$

S: susceptible
I: infected
R: recovered

N_{AB}: AB links

p: infection rate
r: recovery rate
q: resusceptibility rate
w: rewiring rate

Network dynamics—rewiring:



Run Monte Carlo simulation for $N=10^4$ nodes, $K=10^5$ links

(Shaw and Schwartz PRE 77: 066101, 2008)

Mean Field Approximation

$$S \xrightarrow{p} I \xrightarrow{r} R \xrightarrow{q} S$$

- Node dynamics—depends on node pairs (links)

$$\dot{P}_S = qP_R - p\frac{K}{N}P_{SI},$$

$$\dot{P}_I = p\frac{K}{N}P_{SI} - rP_I,$$

$$\dot{P}_R = rP_I - qP_R.$$

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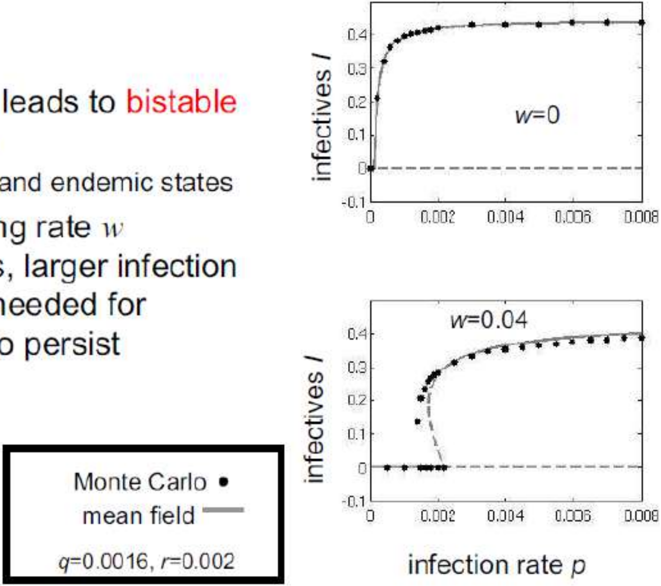
- Link dynamics—depends on triples

$$\dot{P}_{SI} = 2p\frac{K}{N}\frac{P_{SS}P_{SI}}{P_S} + qP_{IR} - rP_{SI} - \boxed{wP_{SI}} - p\left(P_{SI} + \frac{K}{N}\frac{P_{SI}^2}{P_S}\right)$$

Creation of New States

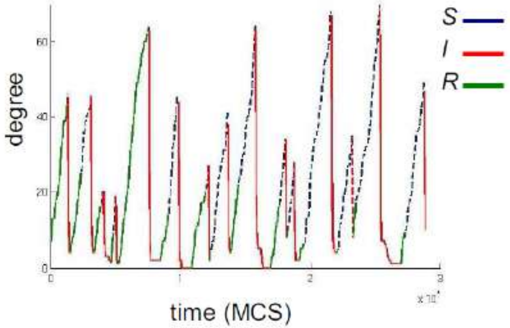
- Rewiring leads to bistable behavior
  - Extinct and endemic states
- As rewiring rate w increases, larger infection rate p is needed for disease to persist



Network structure analysis-Degree distribution

q=0.0016, r=0.002

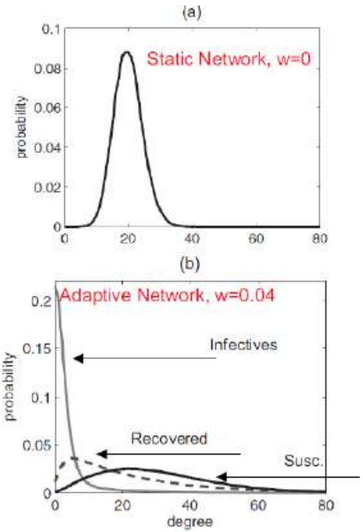
Time series for degree of a single node:



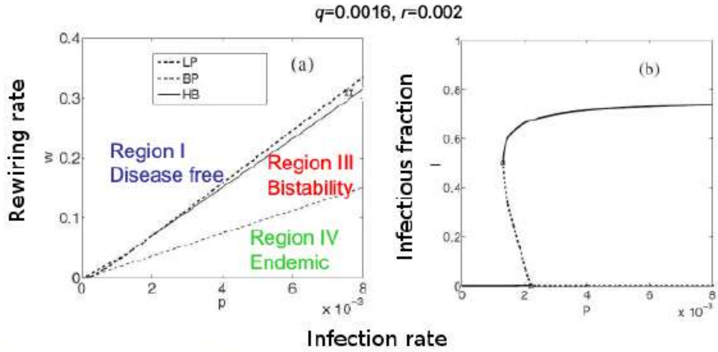
Node degree cycles in time

I loses links

R and S gain links



Bifurcation Structure Analysis: SIRS Mean Field Model



Region I – one stable DFE

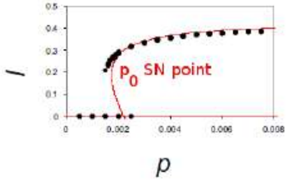
Region II – Stable DFE and unstable state-Oscillations can occur!

