Another important class of processes: epidemic spreading
Epidemiology

Two levels:

Microscopic: researchers try to disassemble and kill new viruses => quest for vaccines and medicines

Macroscopic: statistical analysis and modeling of epidemiological data in order to find information and policies aimed at lowering epidemic outbreaks => macroscopic prophylaxis, vaccination campaigns...
Standard epidemic modeling

**Compartments:** S, I, R...

Neglecting differences in:

- age
- gender
- health
- social class/status
- ...
Standard epidemic modeling

**Compartments:** S, I, R...

- S (susceptible)
- I (infected)
- R (removed)

Similar to chemical reactions:

\[
\text{S} + \beta \rightarrow \text{I}
\]

\[
\text{I} + \mu \rightarrow \text{R}
\]

\[
\text{R} + \beta \rightarrow \text{I}
\]

\[
\text{I} + \mu \rightarrow \text{S}
\]

Time progression:

- \(t=1\)
- \(t=2\)
- \(t=4\)
- \(t=8\)
Standard epidemic modeling

SI

\[ S + I \xrightarrow{\beta} I + I \]

SIS

\[ S + I \xrightarrow{\beta} I \xrightarrow{\mu} S \]

SIR

\[ S + I \xrightarrow{\beta} I \xrightarrow{\mu} R \]
Stages of an epidemic outbreak: population level

Infected individuals => prevalence/incidence
Transmission

S (susceptible)  I (infected)

\[ \beta \]

HOMOGENEOUS MIXING ASSUMPTION

Individual in state S, with \( k \) contacts, among which \( n \) infectious: in the homogeneous mixing approximation, the probability to get the infection in each time interval \( dt \) is:

\[
\text{Proba}(S \rightarrow I) = 1 - \text{Proba(not to get infected by any infectious)} \\
= 1 - (1 - \beta dt)^n \\
\approx \beta n dt \quad (\beta dt << 1) \\
\approx \beta k i dt \quad \text{as} \quad n \sim k i \text{ for homogeneous mixing}
\]

Hypothesis of mean-field nature:
every individual sees the same density of infectious among his/her contacts, equal to the average density in the population
The SI model

N individuals

$I(t) =$ number of infectious, $S(t) = N - I(t)$ number of susceptible

$i(t) = I(t)/N$, $s(t) = S(t)/N = 1 - i(t)$

If $k = <k>$ is the same for all individuals (homogeneous network):

\[
\frac{dI}{dt} = S(t) \times \text{Prob}(S \rightarrow I)
\]

\[
= \beta k S(t) i(t)
\]

\[
\frac{di}{dt} = \beta k i(t) (1 - i(t))
\]
The SI model

\[ S \text{ (susceptible)} \rightarrow \beta \rightarrow I \text{ (infected)} \]

\( N \) individuals

\[ I(t) = \text{number of infectious}, \quad S(t) = N - I(t) \quad \text{number of susceptible} \]

\[ i(t) = \frac{I(t)}{N}, \quad s(t) = \frac{S(t)}{N} \]

\[
\frac{di}{dt} = \beta \langle k \rangle i(1 - i)
\]

\[
i(t) = \frac{i_0 \exp(t/\tau)}{1 + i_0(\exp(t/\tau) - 1)} \quad \tau = \frac{1}{(\beta \langle k \rangle)}
\]
The SIS model

$N$ individuals

$I(t)$ = number of infectious, $S(t) = N - I(t)$ number of susceptible

$i(t) = I(t)/N$, $s(t) = S(t)/N$

Homogeneous mixing

$$\frac{di}{dt} = \beta \langle k \rangle i (1 - i) - \mu i$$

Competition of two time scales: $1/\mu$ and $1/(\beta \langle k \rangle)$
The SIR model

$N$ individuals
$I(t) =$ number of infectious, $S(t)$ number of susceptible, $R(t)$ recovered
$i(t) = I(t)/N$, $s(t) = S(t)/N$, $r(t) = R(t)/N = 1 - i(t) - s(t)$

Homogeneous mixing:

\[
\frac{ds}{dt} = -\beta \langle k \rangle i(t) s(t)
\]

\[
\frac{di}{dt} = \beta \langle k \rangle i(t) s(t) - \mu i(t)
\]

\[
\frac{dr}{dt} = \mu i(t)
\]

