

Burstiness and Spreading on Temporal Networks

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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)

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

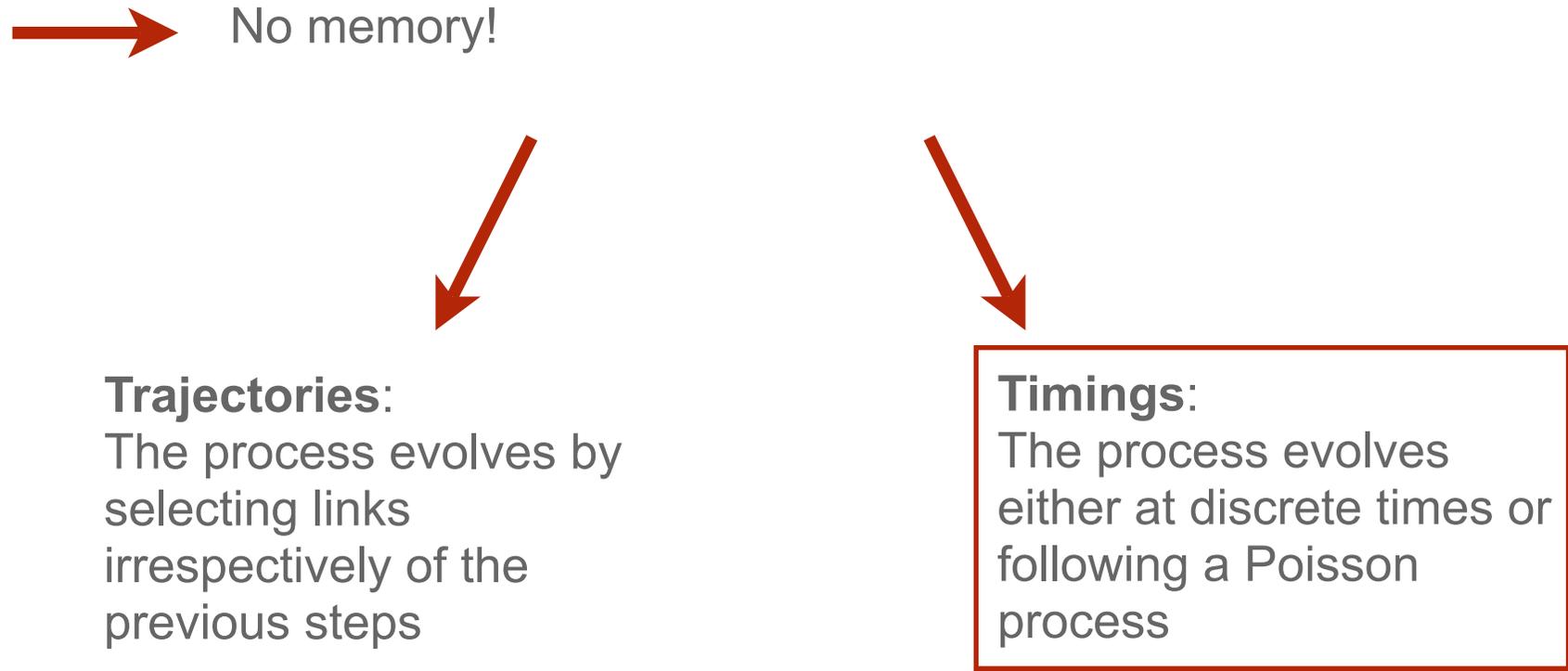
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Dynamics on stochastic temporal networks

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

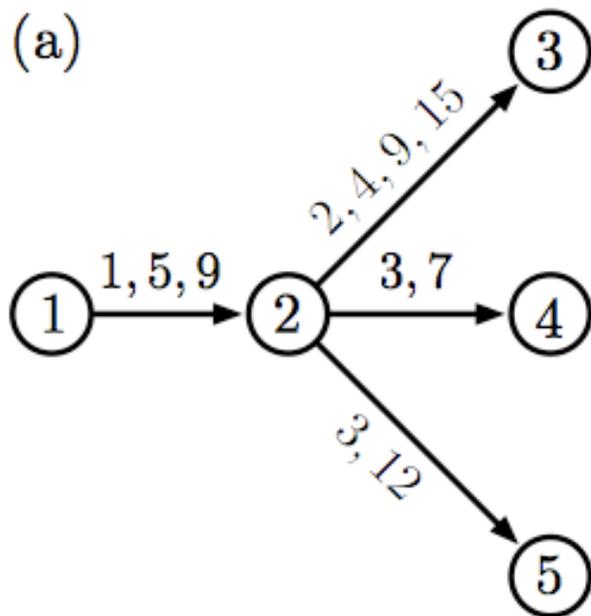
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Non-trivial patterns of activation of nodes and edges
Burstiness: intermittent switching between periods of low activity and high activity, and a fat-tailed inter-event time distributions.