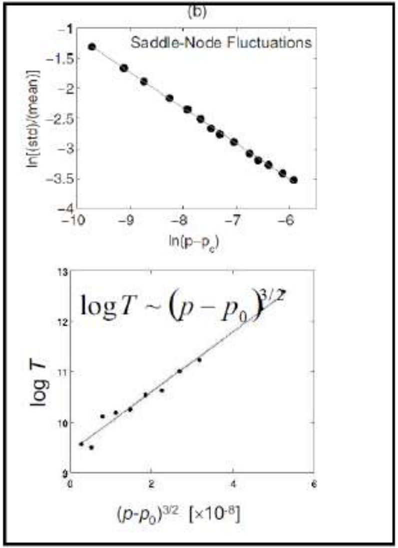
Region III – Bistability

Region IV – One stable endemic state

Fluctuations and Extinction Times

- Lifetime is defined as the time to extinction of I nodes
- Fluctuations increase near the Saddle-Node point p_0 (Bistable state)
  - Scaling of fluctuations explained by noise-induced dynamics near a saddle-node point
- Mean lifetime T of the endemic state becomes shorter near the bifurcation point
- Lifetime scaling is consistent with a saddle-node bifurcation





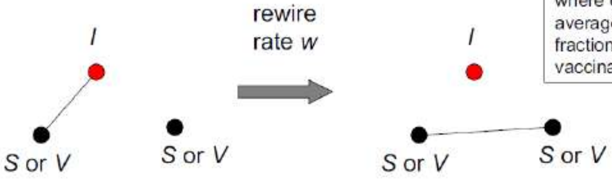
Extinction and Control in Adaptive Networks

Adaptive network with vaccinations

Epidemic dynamics:

$$
\begin{tikzcd}
S \arrow[r, shift left, "p"] \arrow[d, shift right, "\eta(t)"'] & I \arrow[l, shift left, "r"] \\
V \arrow[u, shift right, "q"'] &
\end{tikzcd}
$$

Network dynamics—rewiring:



S: susceptible
I: infected
V: vaccinated

p: infection rate
r: recovery rate
η(**t**): vaccination rate
q: resusceptibility rate
w: rewiring rate

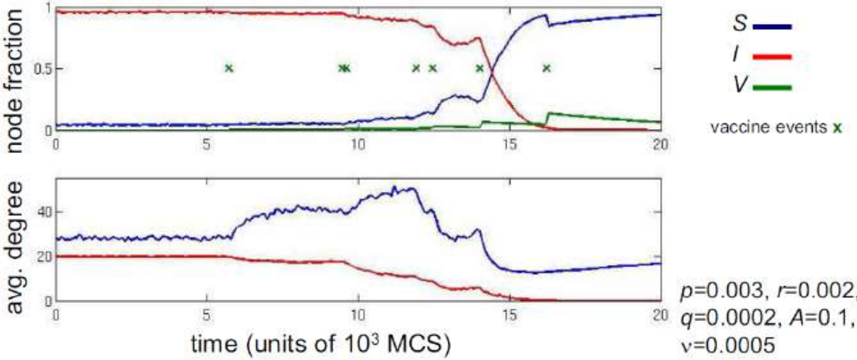
Vaccination rate is Poisson where events happen with average frequency ν and a fraction A of susceptibles are vaccinated in each event.

Run Monte Carlo simulation for N=10^4 nodes, K=10^5 links

L. B. Shaw and I. B. Schwartz, Phys. Rev. E (2010).

Effect of vaccination and rewiring on degree

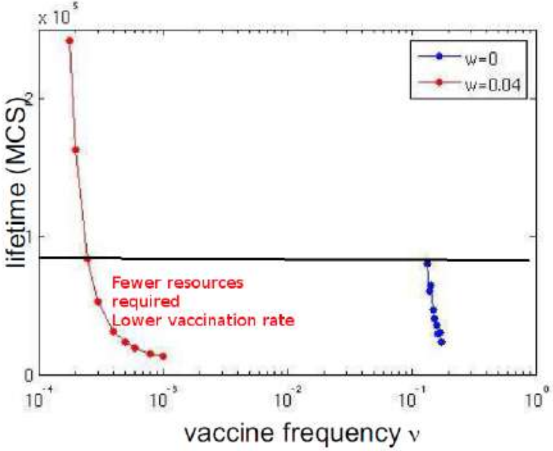
- Vaccination occurs on susceptible nodes
- In the adaptive network, susceptible nodes have higher degree due to rewiring
- Vaccination of high degree nodes provides better protection (e.g., Pastor-Satorras and Vespignani PRE 65: 036104, 2002)
- In the static network, high degree nodes tend to be infected and are not vaccinated



p=0.003, r=0.002, q=0.0002, A=0.1, ν=0.0005

Adaptive network with vaccinations

- Poisson-distributed pulse vaccine control
- Compute lifetime of the infected state
- Average over 100 runs
- Rewiring in combination with vaccination significantly shortens the disease lifetime



p=0.003, r=0.002, q=0.0002, A=0.1

Analyzing Fluctuations to Extinction

All-to-All Connected Networks

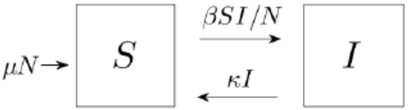
Basic SIS model-All-to-All Coupled Population Network

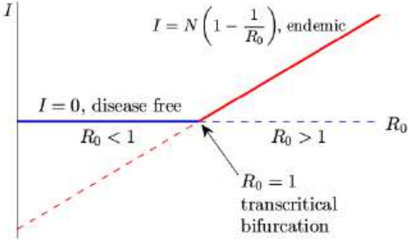
Compartmental model - No network structure:

Two state variables:
Susceptibles, S
Infectives, I Total population size, N

$S + I = N$
Assume N is large.

Parameters:
birth and death rates, μ
contact rate, β
recovery rate, κ





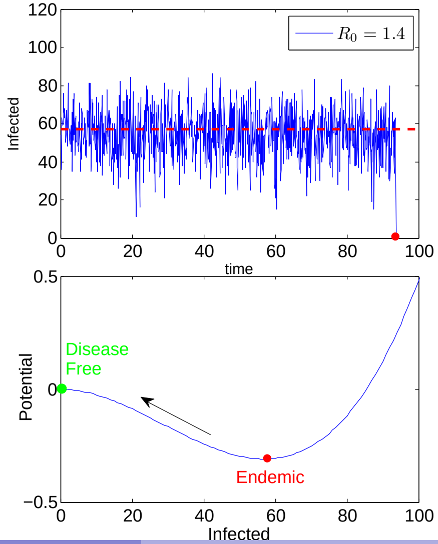
Reproductive infection rate
$R_0 = \beta/(\mu + \kappa)$
Distance to the bifurcation point

Parameters for diseases available in Anderson and May (1991)

Stochastic modeling

There exists randomness, or noise, in the finite N model *

- Internal noise: Randomness of the interactions in the system
- Extinction - Analogous to arbitrarily small noise inducing escape of a particle from a potential well.

