Burstiness and Spreading on Temporal Networks

R. Lambiotte
Department of Mathematics
University of Namur

Generalized Master Equations for Non-Poissonian Dynamics on Networks, Till Hoffmann, Mason Porter and R.L., Physical Review E 2012
Random Walks on Stochastic Temporal Networks, Till Hoffmann, Mason Porter and R.L., in Temporal Networks (Springer 2013)
Burstiness and spreading on temporal networks, R.L., L. Tabourier and J.C. Delvenne, EPJB 2013 (in press)

naxys
Namur Center for Complex Systems

Network science in a nutshell

A static network is built from empirical data or from a model

A model (SI, synchronization) is studied on the network

→ No memory!

↙ **Trajectories**:
The process evolves by selecting links irrespectively of the previous steps

↘ **Timings**:
The process evolves either at discrete times or following a Poisson process

→ Implicit Markov assumption

How realistic is this modelling assumption? If unrealistic, is it possible to bring memory into the modelling?

Networks with Memory, Martin Rosvall, Alcides V. Esquivel, Andrea Lancichinetti, Jevin D. West, Renaud Lambiotte, arXiv:1305.4807

Network science in a nutshell

A static network is built from empirical data or from a model

A model (SI, synchronization) is studied on the network

→ No memory!

Trajectories:
The process evolves by selecting links irrespectively of the previous steps

Timings:
The process evolves either at discrete times or following a Poisson process

→ Implicit Markov assumption

How realistic is this modelling assumption? If unrealistic, is it possible to bring memory into the modelling?

Dynamics on stochastic temporal networks

What is the effect of the temporality of the network on a spreading process?

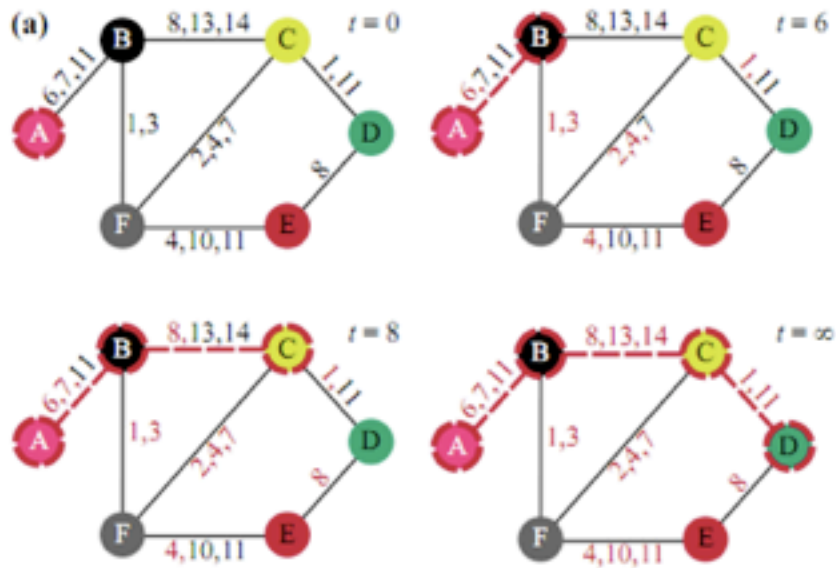
More and more empirical data incorporate information about the timing of activation of edges (e.g. **when** a phone call is made)

Non-trivial patterns of activation of nodes and edges
Burstiness: intermittent switching between periods of low activity and high activity, and a fat-tailed inter-event time distributions.

Temporal Networks, Petter Holme, Jari Saramäki, Phys. Rep. 519, 97-125 (2012)

Simulations on temporal graphs...