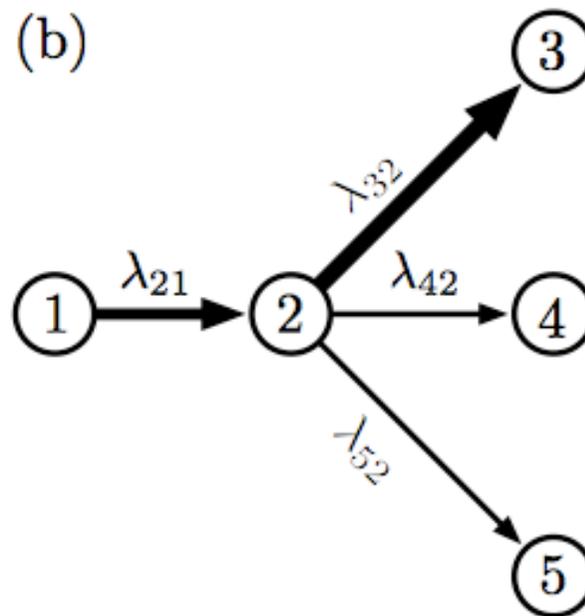
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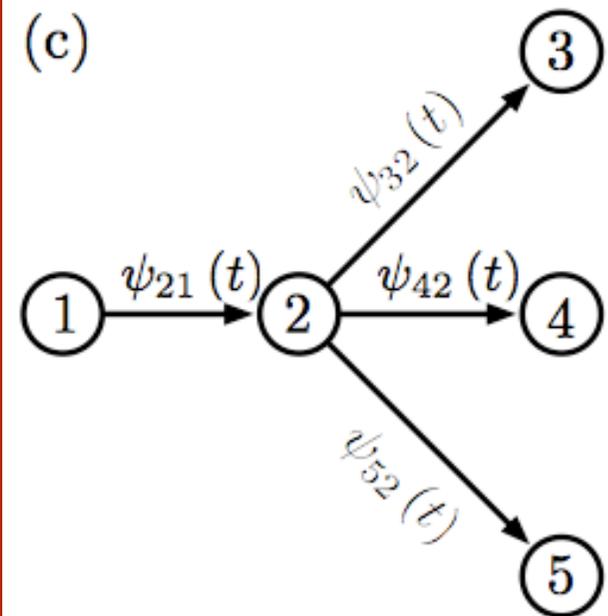
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Temporal network,
usually studied by
means of computer
simulations



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

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 .

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

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

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

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

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

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

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.