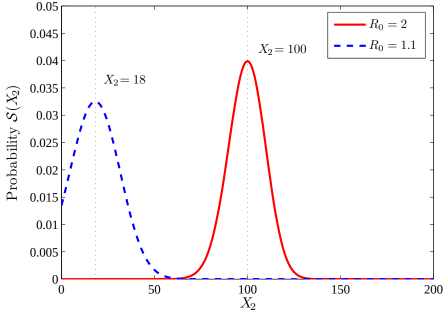


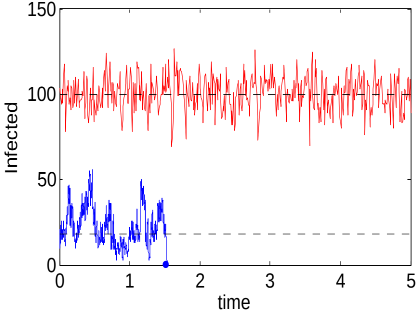
*Schwartz et al J R Soc Interface 8: 1699-1707 (2011)

Characterizing the "almost constant" density

The extinct state ($X_2 = I = 0$) is an absorbing boundary and the system approaches it as $t \to \infty$.

However, if the population size is sufficiently large, the probability density will be Quasi-stationary- $\partial \rho / \partial t \approx 0$.





If $\partial \rho / \partial t \approx 0$, then the value of $\rho(0, t)$ is exponentially small and we define extinction as a rare event.

Master Equation Approach-Modeling the Density

Consider a well-mixed finite population of size N

- Discrete state vector $\mathbf{X} = (S, I, R, ...)$.
- Probability $\rho(\mathbf{X}, t)$ of finding the system in state $\mathbf{X}$ at time t:
- Random state transition rates of increment $\mathbf{r}$: $W(\mathbf{X}, \mathbf{r})$.

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The master equation definition

$$\frac{\partial \rho(\mathbf{X}, t)}{\partial t} = \sum_{\mathbf{r}} [\underbrace{W(\mathbf{X} - \mathbf{r}; \mathbf{r})\rho(\mathbf{X} - \mathbf{r}, t)}_{\substack{\text{the gain to state } \mathbf{X} \\ \text{from state } \mathbf{X}\text{-}\mathbf{r}}} - \underbrace{W(\mathbf{X}; \mathbf{r})\rho(\mathbf{X}, t)}_{\substack{\text{the loss of state } \mathbf{X} \\ \text{to other states}}}].$$

It is the gain-loss equation for the probabilities of the separate states **X**.

Van Kampen, N.G., Stochastic processes in physics and chemistry, Elsevier (1992).

Approximating quasi-stationary solutions

To analyze the master equation, make the ansatz:

$$\rho(\mathbf{X}, t) \approx \exp(-N\mathcal{S}(\mathbf{q})), \text{ for } \mathbf{q} = \mathbf{X}/N.$$

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Large N assumption: Action $\mathcal{S}$ satisfies Hamilton-Jacobi equation:

$$\frac{\partial \mathcal{S}}{\partial t} + H\left(\mathbf{q}, \frac{\partial \mathcal{S}}{\partial \mathbf{q}}\right) = 0,$$

with Hamiltonian

$$H(\mathbf{q}; \mathbf{p}) = \sum_{\mathbf{r}} w(\mathbf{q}; \mathbf{r})[\exp(\mathbf{p} \cdot \mathbf{r}) - 1]$$

where w($\mathbf{q}$;$\mathbf{r}$)= W($\mathbf{q}$;$\mathbf{r}$)/N Conjugate momenta $\mathbf{p} = \partial\mathcal{S}/\partial\mathbf{q}$.

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Since we assume the distribution is quasi-stationary, $\frac{\partial \mathcal{S}}{\partial t} = 0$.

Kubo, et al., J. Stat. Phys. 9 (1973); Gang, PRA, 36 (1987); Dykman, et al., J. Chem Phys, 100 (1994); Elgart, et al., PRE, 70 (2004); and many others.

The most likely observed paths to extinction

Shape of distribution described by the Hamiltonian equations of motion:

$$\dot{\mathbf{q}} = \partial_{\mathbf{p}} H(\mathbf{q}, \mathbf{p}; t), \quad \dot{\mathbf{p}} = -\partial_{\mathbf{q}} H(\mathbf{q}, \mathbf{p}; t), \quad \text{plus BC}$$

Deterministic system describe the dynamics of the stochastic system.

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- A heteroclinic trajectory connects the endemic state to the stochastic extinction state.

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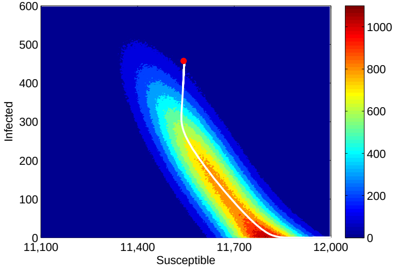
We call it the optimal path to extinction.

The optimal path maximizes the PDF of the pre-history solutions that go extinct.