Simulations on temporal graphs



Temporal Networks, P. Holme and J. Saramäki, arXiv: 1108.1780

... and comparison with null models

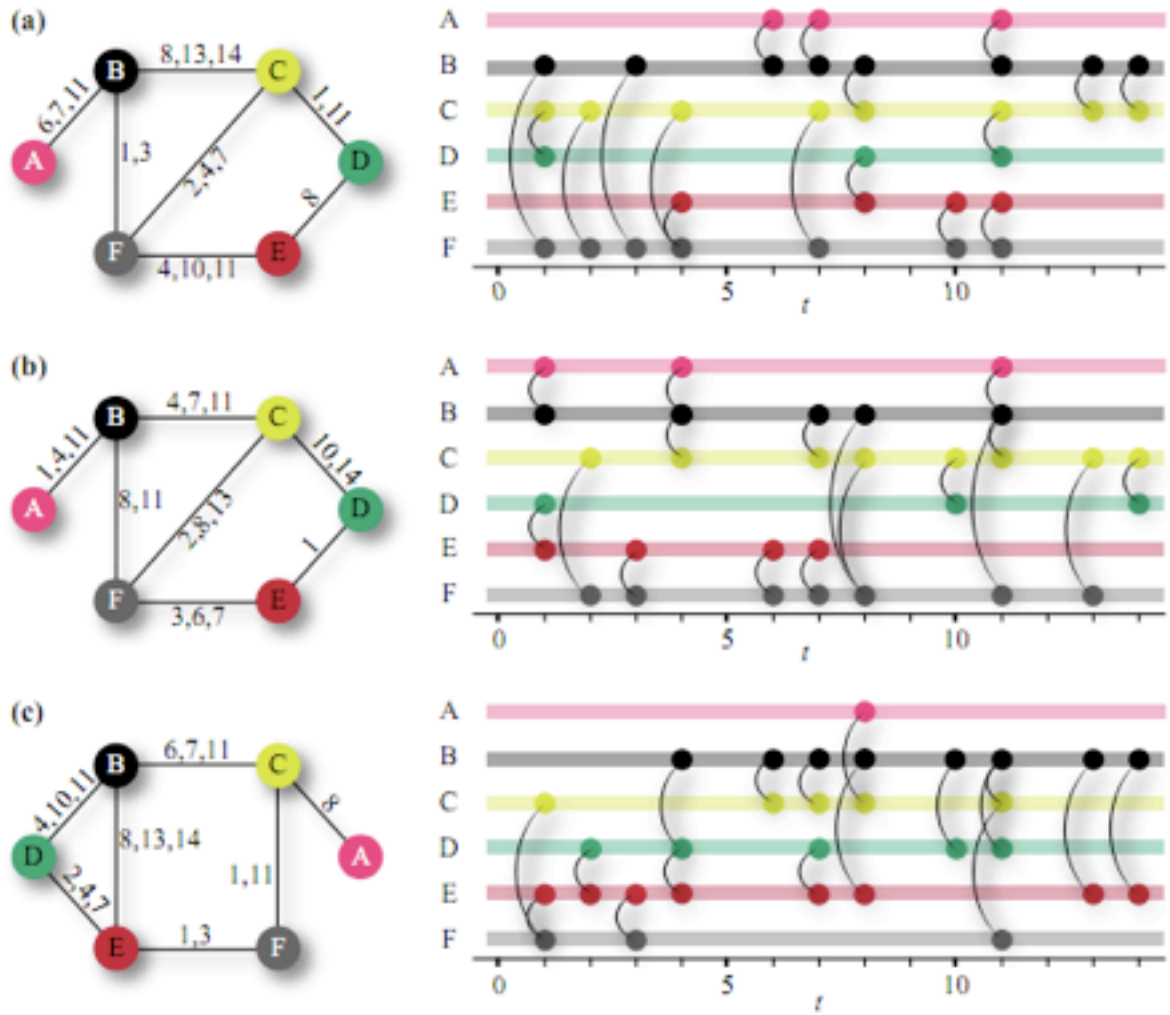


FIG. 11: Illustration of two types of randomization null-models for contact sequences. (a) shows a contact sequence (the same as in Fig. 1). In (b) it is randomized by the Randomly Permuted times procedure such that contacts happen the same number of time per edge, and the aggregated network topology is the same. In (c) the contact sequence in (a) is randomized by the Randomized edges (RE) procedure. With RE, the time sequence of the contacts along an edge is conserved, and so is the degree sequence of the original network, but all other structure of the topology is destroyed. (The latter statement is perhaps not so well illustrated by this figure as there are not so many graphs with the degree sequence of the original, aggregate graph.)