Competition of two time scales: $1/\mu$ and $1/(\beta \langle k \rangle)$
SIS and SIR models: linear approximation

Short times, $i(t) << 1$ (and $r(t) << l$ for the SIR)

$$\frac{di}{dt} \approx (\beta \langle k \rangle - \mu) i(t)$$

Exponential evolution $\exp(t/\tau)$, with

$$\frac{1}{\tau} = \beta \langle k \rangle - \mu$$

If $\beta \langle k \rangle > \mu$ : exponential growth
If $\beta \langle k \rangle < \mu$ : extinction

Epidemic threshold condition: $\beta \langle k \rangle = \mu$
Long time limit, SIS model

Stationary state: \(\frac{di}{dt} = 0\)

\[\mu i_\infty = \beta \langle k \rangle i_\infty (1 - i_\infty)\]

\[\beta \langle k \rangle < \mu \Rightarrow i_\infty = 0\]

\[\beta \langle k \rangle > \mu \Rightarrow i_\infty = 1 - \frac{\mu}{(\beta \langle k \rangle)}\]

Epidemic threshold condition: \(\beta \langle k \rangle = \mu\)

Phase diagram:
Immunization

Fraction $g$ of immunized (vaccinated) individuals:
reduce population of susceptible individuals

\[
S \rightarrow S \times (1 - g)
\]

\[
\frac{di}{dt} = \beta ki(t)(1 - i(t))(1 - g)
\]
Immunization

Fraction $g$ of immunized (vaccinated) individuals:
reduce population of susceptible individuals

Equivalent to a reduction of $\beta$:

$$
\beta \rightarrow (1 - g)\beta \\
\lambda \rightarrow (1 - g)\lambda
$$

$=> \text{critical immunization threshold}$

$$
g_c = 1 - \frac{\mu}{(\beta \langle k \rangle)}
$$

Fraction of population to vaccinate

to prevent an outbreak

$\lambda_c = \langle k \rangle^{-1}$
Homogeneous mixing: summary

\[ \lambda_c = \langle k \rangle^{-1} \]

\[ \lambda = \beta / \mu \]

Competition of time scales

=> Epidemic threshold condition: \( \beta \langle k \rangle = \mu \)
Homogeneous mixing: summary

immunization threshold bringing the system under the epidemic threshold by depleting the susceptible population

\[ g_c = 1 - \frac{\mu}{\beta \langle k \rangle} \]
Going beyond: additional compartments

- S: susceptible
- E: exposed
- I: infectious
- R: recovered

- S: susceptible
- I: infectious
- R: recovered
- S: susceptible
Going beyond: additional compartments

Diagram showing the flow of individuals through different compartments: S (susceptible), E (exposed), I (infectious), La (asymptomatic infectious), and R (recovered). Additional compartments include It (treated infectious).
Going beyond: population structure

Different classes of individuals: age, gender, etc…

=> potentially different

• transmissibility
• contact rates
Going beyond: population structure

Different classes of individuals: age, gender, etc...
=> potentially different
  • transmissibility
  • contact rates

Contact matrices

Ex: flu => different contact rates for children and adults
Going beyond: population structure

Different classes of individuals: age, gender, etc…

=> potentially different

- transmissibility
- contact rates

Ex: HIV => different transmissibility depending on gender
Wide spectrum of complications and complex features to include...

- Ability to explain trends at a population level
- Model realism loses in transparency. Validation is harder.
Complex networks
Complex networks

Diseases propagate on networks:

Social (contact) networks

Technological networks:

- Internet, Web, P2P, e-mail...

...which are complex, heterogeneous networks

Usual mean-field: neglects the degree heterogeneity

Extension of mean-field theory to take it into account
Degree-based mean-field theory

Number of contacts (degree) can vary a lot huge fluctuations ($<k^2> \gg <k>$)

Heterogeneous (degree-based) mean-field: density of Susceptible in the class of degree $k$, $s_k = S_k/N_k$
Infectious in the class of degree $k$, $i_k = I_k/N_k$
(Recovered in the class of degree $k$, $r_k = R_k/N_k$ )

$s(t) = \sum P(k) s_k$, $i(t) = \sum P(k) i_k$, $r(t) = \sum P(k) r_k$
Degree-based representation

