Adaptive Networks: Coevolution of Disease and Topology

Vincent Marceau, Pierre-André Noël, Laurent Hébert-Dufresne, Antoine Allard and Louis J. Dubé

Département de Physique, de Génie Physique, et d'Optique, Université Laval, Québec, Canada

Introduction

The majority of ***network-based*** *epidemiological models rely on the paradigm of* ***static networks****. They neglect* ***mutual interactions*** *thay may exist between the* ***states of the nodes*** *and the underlying* ***network topology****.*

Adaptive networks *are a new class of networks in which there exists a* ***feedback loop*** *between the dynamics* ***on*** *the network and the dynamics* ***of*** *the network.*

Existing analytic approaches*: based on mean-field formalisms derived from low-order moment closure approximations.*

Advantages

- *Low complexity.*
- *Sufficient to highlight novel dynamical features.*

Main drawback

- *Unable to reproduce the time evolution of disease and topology from various initial configurations.*

Why adaptive networks in epidemiology?

Healthy individuals may avoid contacts with those who are infectious to reduce their chances of catching the disease.

Our goal is to develop an ***integrated analytic approach*** *able to describe with accuracy the* ***time evolution*** *of both dynamical elements of adaptive networks,* ***process*** *and* ***structure****.*

Model

Network model. *A population of N individuals is represented by a* ***contact network*** *of N nodes and M links.*

SIS dynamics. *At a given time t, each individual is in a specific* ***state****:*

Susceptible *individuals do not have the disease but can get infected by their infectious neighbors.*

Infectious *individuals have the disease and can transmit it to their susceptible neighbors. They recover and become susceptible again after a given infectious period.*

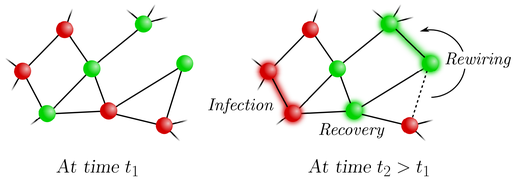
Adaptive rewiring. *For every link between a susceptible (S) and an infectious (I) individual (SI link), the S individual can decide to* ***break*** *the connection and* ***reconnect*** *it with another randomly chosen S individual in the network.*

[Gross et al., 2006]

Important parameters:

- α : *rate at which I individuals recover.*
- β : *rate at which I individuals infect their S neighbors.*
- γ : *rate at which links are rewired.*
- $\langle k \rangle$: *average degree of the network ($\langle k \rangle = 2M/N$).*

Illustration:



At time t_1 *At time $t_2 > t_1$*

Formalism

Improved compartmental formalism *in which nodes are categorized according to* ***their state*** *but also according to* ***the state of their neighbors****.*

[Noël et al., 2009, Marceau et al.]

- $S_{kl}(t)$: *fraction of nodes that are susceptible and have k neighbors of which l are infectious at time t.*
- $I_{kl}(t)$: *fraction of nodes that are infectious and have k neighbors of which l are infectious at time t.*
- $p_k(t)$: *fraction of nodes that have k neighbors at time t.*

Zeroth order moments:

$$S \equiv \sum_{kl} S_{kl} \ , \ I \equiv \sum_{kl} I_{kl}$$

First order moments:

$$S_S \equiv \sum_{kl} (k-l) S_{kl} \ , \ S_I \equiv \sum_{kl} l S_{kl} \ , \ I_S \equiv \sum_{kl} (k-l) I_{kl} \ , \ I_I \equiv \sum_{kl} l I_{kl}$$

Second order moments:

$$S_{SI} \equiv \sum_{kl} (k-l) l S_{kl} \ , \ S_{II} \equiv \sum_{kl} l^2 S_{kl} \ , \ \textit{etc.}$$

Dynamical equations

$$\begin{aligned}
\frac{dS_{kl}}{dt} =& \alpha I_{kl} - \beta l S_{kl} + \alpha \Big[(l+1) S_{k(l+1)} - l S_{kl} \Big] \\
&+ \beta \frac{S_{SI}}{S_S} \Big[(k-l+1) S_{k(l-1)} - (k-l) S_{kl} \Big] \\
&+ \gamma \Big[(l+1) S_{k(l+1)} - l S_{kl} \Big] + \gamma \frac{S_I}{S} \Big[S_{(k-1)l} - S_{kl} \Big] \\
\frac{dI_{kl}}{dt} =& - \alpha I_{kl} + \beta l S_{kl} + \alpha \Big[(l+1) I_{k(l+1)} - l I_{kl} \Big] \\
&+ \beta \frac{S_{II}}{S_I} \Big[(k-l+1) I_{k(l-1)} - (k-l) I_{kl} \Big] \\
&+ \gamma \Big[(k-l+1) I_{(k+1)l} - (k-l) I_{kl} \Big]
\end{aligned}$$

Initial conditions. *The dynamics is initialized by infecting at random a fraction ϵ of the individuals in the population:*

$$S_{kl}(0) = (1-\epsilon) p_k(0) \binom{k}{l} \epsilon^l (1-\epsilon)^{k-l}$$

$$I_{kl}(0) = \epsilon p_k(0) \binom{k}{l} \epsilon^l (1-\epsilon)^{k-l}$$

Conservation of nodes: $S + I = 1$

Conservation of links: $S_S + S_I + I_S + I_I = \langle k \rangle$

Undirected nature of links: $S_I = I_S$

Observables

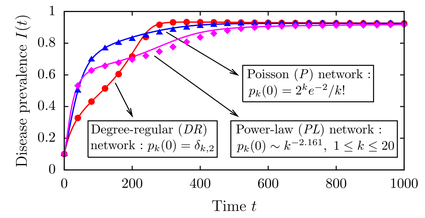
Several ***relevant observables*** *can be readily computed from this formalism. They can help us to understand the interplay between disease and topology in the system.*