... and comparison with null models

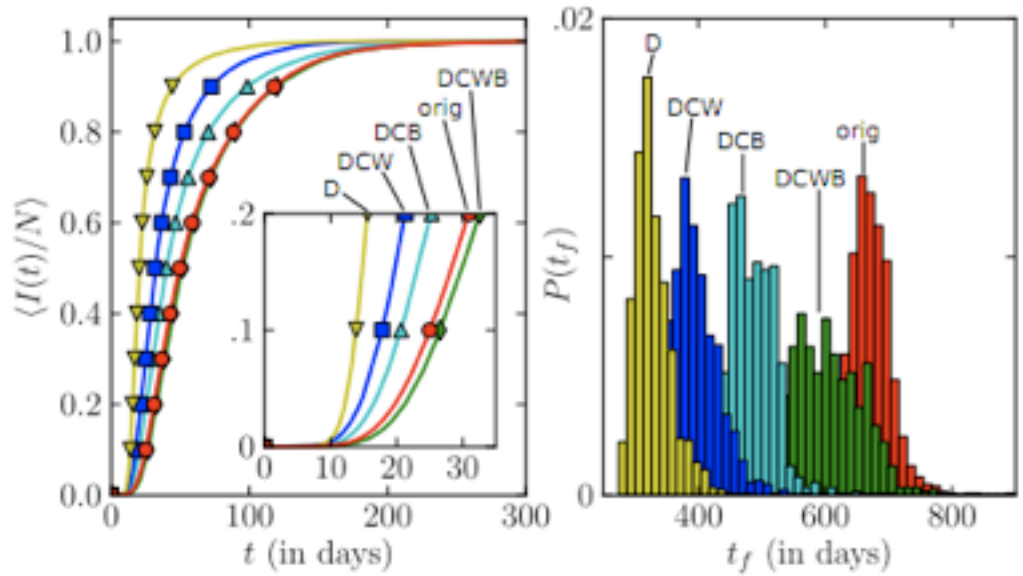


FIG. 1: (color online) (Left) Fraction of infected nodes $\langle I(t)/N \rangle$ as a function of time for the original event sequence ($\circ$) and null models: equal-weight link-sequence shuffled DCWB ($\Diamond$), link-sequence shuffled DCB ($\triangle$), time-shuffled DCW ($\square$) and configuration model D ($\triangledown$). Inset: $\langle I(t)/N \rangle$ for the early stages, illustrating differences in the times to reach $\langle I(t)/N \rangle = 20\%$. (Right) Distribution of full prevalence times $P(t_f)$ due to randomness in initial conditions.

... but lack of theoretical understanding

- slowing down compared to random times:

  see e.g. *Small but slow world: how network topology and burstiness slow down spreading*, M. Karsai et al, Phys Rev E 83, 025102(R) (2011); *Dynamical strength of social ties in information spreading*, Miritello et al, Phys. Rev. E 83, 045102(R) (2011)

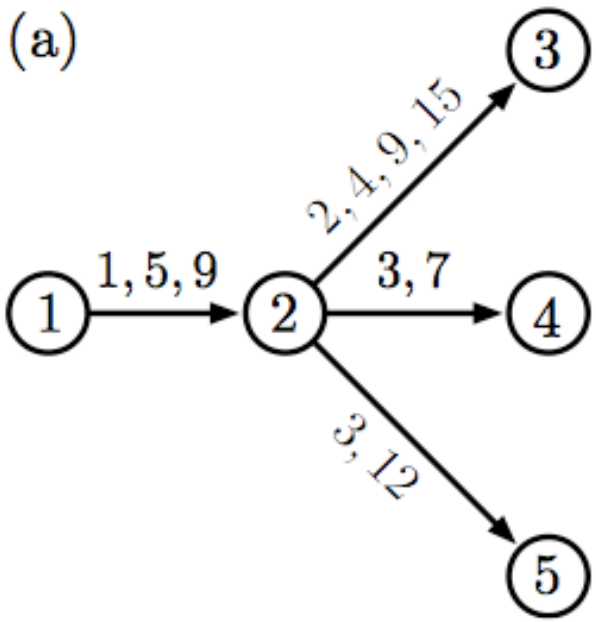
- faster than random reference: see

  Simulated Epidemics in an Empirical Spatiotemporal Network of 50,185 Sexual Contacts, L.E.C. Rocha et al, PLoS Comput. Biol. 7, e1001109 (2011)

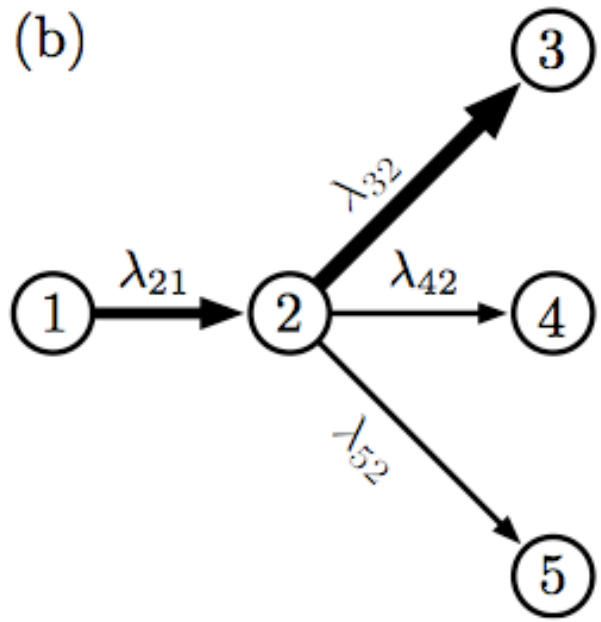
Dynamics on stochastic temporal networks

What is the effect of the temporality of the network on a spreading process?