MF-like assumption: all individuals in a given class are “equivalent”
The SIS model in the degree-based MF theory

\[
\frac{dI_k}{dt} = S_k(t) \times \text{Proba}(S_k \rightarrow I_k) - \mu I_k(t)
\]

interaction with nodes of any degree \(k'\)

\(S_k\) (susceptible)

interaction with nodes of any degree \(k'\)

\(I_k\) (infected)

\(S_k\) (susceptible)

interaction with nodes of any degree \(k'\)

degree \(k\)

degree \(k\)

degree \(k\)

degree \(k\)
The SIS model in the degree-based MF theory

\[ \text{Proba}(S_k \rightarrow I_k) \]

Number \( k \) of possible contacts

Proba of a contact with a node of degree \( k' \)

Proba that the node is infectious:

\[ i_{k'} \]

\[ P(k' | k) = \text{the probability that a link originated in a node with connectivity } k \text{ points to a node with connectivity } k' \]
The SIS model in the degree-based MF theory

\[ \frac{di_k}{dt} = \beta k (1 - i_k) \Theta_k - \mu i_k \]

\[ \Theta_k = \text{Proba that any given link points to an infected node} \]

\[ \Theta_k = \sum_{k'} P(k'|k) i_{k'} \]

\[ P(k'|k) = \text{the probability that a link originated in a node with connectivity } k \text{ points to a node with connectivity } k' \]
In uncorrelated networks:

\[ \Theta_k = \Theta = \sum_{k'} \frac{k'}{\langle k \rangle} P(k') i_{k'} \]

Short times, \( i_k(t) \ll 1 \)

\[ \frac{d\Theta}{dt} = \left( \beta \frac{\langle k^2 \rangle}{\langle k \rangle} - \mu \right) \Theta \]

Epidemic threshold condition

\[ \frac{\beta}{\mu} = \frac{\langle k \rangle}{\langle k^2 \rangle} \]
The SIS model in the degree-based MF theory

Epidemic threshold in uncorrelated networks

\[
\frac{\beta}{\mu} = \frac{\langle k \rangle}{\langle k^2 \rangle}
\]

Heterogeneous, infinite network:

\[
\langle k^2 \rangle \to \infty
\]

Condition always satisfied

Finite prevalence for any spreading parameters
Epidemic phase diagram in heterogeneous networks

- Wide range of spreading rate with low prevalence
- Lack of healthy phase = standard immunization cannot drive the system below threshold!!!
Finite size effects

Finite number of nodes $N$

$\Rightarrow$ *Finite cut-off for $P(k)$*

$\Rightarrow$ *Finite* $\kappa = \langle k^2 \rangle / \langle k \rangle$

$\Rightarrow$ *Finite epidemic threshold*

Ratio of epidemic threshold to the value obtained in a homogeneous network:
Spreading dynamics

Short times:
\[
\frac{d\Theta}{dt} = \left( \beta \frac{\langle k^2 \rangle}{\langle k \rangle} - \mu \right) \Theta
\]

=> Exponential growth: \( \Theta = \Theta_0 \exp(t/\tau) \)

\[
\frac{di_k}{dt} \approx \beta k \Theta - \mu i_k
\]

\( i_k \propto k \exp(t/\tau) \)
Consequences on immunization strategies

Uniform immunization:
Fraction $g$ of randomly chosen immunized (vaccined) individuals:

$$\beta \rightarrow \beta (1-g)$$

$\Rightarrow$ inefficient: need

$$(1 - g) \frac{\beta}{\mu} < \frac{\langle k \rangle}{\langle k^2 \rangle}$$

$$g > g_c = 1 - \frac{\mu \langle k \rangle}{\beta \langle k^2 \rangle}$$

tends to 1
Proportional immunization

\( g_k \) fraction of immunized individuals of degree \( k \), such that:

\[
\beta_k (1 - g_k) = \beta' = \text{cst}
\]

\[
\frac{d j_k}{dt} = \beta' (1 - j_k) \Theta_k - \mu j_k
\]

Short times (uncorr. nets):

\[
\frac{d \Theta}{dt} = (\beta' - \mu) \Theta
\]

Epidemic threshold recovered!

