Propagation dynamics on networks featuring complex topologies

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

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

Motivation

Most descriptions of propagation dynamics on networks are rooted in the **random graph paradigm**:

- negligeable probability of **loops**; and
- negligeable **correlation** (in both close and long range) between the existence probability of two given links.

In other words: No evident structure in the network topology.

Is this approximation important?

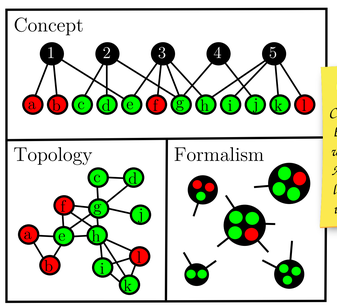
Real networks are usually constructed on precise rules from which structure emerges; e.g. *the friend of my friend is my friend.* Furthermore, dynamics on networks is sensitive to network structure (as will be exposed in the following).

Our goal is to include a **level of substructure** in our description of propagation on networks featuring structured topologies.

Case study

Suceptible-Infectious-Susceptible (**SIS**) model of epidemic spread on **social networks** featuring **community structure**.

Each **individual** belongs to m cliques, each containing n participants (m and n are respectively taken from distributions $\{g_m\}$ and $\{p_n\}$). Links are shared with **probability ϵ** among pairs of participants.



Community structure?

Community structure is defined by densely connected structures with sparser links inbetween. A more general description, with links outside of any structure, is used in Hébert-Dufresne et al.

At a given time, each individual is in a specific **state**:

- **S**usceptible individuals do not have the disease but can get infected by contact with infectious at rate τ.
- **I**nfectious individuals have the disease and can transmit it to susceptible neighbors at rate τ. They can also recover from the disease at rate r.

For static properties of the topology (e.g. degree distribution, clustering coefficient, giant component) see article by M.E.J. Newman (2003).

Our approach, based on the **mean-field coupling of topological patterns and topological elements** of a **social structure** , yields:

- **time evolution** of the state distribution of cliques and individuals, as well as the total epidemic size at any given time;
- analytical solution for the **stable state** (i.e. global state I^* where total infections equal total recoveries); and
- analytical solution for the **epidemic threshold** (i.e. infection rate τ_c which allows a macroscopic final epidemic size).

Philosophy and mathematics

Basics

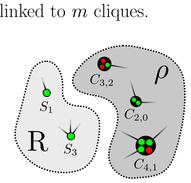
A **dynamical mean-field** description of **compartmentalized behaviors** for both the **network elements** (individuals) and their **recurrent topological patterns** (cliques).

We follow the probability densities within two state ensembles:

- $C_{n,i}$, cliques whose population is n with i infectious; and
- S_m, individuals who are susceptible and linked to m cliques.

with two mean-fields of infection inflow:

- $R(t)$, mean infectious neighbors per clique for susceptible individuals
- $\boldsymbol{\rho}(t)$, mean infectious neighbors outside of a given clique per susceptible.



$$R(t) = \epsilon \left[\frac{\sum_{n,i} i(n-i)C_{n,i}}{\sum_{n,i}(n-i)C_{n,i}}\right] \quad \text{and} \quad \rho(t) = \left[\frac{\sum_m m(m-1)S_m}{\sum_m mS_m}\right] R(t).$$

Master equations

$$\begin{aligned}\frac{dC_{n,i}}{dt} = &\ \tau\,(n-i+1)\,(\epsilon\,(i-1)+\rho(t))\,C_{n,i-1}(t) + r\,(i+1)\,C_{n,i+1}(t) \\ &- \tau\,(n-i)\,(\epsilon i+\rho(t))\,C_{n,i}(t) - riC_{n,i}(t) \\ \frac{dS_m}{dt} = &\ r(1-S_m(t)) - \tau m S_m(t) R(t)\end{aligned}$$

Observables

$$\langle i\rangle_n = \frac{1}{np_n}\sum_i iC_{n,i}(t) \qquad \text{[Disease prevalence in cliques of size } n\text{]}$$

$$I(t) = 1 - \sum_m S_m(t) \qquad \text{[Global disease prevalence]}$$

Stable state, I^*: ρ^* is obtained from its transcendental definition using $\{S_m^*\}$ obtained from their master equation and $\{C_{n,i}^*\}$ fixed by the conservation equation $\sum_i C_{n,i} = p_n$ and a recursive solution:

$$\begin{aligned}C_{n,i+1}^* = &\frac{1}{(i+1)r}\Big\{\left[\tau(n-i)(i\epsilon+\rho^*)+ri\right]C_{n,i}^* \\ &- \left[\tau(n-i+1)\left((i-1)\epsilon+\rho^*\right)\right]C_{n,i-1}^*\Big\}.\end{aligned}$$

Epidemic threshold, τ_c (phase transition in τ where non-null stable state appears): given by the only real positive solution of:

$$\frac{\mu_2}{\nu}\sum_{n,i} p_n \left(\frac{\epsilon\tau_c}{r}\right)^i \left[\prod_{j=0}^{i}(n-j)\right] = 1$$

where μ_2 is the mean number of excess cliques per individual and ν the mean number of individuals per clique.