The Stochastic SIS model - Topology*

Constrain the population, N: $S + I = N$

Hamiltonian equations of motion- scaled infectives $x_2 = I/N$ and momenta p_2

The Stochastic SIS model - Topology*

Constrain the population, N: $S + I = N$

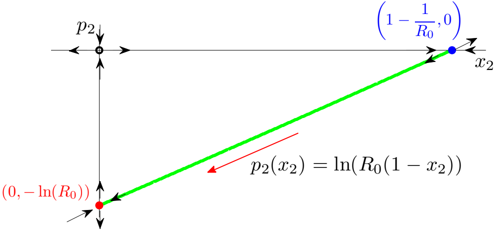
Hamiltonian equations of motion- scaled infectives $x_2 = I/N$ and momenta p_2

The Hamiltonian system has three steady states: $\boxed{R_0 = \beta/(\mu+\kappa) > 1}$

- The disease free equilibrium, $(x_2, p_2) = (0, 0)$.
- The endemic state, $(x_2, p_2) = (1 - \frac{1}{R_0}, 0)$.
- The stochastic extinction state, $(x_2, p_2) = (0, -\ln(R_0))$.

Find the action along the path

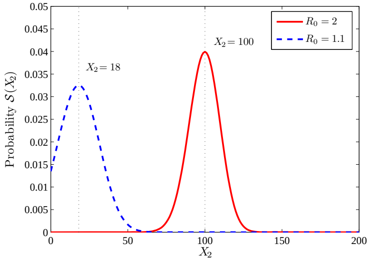
$$\mathcal{S}_{opt} = \int_{1-\frac{1}{R_0}}^{0} -\ln(R_0(1-x_2))\, dx_2$$
$$= \ln(R_0) - 1 + \frac{1}{R_0}$$

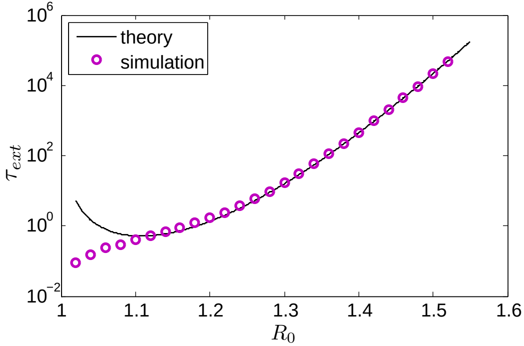


Forgoston et al Bull Math Bio 73: 495-514 (2011)

The Stochastic SIS model - Mean Time to extinction

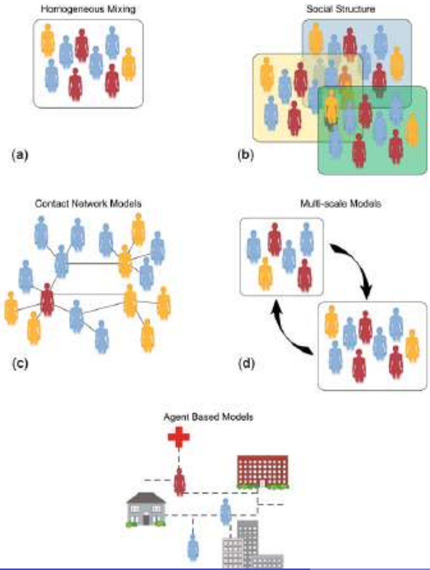
Approximate the mean time to extinction $\tau_{ext} \propto 1/\rho_{ext}$





Doering, et al., Multiscale Model. Simul. (2005); Dykman et al, PRL 101 (2008);
Schwartz et al, J Stat Mech, P01005 (2009).

Fluctuation Analysis on Networks



- Extend fluctuation analysis and control to stochastic networks
- Examine stochastic networks with no rewiring

  Rewiring rate $w = 0$

Rewiring for adaptation, IB Schwartz and LB Shaw Physics 3 (17) (2010)

Homogeneous Networks-Extinction

- Structure of the mean field:

  N nodes, K links
  $2K/N$ mean degree

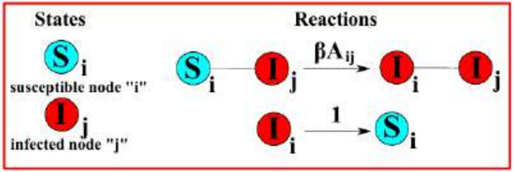
  $\mathbf{X} = [N_S, N_I, N_{SS}, N_{SI}, N_{II}]$
  N_S, N_I are nodes
  N_{SS}, N_{SI}, N_{II} are links
  A_{ij} is the adjacency matrix, λ is max eigenvalue

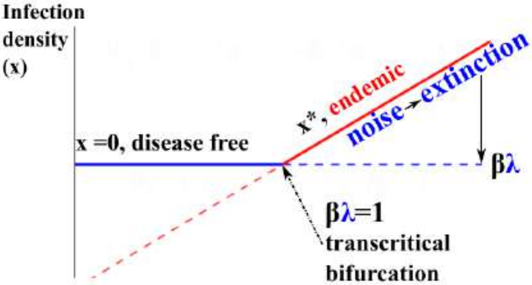
- $R_0 \equiv$ Reproduction infection no. where

  $R_0 \equiv \beta\lambda$

  BS Lindley, LB Shaw, IB Schwartz EPL 108, 58008 2014

- Large fluctuation analysis for SIS stochastic networks





Modeling the Transitions for a Homogeneous Network

- Constructive approach - Perturb from global to local population and track the optimal path
- $\epsilon = 0$ corresponds to all to all coupling
- $\epsilon = 1$ corresponds to local network coupling
- β is infection rate
- r is a recovery rate
- $\mathbf{X} = [N_S, N_I, N_{SS}, N_{SI}, N_{II}]$

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Transition rates[a]

$$
\begin{aligned}
W(\boldsymbol{X}, \nu_1) &= \epsilon\beta N_{SI} && S \rightarrow I \quad \textit{Local} \\
W(\boldsymbol{X}, \nu_2) &= (1-\epsilon)\beta \frac{2K}{N}\frac{N_S N_I}{N} && S \rightarrow I \quad \textit{Global} \\
W(\boldsymbol{X}, \nu_3) &= rN_I, && I \rightarrow S \quad \textit{Recovery}
\end{aligned}
$$