Efficient immunization: need \( \beta' < \mu \) i.e.,

\[
g_k > 1 - \frac{\mu}{\beta k}
\]

Targeted immunization

=> immunize fraction $g$ of individuals with largest connectivity

$$\frac{\langle k \rangle_g}{\langle k^2 \rangle_g} > \frac{\beta}{\mu}$$

similar to targeted attacks!!!

immunizing $\leftrightarrow$ removing nodes and links

Ex of explicit calculation for BA network:

$$g_c \propto \exp(-2\mu/m\beta)$$

Immunization

NB: when network’s topology unknown: acquaintance immunization

What does HMF neglect

1. Structural correlations in the network

(HMF equivalent to an annealed network approximation)

=> Quenched Mean-Field, which takes into account the network structure
=> Epi threshold=1/(largest vp of adjacency matrix)

2. Dynamical correlations

(emerging during the spreading process)
Some more complications

Degree correlations

Clustering

Directed networks

Weights

Community structures

Initial (local) faster spread, slowing down at global scale

Strength of weak ties (Granovetter 1973, Onnela et al. 2007)

Immunization of bridges
Wide spectrum of complications and complex features to include…

Ability to explain trends at a population level

Model realism loses in transparency.
Validation is harder.
Meta-population models

Intra-population infection dynamics by stochastic compartmental modeling
Inside each population: homogeneous mixing

Baroyan et al. (1969)
Ravchev, Longini (1985)
Modeling of global epidemics propagation

multi-level description:

- intra-city epidemics
- inter-city travel

Baroyan et al. (1969)
Ravchev, Longini (1985)
Why is a large-scale approach needed?

14th century - Black death
Why is a large-scale approach needed?
Why is a large-scale approach needed?

2009 - H1N1 pandemic

Recent advances

• availability of data at various scales
  – census
  – transportation systems
  – behaviour

• ability to analyze/model/generalize data

• ability to integrate these data into data-driven models

Development of models
  • data-driven
  • large-scale

Evaluation of scénarii
Prediction of the future evolution of a spread
Testing prevention or mitigation measures
A recent large-scale platform

http://www.gleamviz.org

simulation platform for the worldwide propagation of diseases, used in real time during the H1N1 pandemic and to give forecast w.r.t. the Ebola crisis

D. Balcan, V. Colizza, B. Gonçalves, H. Hu, J.J. Ramasco, A. Vespignani

GLEaM in brief

Population distribution:
detailed population data from
1/4x1/4 degree tassellation.

Local mobility:
census data from about 30 countries
in the 5 continents extended to all
countries.

Long range travel:
3362 cities in 220 countries.
More than 16000 connections
with travel flows.

Epidemic compartmental model
Metapopulation model with homogeneous mixing
assumption.
H1N1 pandemics: Prediction months in advance of the epidemic peak timing

see www.gleamviz.org
Assessing the international spreading risk associated with the 2014 West African Ebola outbreak

The 2014 West African Ebola Outbreak is so far the largest and deadliest recorded in history. The affected countries, Sierra Leone, Guinea, Liberia, Nigeria, and recently Senegal have been struggling to contain and to mitigate the outbreak. We have developed in the past week a modeling approach aimed at assessing the progression of the epidemic in West Africa and its international spread under the assumption that the EVD outbreak continues to evolve at the current pace.

Our results have been published in PLOS Currents Outbreaks. However, our modeling work has been motivated by the need for a rapid assessment of the EVD outbreak trends and the obtained results may change as more information becomes available from the EVD affected region and more refined sensitivity analysis can be implemented computationally. For this reason, the paper on PLOS Current Outbreaks shall be considered as a live paper that is constantly updated with new data, projections and analysis.

The collaboration

NORTHEASTERN UNIVERSITY
M.F.C. Gomes, A.Pastore y Piontti, A. Vespignani

ISI FOUNDATION
L. Rossi

FRED HUTCHINSON CANCER RESEARCH CENTER
D. Chao, M. E. Halloran

UNIVERSITY OF FLORIDA
I.M. Longini Jr.

Published paper

http://www.mobs-lab.org/ebola.html
Travel limitations?