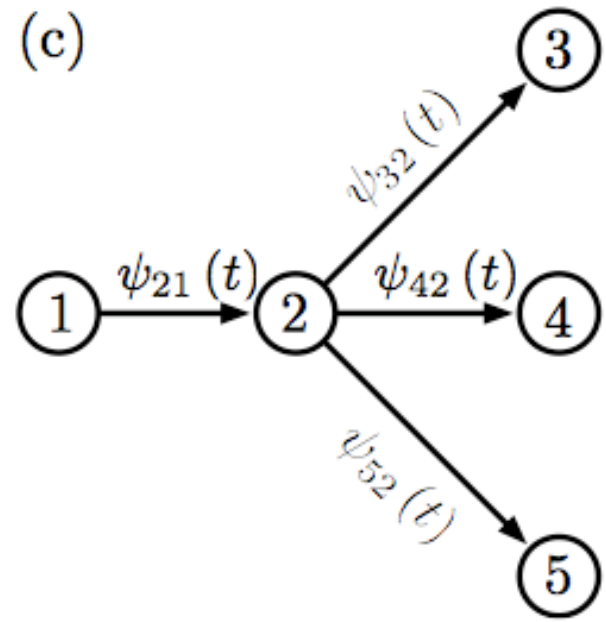
More and more empirical data incorporate information about the timing of activation of edges (e.g. **when** a phone call is made)



Temporal network, usually studied by means of computer simulations



Static network representation where the timing of events are Poisson processes



Stochastic temporal network where the sequence of activation times is a stochastic model that preserves the observed inter-event distribution

Temporal Networks, Petter Holme, Jari Saramäki, Phys. Rep. 519, 97-125 (2012)

From data to models

Let us consider a system of N nodes observed during a time interval T

We focus on the activation of edges going from i to j.

$$t_{ij} = \{t_{ij}^{(1)}, t_{ij}^{(2)}, ..., t_{ij}^{(n_{ij})}\}$$

The exact sequence of activation times is by a random sequence where events take place according to an inter-activation time fitted on the data

$f_{ij}(\tau)d\tau$ → probability to observe a time interval of duration in $[\tau, \tau + d\tau]$ between two activations of the edge

$\int_0^\infty \tau f_{ij}(\tau)d\tau = \langle\tau\rangle_{ij}$ → expected time between two activations of an edge

From data to models

When modeling the diffusion of an entity on the network, the distribution $f_{ij}(\tau)$ only plays an indirect role. The important quantity is instead the waiting time distribution $\psi_{ij}(t)$ that the entity arriving on *i* has to wait for a duration *t* before an edge towards *j* is available.

In epidemic spreading, it is the time it takes for a newly infected node to spread the infection further via the corresponding link.

Assuming that the activations of neighbouring edges are independent

$$\psi_{ij}(t) = \frac{1}{\langle \tau \rangle_{ij}} \int_t^\infty f_{ij}(\tau) d\tau$$

If the activations of neighbouring edges are dependent, $\psi_{ij}(t)$ can be directly measured in empirical data

A.O. Allen. Probability, Statistics, Queueing Theory: With Computer Science Applications, 1990.

From data to models

$$\langle t \rangle_{ij} = \int_0^\infty t \psi_{ij}(t) dt = \frac{1}{2} \frac{\langle \tau^2 \rangle_{ij}}{\langle \tau \rangle_{ij}}$$

At a fixed value of the average inter-activation time, the waiting time can be arbitrarily large if the variance of inter-activation times is sufficiently large. This paradox, often called waiting time paradox or bus paradox in queuing theory, is an example of length-biased sampling.

Waiting-times and inter-activation times have the same distribution when the process is Poissonian, in which case

$$\psi_{ij}(t) = f_{ij}(t) = \frac{1}{\langle t \rangle_{ij}} \exp\left(-\frac{t}{\langle t \rangle_{ij}} \right)$$

Their tail has the same nature in the case of power-law tails

$$\psi_{ij}(t) \sim t^{-\alpha} \Leftrightarrow f_{ij}(\tau) \sim \tau^{-(\alpha+1)}$$

A.O. Allen. Probability, Statistics, Queueing Theory: With Computer Science Applications, 1990.

Effect on spreading

In the literature, authors focus on a limited number of families of distributions (gamma, power-law, stretched exponential, log-normal), and on the effect of the tail of the distribution.

Non-Markovian Infection Spread Dramatically Alters the Susceptible-Infected-Susceptible Epidemic Threshold in Networks

P. Van Mieghem* and R. van de Bovenkamp
Faculty of Electrical Engineering, Mathematics and Computer Science, Delft University of Technology, P. O. Box 5031, 2600 GA Delft, The Netherlands
(Received 28 September 2012; published 5 March 2013)

Most studies on susceptible-infected-susceptible epidemics in networks implicitly assume Markovian behavior: the time to infect a direct neighbor is exponentially distributed. Much effort so far has been devoted to characterize and precisely compute the epidemic threshold in susceptible-infected-susceptible Markovian epidemics on networks. Here, we report the rather dramatic effect of a nonexponential infection time (while still assuming an exponential curing time) on the epidemic threshold by considering Weibullean infection times with the same mean, but different power exponent α. For three basic classes of graphs, the Erdős-Rényi random graph, scale-free graphs and lattices, the average steady-state fraction of infected nodes is simulated from which the epidemic threshold is deduced. For all graph classes, the epidemic threshold significantly increases with the power exponents α. Hence, real epidemics that violate the exponential or Markovian assumption can behave seriously differently than anticipated based on Markov theory.