[a]Tim Rogers et al J. Stat. Mech. Theory and Experiment, PO8018 2012

Modeling the Transitions for a Homogeneous Network

- Constructive approach - Perturb from global to local population and track the optimal path
- $\epsilon = 0$ corresponds to all to all coupling
- $\epsilon = 1$ corresponds to local network coupling

- β is infection rate
- r is a recovery rate
- $\mathbf{X} = [N_S, N_I, N_{SS}, N_{SI}, N_{II}]$

Transition rates[a]

$$W(\boldsymbol{X}, \nu_1) = \epsilon\beta N_{SI} \qquad S \to I \quad \textit{Local}$$

$$W(\boldsymbol{X}, \nu_2) = (1-\epsilon)\beta \frac{2K}{N}\frac{N_S N_I}{N} \qquad S \to I \quad \textit{Global}$$

$$W(\boldsymbol{X}, \nu_3) = rN_I, \qquad I \to S \quad \textit{Recovery}$$

Increments

$$\boldsymbol{\nu}_1 = \left[-1, 1, -\frac{2N_{SS}}{S}, \frac{2N_{SS}}{S} - \left(1 + \frac{N_{SI}}{S}\right), \left(1 + \frac{N_{SI}}{S}\right)\right]$$

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$$\boldsymbol{\nu}_2 = \left[1, -1, \frac{2N_{SI}}{I}, -\frac{N_{SI}}{I} + \frac{2N_{II}}{I}, -\frac{2N_{II}}{I}\right].$$

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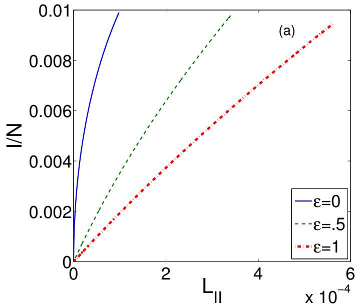
$$H(\boldsymbol{x}, \boldsymbol{p}) = \sum_{k=1}^{3} w(\boldsymbol{x}, \boldsymbol{\nu}_k)(e^{\boldsymbol{p}\cdot\boldsymbol{\nu}_k} - 1)$$

Optimal paths for a Stochastic Network

Computed paths from theory

Lindley etal, Physica D 255, 22-30 (2013)



Infective fraction vs II links

$p = 1.03x10^{-4}$, $r = 0.002$,$N = 10^4$,$K = 10^5$

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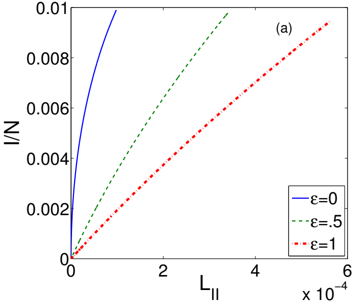

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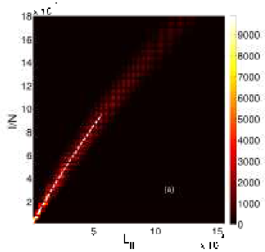
Compared to Monte Carlo PDF ($\epsilon = 1$)



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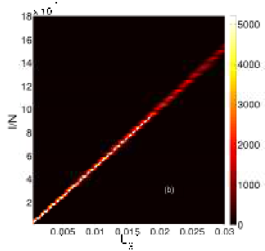
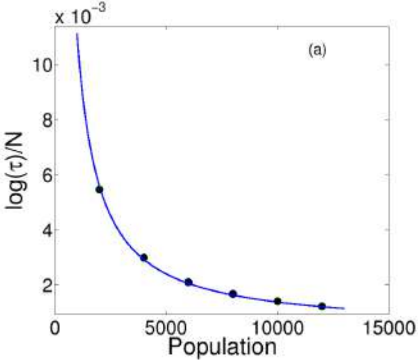


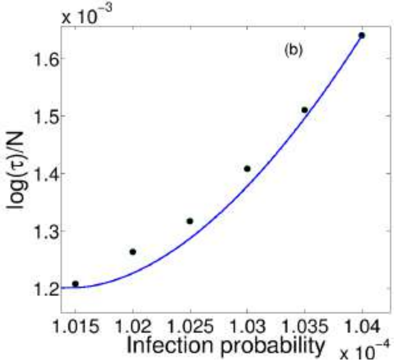
SI Links

Extinction Times for a Stochastic Network-No rewiring

As a function of population size



As a function of infection probability



$$log(\tau)/N \approx S + log(B)/N$$

Pre-factor at $\epsilon = 0$: $B = \frac{\sqrt{2\pi \frac{R_0^{eff}}{N}}}{r(R_0^{eff}-1)^2}$: $R_0^{eff} = 2pK/(Nr)$

No fitting parameters

Extinction in heterogeneous networks-General Theory

Extinction in heterogeneous networks-General Theory

Consider SIS transitions on network having degree distribution g_k

- Assume adjacency matrix follows : $A_{ij} \approx k_i k_j / (N \langle k \rangle)$.
- Bin infected nodes of degree k, I_k
- Transition rates

  Infection rate $w_k^{inf}(\mathbf{I}) = \beta k (N_k - I_k) \sum_{k'} k' I_{k'} / (N \langle k \rangle)$ with $I_k \to I_k + 1$

  Recovery rate $w_k^{rec}(\mathbf{I}) = \alpha I_k$ with $I_k \to I_k - 1$.