---

Analytical approach

=> Degree-based mean-field

Diffusion (random walk) between nodes

Reaction (SIS, SIR) inside each node
Degree-based mean-field approach: Diffusion

N nodes, W walkers

Node i => $W_i$ walkers

$W = \sum_i W_i$

Degree block variables

$$W_k = \frac{1}{N_k} \sum_{i|k_i=k} W_i$$

$N_k = NP(k) =$ number of nodes of degree $k$

Evolution equation:

$$\partial_t W_k(t) = -r_k W_k(t) + k \sum_{k'} d_{k'k} P(k'|k) W_{k'}(t)$$

Walkers going out of nodes

$r_k = k \sum_{k'} d_{k'k} P(k'|k)$

Walkers going into nodes

Diffusion rate along edges $k'-k$
Degree-based mean-field approach: Diffusion

Simplest case: uniform diffusion \( r_k = r; d_{k',k} = r/k' \)

Uncorrelated random networks:

\[
\partial_t W_k(t) = -rW_k(t) + \frac{k}{\langle k \rangle} \sum_{k'} P(k') r W_{k'}(t)
\]

Stationarity \( \Rightarrow \)

\[
W_k(t) = \frac{k}{\langle k \rangle} \frac{W}{N}
\]
Degree-based mean-field approach: Diffusion

Example of other diffusion rates: \[ d_{kk'} = w_0(kk')^\theta / T_k, \quad r_k = r \]

Uncorrelated random networks:

\[
\partial_t W_k(t) = -r W_k(t) + r k^{1+\theta} \frac{w_0}{A\langle k \rangle} \sum_{k'} P(k') W_{k'}(t)
\]

Stationarity: \[ W_k(t) = \frac{k^{1+\theta}}{\langle k^{1+\theta} \rangle} \frac{W}{N} \]

Degree-based mean-field approach: Diffusion

Diffusion rate keeping constant populations: important in the perspective of modelling travel behaviours

Number of travellers between 2 subpopulations per unit time=fixed

Proba per unit time to go from $i$ to $j$:\[ \frac{w_{ij}}{W_i} \]

\[ \partial_t W_i = \sum_j W_j \frac{w_{ij}}{W_j} - W_i \sum_j \frac{w_{ij}}{W_i} = 0 \]

Any population distribution is stationary
Degree-based mean-field approach:

Diffusion

Diffusion rate keeping constant populations: important in the perspective of modelling travel behaviours

In the degree-based framework

\[ d_{kk'} = \frac{w_{kk'}}{W_k} \quad (w_{kk'} = w_{k'k}) \]

\[ r_k = k \sum_{k'} d_{kk'} P(k'|k) \]

\[ \partial_t W_k(t) = -r_k W_k(t) + k \sum_{k'} d_{kk'} P(k'|k) W_{k'}(t) \]

\[ = -k \sum_{k'} \frac{w_{kk'}}{W_k} P(k'|k) W_k + k \sum_{k'} w_{k'k} P(k'|k) \]

\[ = 0 \]

Any population distribution is stationary

Degree-based mean-field approach: SIS

In each node i: S_i susceptible, I_i infectious, \( W_i = S_i + I_i \)

Degree block variables

\[
S_k = \frac{1}{N_k} \sum_{i \mid k_i = k} S_i \\
I_k = \frac{1}{N_k} \sum_{i \mid k_i = k} I_i
\]

Each time step: 2 processes
1- reaction
2- diffusion

Degree-based mean-field approach: SIS

Each time step: 2 processes

1- reaction

$$I_k \rightarrow I_k - \mu I_k + \beta \Gamma_k$$

2- diffusion

$$I_k \rightarrow (I_k - \mu I_k + \beta \Gamma_k)(1 - r_k) + \sum_{k'} k P(k'|k) d_{k'k} ((1 - \mu) I_{k'} + \beta \Gamma_{k'}$$

Uniform diffusion or diffusion with constant populations

=> epidemic threshold

$$\beta / \mu = 1$$

Degree-based mean-field approach: SIR case

\[ \beta/\mu > 1 \]

- Zero diffusion: epidemics confined in first subpopulation
- Infinite diffusion: population well-mixed

expect a transition between
- confined epidemics at low diffusion rates
- global invasion at large diffusion rates

NB: for SIS, as soon as non-zero diffusion, global invasion as there is a stationary state

Degree-based mean-field approach: SIR case

Problem:

Can be very small: travel of fractions of individuals

\[
\frac{\partial t}{I_k} = -r_k I_k + (-\mu I_k + \beta \Gamma_k)(1 - r_k) \\
+ k \sum_{k'} P(k' | k) d_{k' k} ((1 - \mu) I_{k'} + \beta \Gamma_{k'})
\]

Continuous approximation cannot capture the global invasion threshold

Need to take into account discreteness & stochasticity

Invasion: branching process

$D_k^0, D_k^1,...$  # of *diseased* nodes (i.e., with at least one infected individual) of degree $k$, at generation $n=0, 1, ...$

Global invasion threshold

\[ R_* = (R_0 - 1) \frac{\langle k^{2+2\theta} \rangle - \langle k^{1+2\theta} \rangle}{\langle k \rangle} \frac{w_0 \alpha}{\mu} \]

Ex: SIR, \( \alpha \sim 2(R_0 - 1)/R_0^2 \)

\[ w_{0c} = \frac{\mu R_0^2}{2(R_0 - 1)^2} \frac{\langle k \rangle}{\langle k^{2+2\theta} \rangle - \langle k^{1+2\theta} \rangle} \]

phase transition in mobility

Real-world network: \( w_0 \) 10 times larger than \( w_{0c} \) !!!

Explains empirical results!!

Going beyond

Population structure (age/gender) and travel behaviours

(Apolloni et al., BMC ID 2013)

Length of stay at destination

(Poletto et al., J. Th. Biol. 2013)

Change of behaviour

Epidemics in multiplex networks

Interdependent networks (power-grid - communication/computer network)
Layers of social networks
Different transportation networks

Effect of coupling on cascading failures (percolation processes)
Epidemics on multiplex networks
Cooperation in multiplex social networks
Modification of infectiousness of disease (a) if spreader or susceptible are infected with disease (b)

Modification of recovery rate for disease (a) if infectious is also infected with disease (b)

mutual enhancement or partial cross-immunity

Sanz et al., arXiv:1402.4523
Epidemics in time-varying networks

Networks= (often) dynamical entities

(communication, social networks, online networks, transport networks, etc…)

• Which dynamics?
• Characterization?
• Modeling?
• Consequences on dynamical phenomena?
  (e.g. epidemics, information propagation…)

Time-varying networks: often represented by aggregated views
Example: contacts in a primary school, dynamic view

Example:
contacts in a primary school, aggregated view
Definition: temporal network

Temporal network: \( T=(V,S) \)

- \( V \) = set of nodes

\[
s_{ij} \in S: s_{ij} = \{(t_{ij}^{s,1}, t_{ij}^{e,1}) \cdots (t_{ij}^{s,\ell}, t_{ij}^{e,\ell})\}
\]

Other representation:
time-dependent adjacency matrix:
a(i,j,t)= 1 \iff i and j connected at time t
Reachability in temporal networks

Aggregation of temporal network

\[ w_{ij} = \int_{t_{min}}^{t_{max}} a(i, j, t) dt \]

NB: enough information if underlying process is Poissonian

Aggregation of temporal network

Temporal behavior most often non-Poissonian

=> aggregate view hides important temporal patterns

Burstiness

Generalization of definitions to temporal networks

Reachability issue
=> time respecting path ("journey")
  => set of influence of a node
  => temporal connectivity (similar to case of directed graphs)

Path length => concept of shortest paths
Time respecting path duration => concept of fastest journey

Temporal motifs

Centrality measures

(...)

Complex temporal characteristics

- burstiness
  - non-Poissonian inter-event distributions
  - power-law temporal correlations
- heterogeneity of event durations
  - single events
  - aggregated durations (weights in aggregated networks)
- stationarity of statistical features
- daily, weekly, and organizational rhythms
- weight-topology correlations
- topology-activity correlations (e.g., school)
...
Temporal networks

- Generalization of concepts?
- Centrality of a node?
- Temporal communities?
- Models for temporal networks?
- Impact of temporal features on dynamical processes?
Toy spreading processes on dynamical networks

- deterministic SI process

- fastest paths ≠ shortest paths

Fastest path: A -> B -> C
Shortest path: A - C
Example: shortest vs fastest paths in a temporal contact network

HT09: June, 30th
- Transmission network
- Aggregated network

SG: May, 19th
- Transmission network
- Aggregated network

Conference

Museum
Example: deterministic SI in temporal contact networks
>(Toy) spreading processes on dynamical networks

Use of null models to reveal the role of the temporal aspects
Mobile phone data:
• community structure (C)
• weight-topology correlations (W)
• burstiness on single links (B)
• daily patterns (D)
• event-event correlations between links (E)

Effects of the different ingredients?