DOI: 10.1103/PhysRevLett.110.108701 PACS numbers: 89.75.Hc

"... For all graph classes, the epidemic threshold significantly increases with the power exponents alpha." (parameter of the Weibull)

Effect on spreading

In the literature, authors focus on a limited number of families of distributions (gamma, power-law, stretched exponential, log-normal), and on the effect of the tail of the distribution.

Absence of epidemic outbreaks with heavy-tailed contact dynamics

Byungjoon Min,[1] K.-I. Goh,[1,*] and I.-M. Kim[1]

[1] *Department of Physics, Korea University, Seoul 136-713, Korea*

(Dated: March 20, 2013)

We study the epidemic spreading process following contact dynamics with heavy-tailed waiting time distributions. We show both analytically and numerically that the temporal heterogeneity of contact dynamics can significantly suppress the disease's transmissibility, hence the size of epidemic outbreak, obstructing the spreading process. Furthermore, when the temporal heterogeneity is strong enough, one obtains the vanishing transmissibility, hence the lack of epidemic outbreaks for any finite recovery time, the condition of which was derived.

"... we have shown both analytically and numerically that epidemic outbreaks of the SIR model can be strongly suppressed, and even completely blocked, by the heavy-tailed contact dynamics"

Effect on spreading

In the literature, authors focus on a limited number of families of distributions (gamma, power-law, stretched exponential, log-normal), and on the effect of the tail of the distribution.

Some distribution with some parameters propagates faster than some other distribution with other distributions.

Which properties of the waiting time tend to affect (accelerate or slow down) spreading processes:
The average waiting time?
Its variance?
The tail of the distribution?

None of those. What actually matters is the **time ordering of events**, how the probability mass of different probabilities are distributed.

Effect on spreading: Random Walks

A walker located at a node i remains on it until an edge leaving *i* toward some node j appears. When such an event occurs, the walker jumps to *j* without delay and then waits until an edge leaving *j* appears.

The probability for the walker to jump to *j* depends on $\psi_{ij}(t)$, but also on all $\psi_{ik}(t)$, where *k* are neighbours of *i*, because the walker takes the first edge available for transport. Once a walker has left a node, edges leaving this node become useless for transport. For this reason, the probability to actually make a step from *i* to *j* at time *t* is given by

$$T_{ij}(t) = \psi_{ij}(t) \times \prod_{k \neq j} \int_t^\infty \psi_{ik}(t')\, dt'$$

When two neighbours:

$$T_{ij}(t) = \psi_{ij}(t) \int_t^\infty \psi_{ik}(t')\, dt'$$

The probability for making a jump to node j is given by the effective transition matrix

$$\mathbb{T}_{ij} \equiv \int_0^\infty T_{ij}(t) dt \qquad \sum_j \mathbb{T}_{ij} = 1$$

Generalized Master Equations for Non-Poissonian Dynamics on Networks, Till Hoffmann, Mason Porter and R.L., Physical Review E 2012

Effect on spreading: Random Walks

Generalized Montroll-Weiss Equation (usually for CTRW with non-Poisson inter-event time statistics on lattices)

$$\hat{n}(s) = \frac{1}{s}\left(I - \hat{D}_T(s)\right)\left(I - \hat{T}(s)\right)^{-1} n(0)$$

$$\frac{dn}{dt} = \left(T(t) * \mathcal{L}^{-1}\left\{\hat{D}_T^{-1}(s)\right\} - \delta(t)\right) * K(t) * n(t)$$

Convolution in time

Memory kernel

Effective transition matrix

$$T_{ij}(t) = \psi_{ij}(t) \times \prod_{k \neq i} \chi_{kj}(t)$$

$$= \psi_{ij}(t) \times \prod_{k \neq i}\left(1 - \int_0^t \psi_{kj}(t')\,dt'\right).$$

Time ordering

Generalized Master Equations for Non-Poissonian Dynamics on Networks, T.H., M.A.P. and R.L.

Effect on spreading: Random Walks

The stationary solution is

$$p = \beta D_{\langle t \rangle} x$$

Normalisation constant

Average time spend on a node before leaving it

Dominant eigenvector of the effective transition matrix

The time spent on node is given by the frequency to arrive on it multiplied by the waiting time spent on it.