  $N_K \equiv g_k N$

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Master Equation

$$\frac{\partial \rho}{\partial t}(\mathbf{I}, t) = \sum_k w_k^{inf}(\mathbf{I} - \mathbf{1}_k)\rho(\mathbf{I} - \mathbf{1}_k, t) - w_k^{inf}(\mathbf{I})\rho(\mathbf{I}, t)$$

$$+ \sum_k w_k^{rec}(\mathbf{I} + \mathbf{1}_k)\rho(\mathbf{I} + \mathbf{1}_k, t) - w_k^{rec}(\mathbf{I})\rho(\mathbf{I}, t),$$

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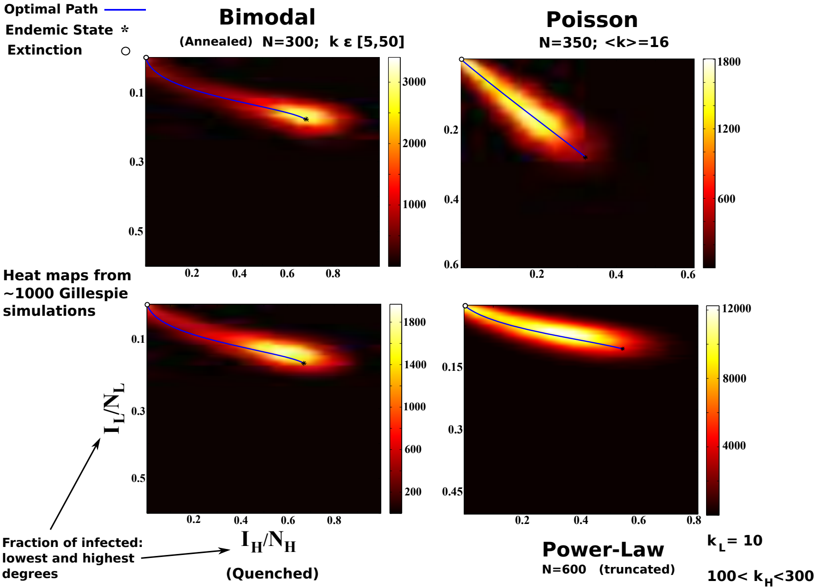
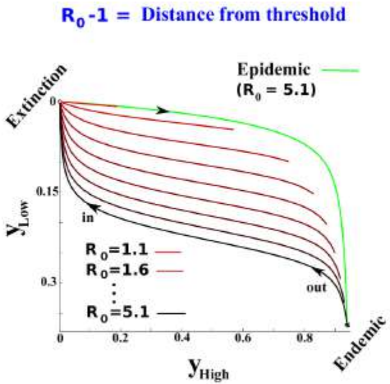
Hamiltonian from WKB ansatz ($\mathbf{x} = \mathbf{I}/N$):

$$H(\mathbf{x}, \mathbf{p}) = \sum_k \left[\beta k (g_k - x_k)(e^{p_k} - 1) \sum_{k'} \frac{k' x_{k'}}{\langle k \rangle} + \alpha x_k (e^{-p_k} - 1) \right].$$

Extinction paths in hetergeneous networks



Extinction Path Projections in a Power Law Network



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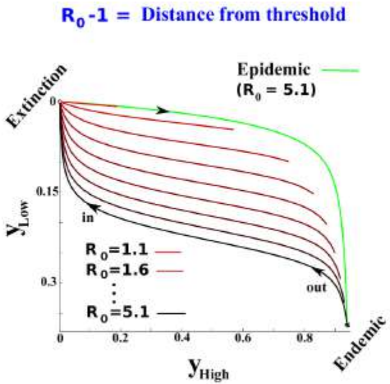
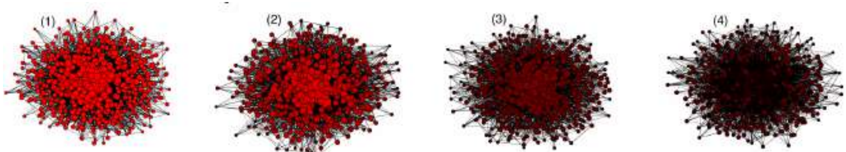
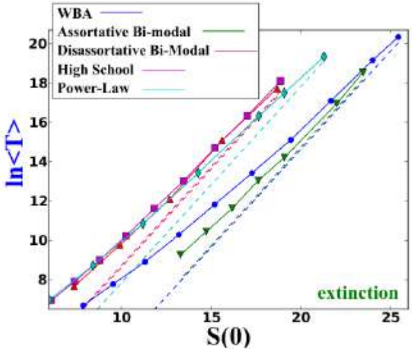




J. Hindes and I. B. Schwartz, Phys. Rev. Lett. **117**, 028302 (2016).

Extinction times and PDF for real networks

Extinction Times vs. Action



Symbols simulation - Dashed lines theory

Extinction times and PDF for real networks

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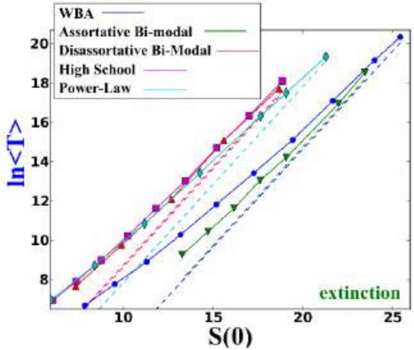
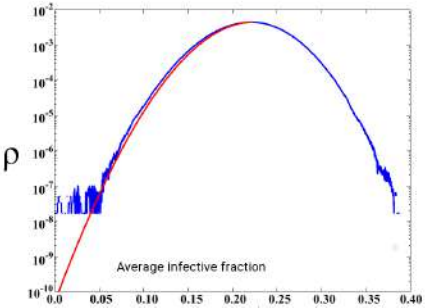


Symbols simulation - Dashed lines theory

Probability vs Average infection fraction



High School Network-788 nodes

Theory is plotted in red

Near bifurcation : $ln < T > \propto N \frac{<k^2>^3}{<k^3>^2} \delta^2$

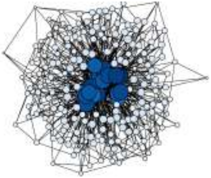
δ is bifurcation distance

Probability exponent decreases with heterogeneity

Optimal external control

Bimodal Network: %90 with
degree 5 and 10% with degree 50

Who to target?