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

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

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

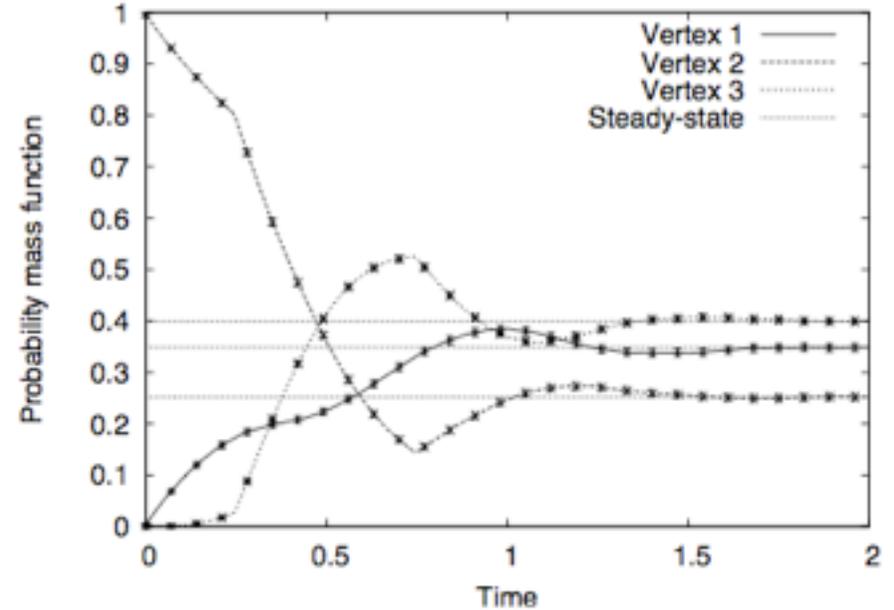
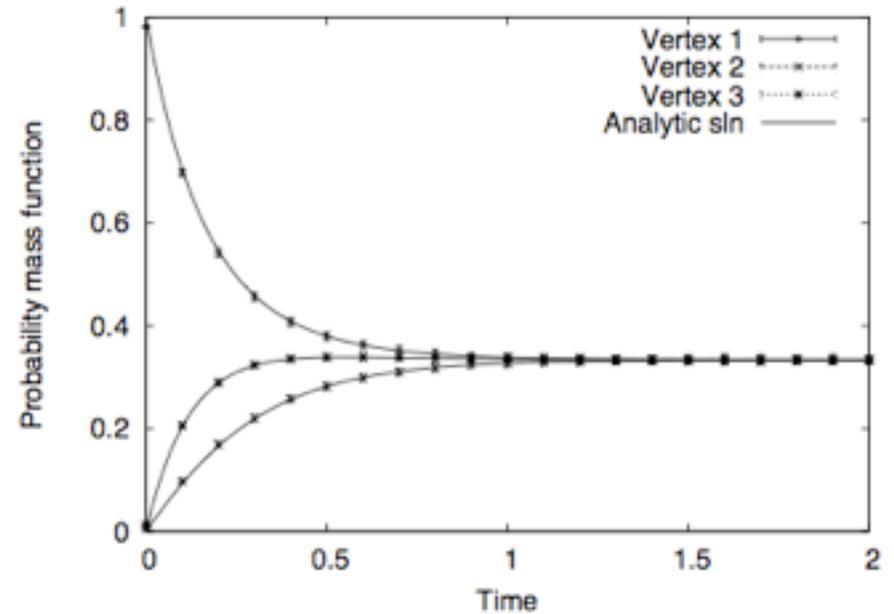
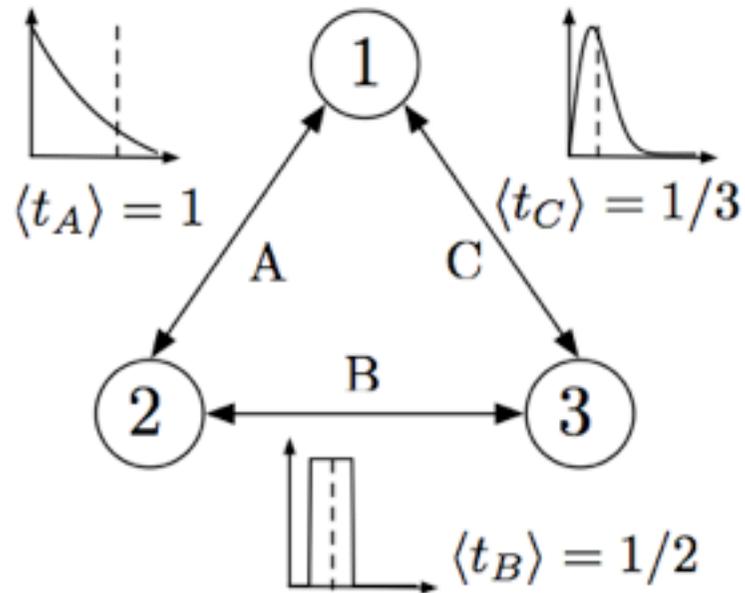
Time ordering