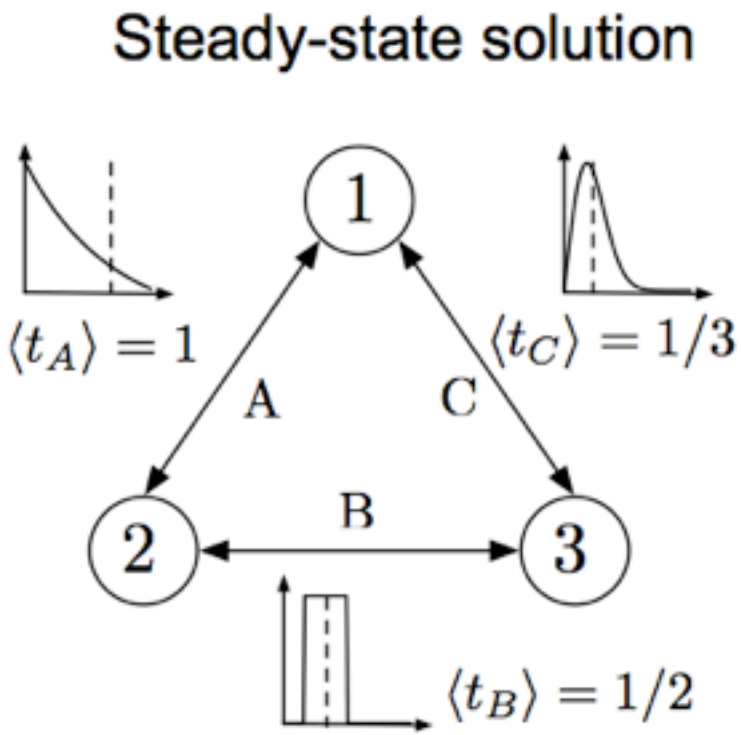
Effect on spreading: Random Walks

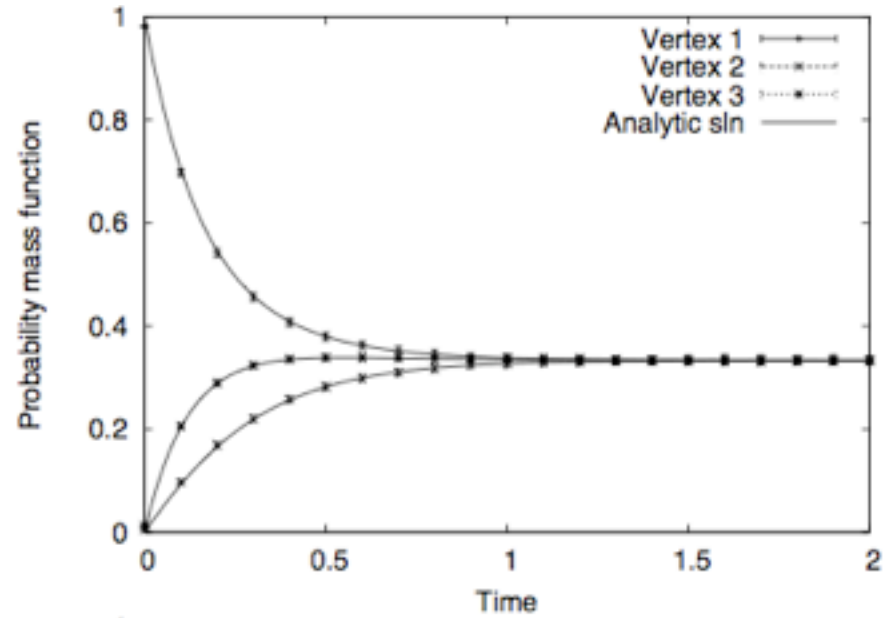
The stationary solution is

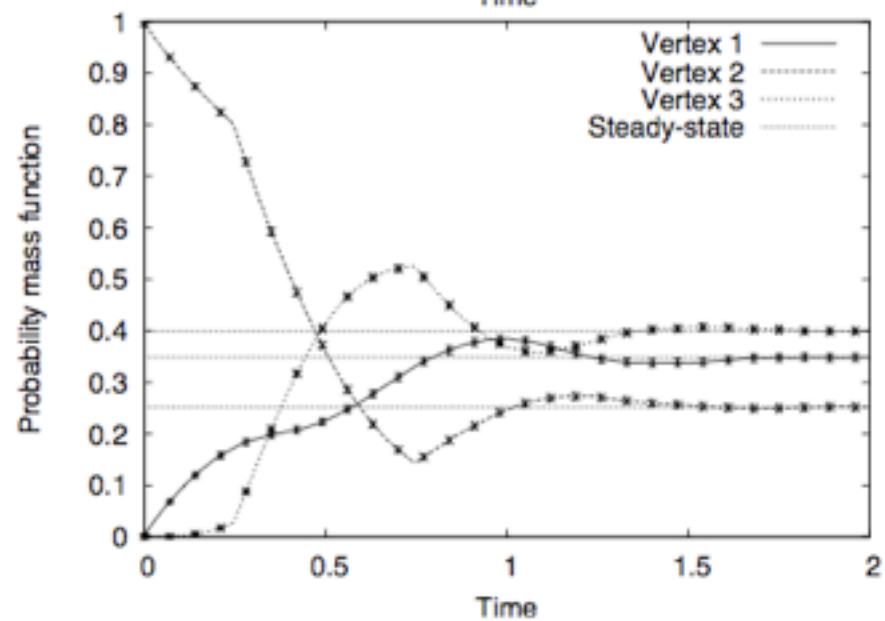
$$p = \beta D_{\langle t \rangle} x$$

Depends on the whole organisation of the graph, and the shape of the waiting time distribution (not only on its average)

Effect on spreading: Random Walks







Generalized Master Equations for Non-Poissonian Dynamics on Networks, T.H., M.A.P. and R.L.

Effect on spreading: Epidemic spreading

Epidemic spreading differs from random walk processes because the number of infected individuals is not conserved. It may decrease when an infected person recovers, or increase when an infected person infects several of its contacts.

When applied on stochastic temporal networks, standard models of epidemic spreading are characterized by two distributions: i) the probability distribution $\psi_{ij}(t)$ that the infected node *i* makes a contact sufficient to transmit the disease to node *j* at time *t*, after he has been infected at time 0; the probability distribution $r_i(t)$ that node *i* infected by the disease recovers at time *t*.

As an infected individual can only transmit the disease to a susceptible neighbor if it is still infected at the time of contact, the probability of transmission from *i* to *j*, at time *t* after *i* has been infected is given by

$$P_{ij}(t) = \psi_{ij}(t) \int_t^\infty r_i(t')dt'$$

The overall probability that node i infects node j before it recovers, called transmissibility, is given by

$$\mathbb{P}_{ij} = \int_0^\infty P_{ij}(t)dt$$

Effect on spreading: Epidemic spreading

Transmissibility (= the probability that an edge leads to a new infection) directly affects the basic reproduction number R, namely the average number of additional people that a person infects before recovering, in the limit when a vast majority of the population is susceptible.