Use series of *null models*!

Null models

<table>
<thead>
<tr>
<th>EVENT SEQUENCE</th>
<th>D</th>
<th>C</th>
<th>W</th>
<th>B</th>
<th>E</th>
</tr>
</thead>
<tbody>
<tr>
<td>Original</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
</tr>
<tr>
<td>Equal-weight link-sequence shuffled</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
</tr>
<tr>
<td>Link-sequence shuffled</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td></td>
</tr>
<tr>
<td>Time shuffled</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Configuration model</td>
<td>✓</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Mobile phone data

- community structure (C)
- weight-topology correlations (W)
- burstiness on single links (B)
- daily patterns (D)
- event-event correlations between links (E)

Bursty dynamics slows down spreading

More results

Rocha et al., PLOS Comp Biol (2011)
- data: temporal network of sexual contacts
- temporal correlations accelerate outbreaks

Pan & Saramaki, PRE (2011)
- data: mobile phone call network
- slower spread when correlations removed

Miritello et al., PRE (2011)
- data: mobile phone call network
- burstiness decreases transmissibility

Takaguchi et al., PLOS ONE (2013)
- data: contacts in a conference; email
- threshold-based spreading model
- burstiness accelerate spreading

Rocha & Blondel, PLOS Comp Biol (2013)
- model with tuneable distribution of inter-event times (no correlations)
- burstiness => initial speedup, long time slowing down
Still somewhat unclear picture

Results

• depend on data set

• depend on spreading model

• generally

  • burstiness slows down spreading
  • correlations (e.g., temporal motifs) favors spreading
  • role of turnover
  • +: effect of static patterns
SIS model on activity-driven network

Model: N nodes, each with an “activity” a, taken from a distribution F(a)

At each time step:

• node i active with probability a(i)
• each active node generate m links to other randomly chosen nodes
• iterate with no memory

Activity-based mean-field theory:

\[
I_{a}^{t+\Delta t} = -\mu \Delta t I_{a}^{t} + I_{a}^{t} + \lambda m (N_{a}^{t} - I_{a}^{t}) a \Delta t \int \frac{d a'}{N} + \lambda m (N_{a}^{t} - I_{a}^{t}) \int \frac{d a'}{N} \frac{I_{a}^{t} a' \Delta t}{N},
\]

SIS model on activity-driven network

Epidemic threshold:

\[ \lambda_c = \frac{1}{m \langle a \rangle + \sqrt{\langle a^2 \rangle}} \]

Immunization strategies

=> take into account temporal structure

Lee et al., PLOS ONE (2012)
=>inspired by “acquaintance protocol” in static networks)
• “Recent”: choose a node at random, immunize its most recent contact
• “Weight”: choose a node at random, immunize its most frequent contact in a previous time-window

Starnini et al., JTB (2012)
• aggregate network on [0,T]
• compare strategies
  • immunize nodes with highest k or BC in [0,T]
  • immunize random acquaintance (on [0,T])
  • recent, weight strategies
• vary T
• find saturation of efficiency as T increases

Liu et al., arXiv:1309:7031 (activity-driven network model=>analytics)
• target nodes with largest activity
• random neighbour (over an observation time T) of random node
Epidemic processes in complex networks

Romualdo Pastor-Satorras,1 Claudio Castellano,2,3 Piet Van Mieghem,4 and Alessandro Vespignani5
1Departament de Física i Enginyeria Nuclear, Universitat Politècnica de Catalunya, Campus Nord B4, E-08034 Barcelona, Spain
2Istituto dei Sistemi Complessi (ISC-CNR), via dei Taurini 19, 00185 Roma, Italy
3Dipartimento di Fisica, “Sapienza” Università di Roma, P.le A. Moro 2, 00185 Roma, Italy
4Delft University of Technology, Delft, The Netherlands
5Laboratory for the Modeling of Biological and Socio-technical Systems, Northeastern University, Boston, MA 02115, USA
6Institute for Scientific Interchange Foundation, Turin 10133, Italy