- $I(t)$: *Total disease prevalence.*
- $s_k(t) \equiv \sum_l S_{kl}/S$: *Susceptible degree distribution.*
- $i_k(t) \equiv \sum_l I_{kl}/I$: *Infectious degree distribution.*
- $S_I(t)$: *Density of SI links.*
- $\kappa_{IS}^S(t) \equiv S_{SI}/S_I$: *Effective branching factor.*
- $C_{SS}(t) \equiv \dfrac{S_S}{S_S + S_I}$: *Susceptible aggregation coefficient.*

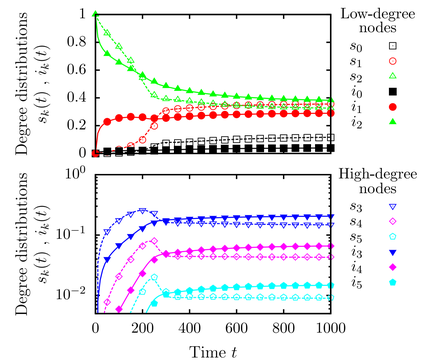
Time evolution

To attest the validity of our approach, we compared ***analytic predictions (lines)*** *with results obtained from* ***Monte-Carlo simulations (symbols)*** *of the dynamics on networks of size $N = 25000$ with $\langle k \rangle = 2$. Parameters used are $\alpha = 0.005$, $\beta = 0.06$, $\gamma = 0.02$ and $\epsilon = 0.1$.*

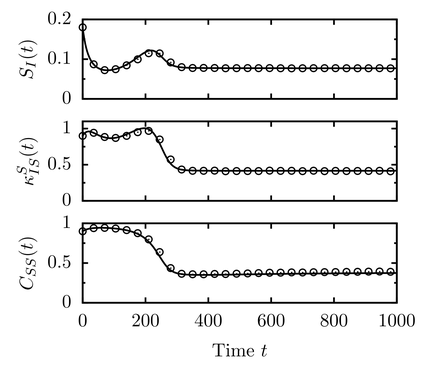
Evolution of the ***disease prevalence*** *$I(t)$ on networks with different initial degree distributions:*



Evolution of the ***susceptible and infectious degree distributions*** *$s_k(t)$ and $i_k(t)$ on a DR initial network:*



Evolution of the ***topological observables*** *$S_I(t)$, $\kappa_{IS}^S(t)$ and $C_{SS}(t)$ on a DR initial network:*

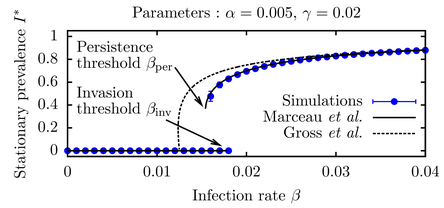


By analyzing the time evolution of disease and topology, we can bring new insights about their ***interplay****. For example, in a DR initial network, the dynamics is characterized by two distinct phases:*

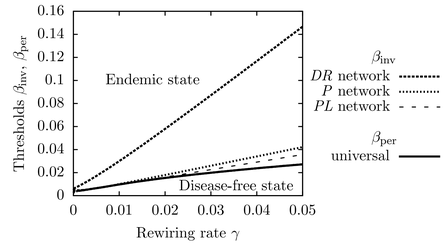
1. *A* ***separation phase****, from $t = 0$ to $t \approx 200$, characterized by a segregation of the network in tightly connected susceptible and infectious communities.*
2. *An* ***invasion phase****, from $t \approx 200$, characterized by the invasion of the susceptible community.*

Stationary states

Our formalism compares favorably with previous approaches about the ***bifurcation structure*** *of systems with initial Poisson networks:*



Location of the ***persistence and invasion thresholds*** *for different initial networks (computed from our formalism):*



Our results suggest two important conclusions:

- *Link rewiring as a control strategy is* ***more efficient*** *on* ***homogeneous networks****.*
- *There exists a* ***universal endemic state****, which depends only on the density parameter $\langle k \rangle$ and interaction parameters α, β, and γ of the system.* ***Initial conditions*** *determine if this state can be reached.*

Conclusion

We have introduced an ***improved compartmental approach*** *able to reproduce with accuracy the* ***complete time evolution of disease and topology*** *on adaptive networks with various initial configurations.*

Future directions

- ***Analytical solution*** *for the endemic state.*
- *Inclusion of* ***more realistic features*** *in the model, e.g. community structure, preferential rewiring.*
- *Simultaneous evolution of the* ***disease*** *and the* ***population awareness****.*

Gross *et al.*, "Epidemic dynamics on an adaptive network," Phys. Rev. Lett. **96** 208701, 2006.

Noël *et al.*, "Time evolution of epidemic disease on finite and infinite networks," Phys. Rev. E **79** 026101, 2009.

Marceau *et al.*, "Adaptive networks: Coevolution of disease and topology," To be published.