The point R = 1 defines the epidemic threshold separating between growing and decreasing spreading.

In tree-like networks, where all nodes have the same transmissibility P, one finds $R = P\,\langle k(k-1)\rangle/\langle k\rangle$, where $\langle k(k-1)\rangle/\langle k\rangle$ is the expected number of susceptible neighbors of an infected node. The epidemic threshold is thus reduced by reducing the transmissibility, at a fixed topology.

B. Karrer and M. E. J. Newman. Phys. Rev. E 82, 016101 (2010).

Effect of the shape of the distribution

$$T_{ij}(t) = \psi_{ij}(t) \int_t^\infty \psi_{ik}(t')\, dt' \qquad P_{ij}(t) = \psi_{ij}(t) \int_t^\infty r_i(t')dt'$$

$$\mathbb{T}_{ij} \equiv \int_0^\infty T_{ij}(t)dt \qquad \mathbb{P}_{ij} = \int_0^\infty P_{ij}(t)dt$$

In general, these equations define the overall probability that an event A takes place before some other event B

$$p_A = \int_0^\infty a(t) \int_t^\infty b(t')dt'dt$$

Effect of the shape of the distribution

N.B.

$$p_A = \int_0^\infty a(t) \int_t^\infty b(t')dt'dt$$

$$p_A + p_B = 1$$

$$p_B = \int_0^\infty b(t) \int_t^\infty a(t')dt'dt$$

$$a(t) = b(t) \longrightarrow p_A = p_B = 1/2$$

$$a(t) = r_A e^{-r_A t} \text{ and } b(t) = r_B e^{-r_B t} \longrightarrow p_A = \frac{r_A}{r_A + r_B}$$

$$b(t) = r_B e^{-r_B t} \longrightarrow p_A = \int_0^\infty a(t) e^{-r_B t} dt$$

Laplace transform in the variable

Effect of the shape of the distribution

$$T_{ij}(t) = \psi_{ij}(t) \int_t^\infty \psi_{ik}(t')\, dt' \qquad P_{ij}(t) = \psi_{ij}(t) \int_t^\infty r_i(t')dt'$$

$$\mathbb{T}_{ij} \equiv \int_0^\infty T_{ij}(t)dt \qquad \mathbb{P}_{ij} = \int_0^\infty P_{ij}(t)dt$$