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

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

Effect on spreading: Random Walks

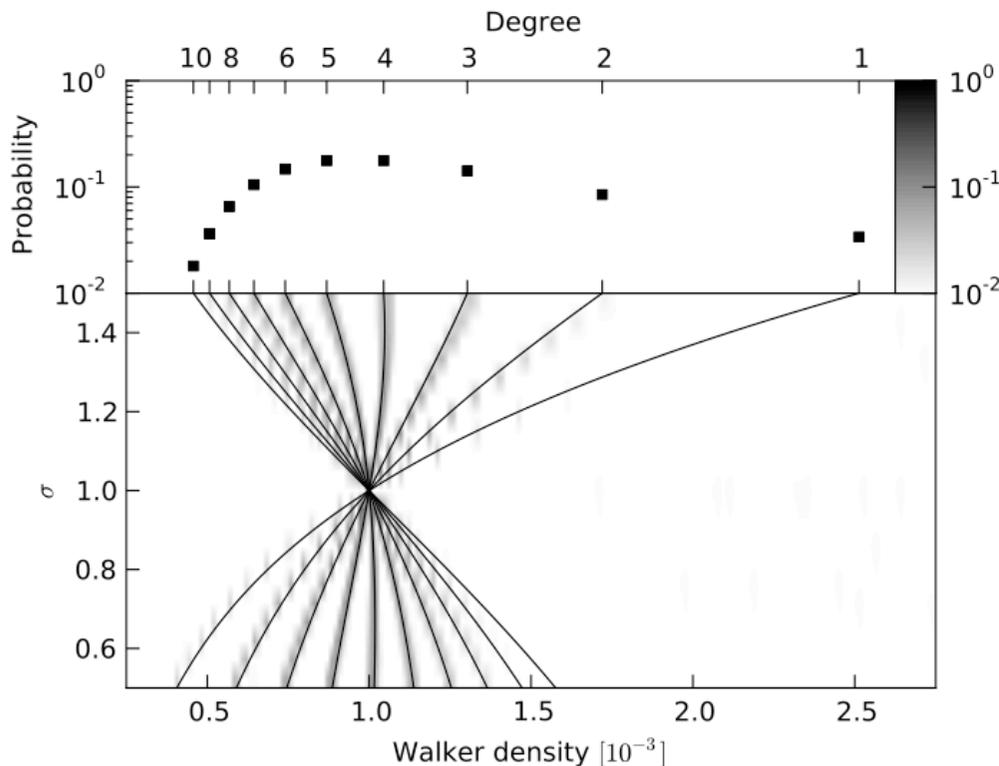
Steady-state solution



Effect on spreading: Random Walks

In systems where all edges have the same waiting time distribution, one can find a simple, closed expression for the stationary distribution

Broader waiting time distributions tend to decrease the density of walkers on hubs



$$\psi(t; \mu, \sigma) = \frac{\left(\frac{t\mu}{\sigma^2}\right)^{\mu^2/\sigma^2 - 1} \exp\left(-\frac{t\mu}{\sigma^2}\right)}{\frac{\sigma^2}{\mu} \Gamma\left(\frac{\mu^2}{\sigma^2}\right)}.$$

When $\sigma/\mu = 1$, the waiting times are exponentially distributed. Hence, we can continuously deform the distribution from an exponential distribution by changing the standard deviation σ while keeping the mean fixed at $\mu = 1$.

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.

Effect of the shape of the distribution

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

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

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

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

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

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

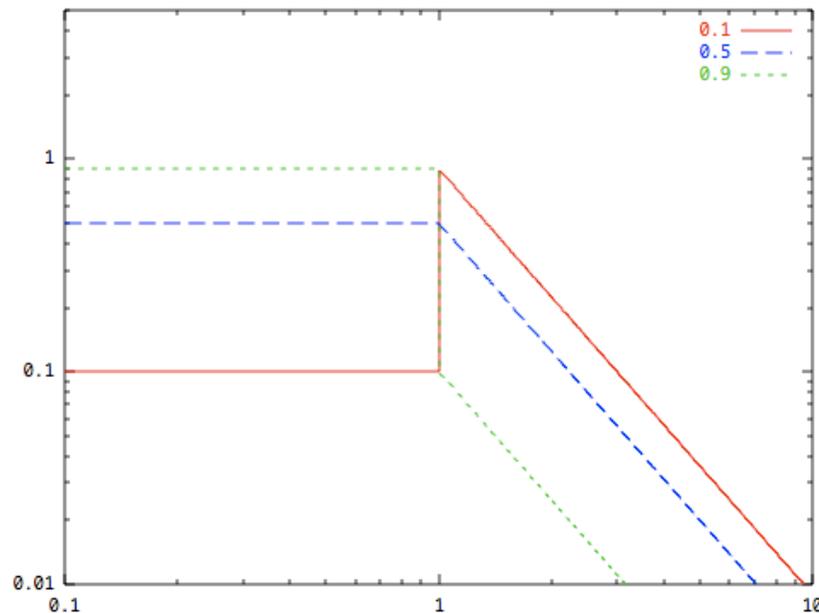
It is not the shape of the tail, nor the moments of the distribution, that affect the pathways of diffusion. What matters is instead the relative position of one distribution with another distribution. For an edge to be important, it should appear often before some other random event.

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

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

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.



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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 $\alpha > 1/2$, and below otherwise. 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.

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