Stochastic model and results

Initial conditions

A fraction I_0 of individuals are randomly chosen to be infectious at $t = 0$:

$$S_m(0) = (1-I_0)g_m \quad \textit{and} \quad C_{n,i}(0) = p_n\binom{n}{i}(I_0)^i(1-I_0)^{n-i}$$

Random networks

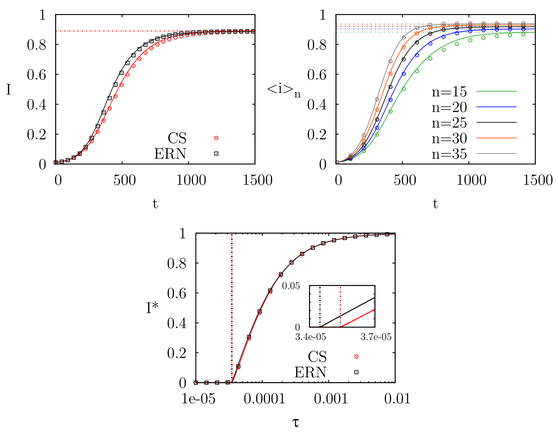
Our formalism can also describe propagation processes on random networks. In fact, to highlight the effect of a given topology on a given process, we will use an equivalent random network.

Equivalent random network (ERN): network with the same degree distribution as the original, but where all links are connected randomly. In order to use the same formalism for an ERN, one sets:

- $p_n = \delta_{2n}$ and $\epsilon = 1$ (cliques thus become simple links); and
- $\{g_m\}$ is chosen equal to the original degree distribution.

Results

Community Structure (CS) versus **ERN**



TOP: ODE integration (continuous lines) and analytical stable state (dotted line) versus numerical results (dots). BOTTOM: Analytical stables states (continuous lines) and epidemic threshold (dotted line) versus numerical results (dots).

About the simulations:

SIS model of disease spread with infection rate $\tau = 0.0005$, recovery rate $r = 0.001$ and initial conditions $I(0) = 1\%$. Simulated on 20 000 networks of 25 000 nodes. Each node has probability $g_m \propto m^{-1}\,\mathrm{e}^{-m/1.2}$ to participate in m cliques whose populations follow a binomial distribution of mean 20. Each possible link within a clique exists with probability $\epsilon = 0.8$.

Substantiation and discussions

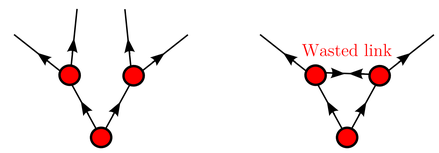
This model highlights **the community effect**. We predict that, versus an equivalent random network, a social topology will feature:

Higher epidemic threshold

- the epidemic threshold is systematically higher in a CS network than in an ERN;
- both epidemic thresholds converge towards the same value if the mean number of cliques per individual goes to infinity.

Longer relaxation times

- CS networks will take a longer time to reach equilibrium than ERNs;
- a quantification of this effect can be approximated with percolation arguments.



The **wasted links** pictured above are an inefficient way of spreading the disease and are the cause of the discussed effects.

Possibility of intervention

- Taking social topology into account allows simulation of realistic intervention scenarios during epidemics;
- e.g. school closings and vaccination of public health workers both correspond to interventions on given cliques of individual.

Future directions

- Dynamical networks that evolve in time.
- Co-evolution of networks and dynamics (retroaction of process dynamics on network topology).
- Interaction of two propagative agents (e.g. disease and information).
- Game theory on complex social networks
- Networks of networks.

NSERC CRSNG · CIHR IRSC Canadian Institutes of Health Research / Instituts de recherche en santé du Canada · Fonds de recherche sur la nature et les technologies Québec

L. Hébert-Dufresne, P.-A. Noël, V. Marceau, A. Allard and L.J. Dubé, *to be published* (2010)

M.E.J. Newman, "Properties of highly clustered networks," Phys. Rev. E **68**, 026121, 2003