In general, these equations define the overall probability that an event A takes place before some other event B

$$p_A = \int_0^\infty a(t) \int_t^\infty b(t')dt'dt$$

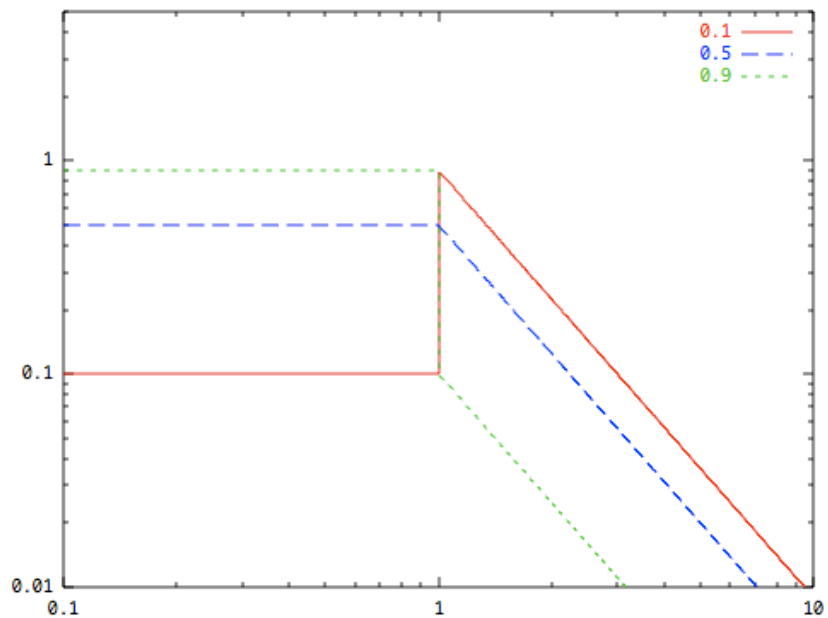
It is not the shape of the tail, nor the moments of the distribution, that affect the pathways of diffusion. What matters is instead the relative position of one distribution with another distribution. For an edge to be important, it should appear often before some other random event.

Effect of the shape of the distribution

Let us consider epidemic spreading on a regular tree of identical nodes with degree 3. Each node has the recovery distribution $r(t) = \delta(t - 1)$, e.g. recovery times occur exactly at the average value 1, and each edge is characterized by the waiting time distribution

$$\psi(t) = \begin{cases} \alpha & \text{for} \quad t < 1 \\ \frac{1-\alpha}{t^2} & \text{for} \quad t \geq 1 \end{cases}$$

where $\alpha \in [0, 1]$ tunes the shape of the distribution. For any value of α: i) the distribution is properly normalized; ii) its average is infinite; iii) it exhibits a power-law tail with exponent 2.



Effect of the shape of the distribution

Let us consider epidemic spreading on a regular tree of identical nodes with degree 3. Each node has the recovery distribution $r(t) = \delta(t - 1)$, e.g. recovery times occur exactly at the average value 1, and each edge is characterized by the waiting time distribution

$$\psi(t) = \begin{cases} \alpha & \text{for} \quad t < 1 \\ \frac{1-\alpha}{t^2} & \text{for} \quad t \geq 1 \end{cases}$$

where $\alpha \in [0, 1]$ tunes the shape of the distribution. For any value of α: i) the distribution is properly normalized; ii) its average is infinite; iii) it exhibits a power-law tail with exponent 2.

Despite sharing these properties, the transmissibility of an edge continuously varies between 0 and 1 when varying α, as

$$\mathbb{P} = \int_0^\infty \psi(t) \int_t^\infty \delta(t' - 1) dt' dt = \int_0^1 \psi(t) dt = \alpha$$

This observations implies qualitatively and quantitatively different spreading behaviours when tuning α, as the system is above the epidemic threshold when α > 1/2, and below otherwise. The important factor is instead the time-ordering of events,

Conclusion

Theoretical framework for temporal networks

Identification of the properties of temporal patterns of edges and nodes that affect pathways of diffusion on time-evolving networks.

It is not the tail of the inter-event time distribution that matters, nor its variance. The important factor is instead the **time-ordering** of events: the **importance of an edge** is the overall probability that it appears before some other event takes place. This measure of dynamical weight depends more critically on the **bulks** of the distribution rather than on their tails, because the probability mass is mainly concentrated in the bulk. In general, if the process is non-Poisson, the importance of an edge is in general not proportional to its number of activations.

Future work will focus on the transient properties of the diffusive processes, (mixing time or peak time), and on the effect of temporality on routing.

Decentralized Routing on Spatial Networks with Stochastic Edge Weights, Till Hoffmann, R.L. and Mason Porter, submitted

Thanks to:

Till Hoffmann and Mason Porter (Oxford): temporal networks

Martin Rosvall (Umea) and Andrea Lancichinetti (Northwestern): pathways

J.C. Delvenne (Louvain) and Lionel Tabourier (Oxford): diffusion

Financial support of IAP DYSCO from Belspo, Région Wallonne and FNRS

Job opening

One post-doc position for the EU Optimizr project starting in September: cascades of information in omline media