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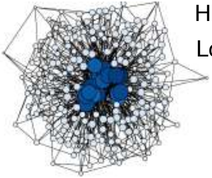

High-degree *recover* rate: $1+\gamma w$

Low-degree *recover* rate: $1+\gamma(1-w)$

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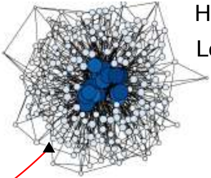

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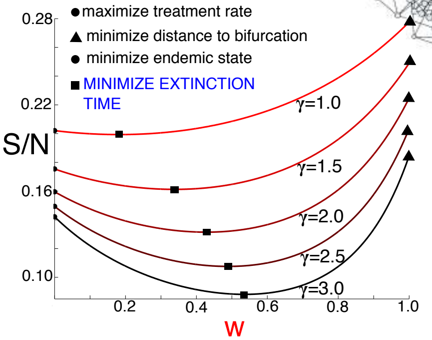

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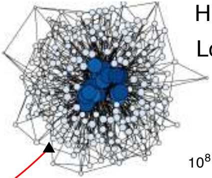

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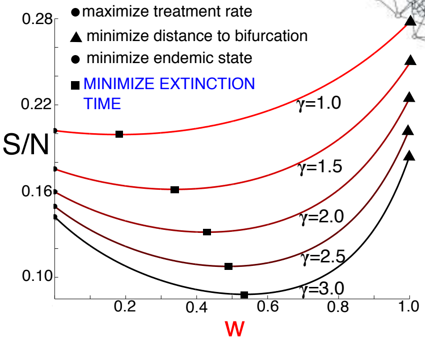

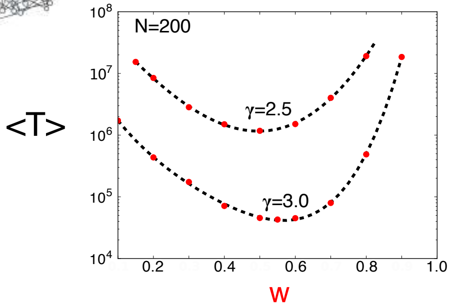

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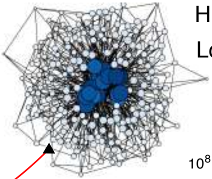

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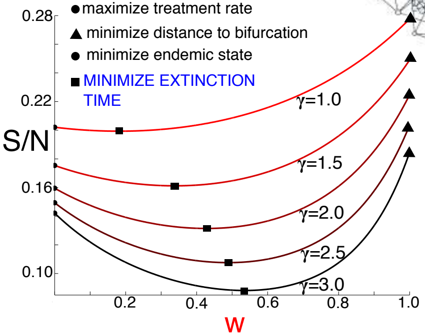

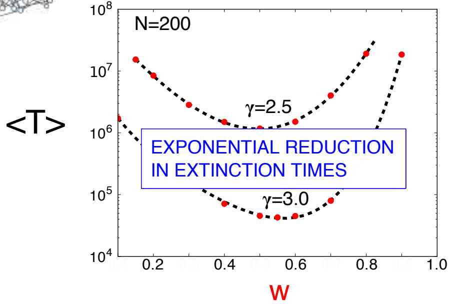

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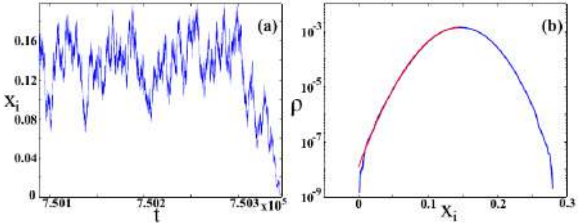
Adaptive Network Control
Enhancing the time to extinction

Adaptive Network Progress

Corrected general extinction theory - statistical moment closure

PDF depends on width of susceptible distribution

(a) Infective fraction vs. time (b) PDF of infective nodes theory(red)



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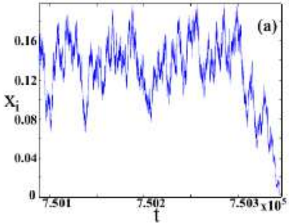

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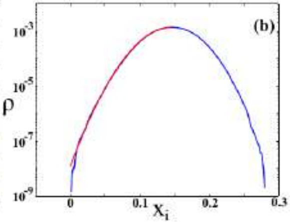
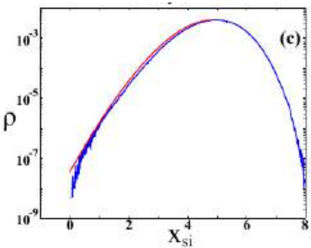
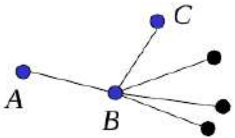


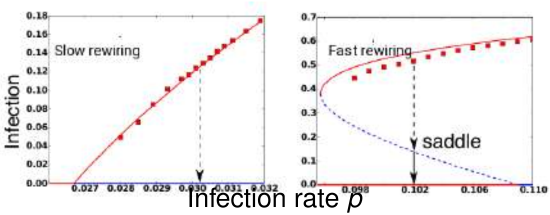
PDF of infective-susceptible links theory(red)



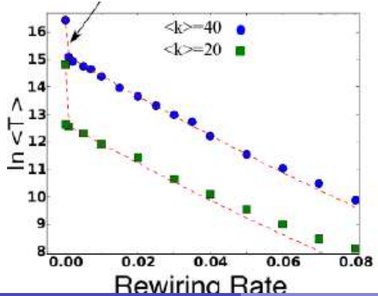


J Hindes, IB Schwartz, LB Shaw, PRE 97 (1), 012308(2018) , arXiv:1708.03193

Adaptive Network Progress



Adaptivity due to rewiring introduces new states

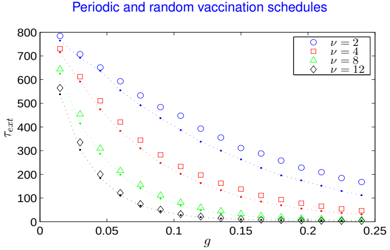


Initial small rewiring causes a discontinuous decrease in extinction times

Extinction times depend on network heterogeneity-Suseptible distribution become more homogeneous.

Conclusions

- A general network formulation of extinction for a disease in a finite population is developed-including adaptive networks.
- We can quantify the effect of treatment programs on extinction rates.
- For limited resources, larger treatment pulses less often are most effective.
- Used optimal paths to predict extinction times in terms of bifurcation parameters for general networks.
- Optimal control may be designed based on minimiziing the action as a function of degree.



Related papers

On Extinction

- J Hindes, IB Schwartz, LB Shaw, Enhancement of large fluctuations to extinction in adaptive networks, PRE 97, 012308 (2018)
- Jason Hindes and Ira B. Schwartz, "Epidemic Extinction and Control in Heterogeneous Networks", Physical Review Letters, 117, 028302 (2016).

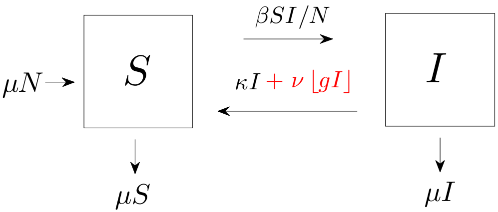
  J. Hindes, I. B. Schwartz, Epidemic Extinction Paths in Complex Networks, PRE95 (5), 052317 (2017)
- Klementyna Szwaykowska, Ira B. Schwartz, Luis Mier-y-Teran Romero et al, "Collective motion patterns of swarms with delay coupling: Theory and experiment," Phys. Rev. E 93, 032307 (2016).
- Brandon S. Lindley, Leah B. Shaw, Ira B. Schwartz, “Rare Event Extinction on Stochastic Networks,” arXiv:1411.0017 (2014), and EPL 108 58008(2014)
- Lora Billings, Luis Mier-y-Teran-Romero, Brandon Lindley, Ira B. Schwartz, “Intervention-Based Stochastic Disease Eradication," PLOS ONE 8 (8), e70211 (2013).
- Brandon S. Lindleyand Ira B. Schwartz, “An iterative action minimizing method for computing optimal paths in stochastic dynamical systems,” Physica D 255, 22-30 (2013).
- Brandon S. Lindley, Luis Mier-y-Teran-Romero, and Ira B. Schwartz, “Noise induced pattern switching in randomly distributed delayed swarms,” American Control Conference (ACC), 2013, 4587-45 (2013).
- Max S. Shkarayev, Ira B. Schwartz, Leah B. Shaw, “Recruitment dynamics in adaptive social networks,” Journal of Physics A: Mathematical and Theoretical 46 (24), 245003 (2013)
- Ira B. Schwartz, Eric Forgoston, Simone Bianco, Leah B. Shaw “Converging towards the optimal path to extinction,” J R Soc Interface 8: 1699-1707 (2011).
- Eric Forgoston, Simone Bianco, Leah B. Shaw, Ira B. Schwartz “Maximal sensitive dependence and the optimal path to epidemic extinction," Bull Math Bio 73: 495-514 (2011).
- LB Shaw, IB Schwartz,” Enhanced vaccine control of epidemics in adaptive networks,” Physical Review E 81 (4), 046120 (2010)
- IB Schwartz, LB Shaw, “Rewiring for adaptation,” Physics 3 (17) (2010)

Future Directions and Things Not Discussed

- How does complex network structure affect route to extinction?
  - Topolgy, deterministic time dependent contacts, etc..
  - Beyond pairwise approximation
  - Non-Markovian assumptions
- Extend theory to other networks
  - Switching and adaptive networks
  - Networks with delays
  - Noise...

Full SIS treatment model (Unconstrained population)

Remove fixed population constrain - *N* fluctuates



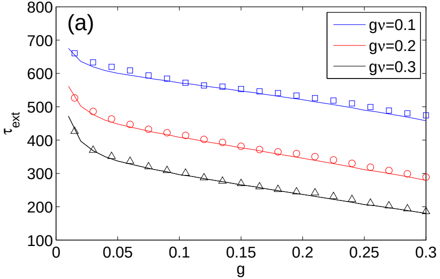
$$W\big((S, I); (\lfloor gI \rfloor, -\lfloor gI \rfloor)\big) = \nu, \quad \text{treatment}.$$

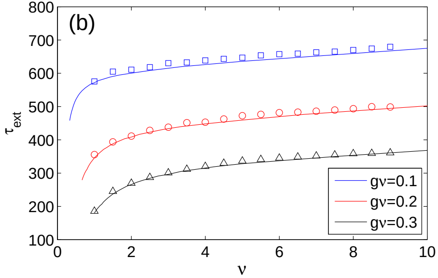
Finite resources of SIS treatment - Optimal schedule

$g\nu =$ constant

Decrease in mean time to extinction as the g increases and ν decreases.

Larger fraction treated fewer times per year is most effective.





Results: average of 10,000 simulations

Master equation theory (solid)

MC simulation (symbols)

Parameters: $g\nu =$ constant

$\beta = 105$ year^{-1}

$N = 8000$ people

Billings et al PLOS ONE 8 (8), e70211 (2013)