Burstiness and Correlations Affect 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
Burstiness and spreading on temporal networks, R.L., L. Tabourier and J.C. Delvenne, EPJB 2013
Networks with Memory, Martin Rosvall, Alcides V. Esquivel, Andrea Lancichinetti, Jevin D. West, Renaud Lambiotte, arXiv:1305.4807
Bottlenecks, burstiness, and fat tails regulate mixing times of non-Poissonian random walks, J.-C. Delvenne, Renaud Lambiotte and L. E. C. Rocha, arXiv:1309.4155

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)

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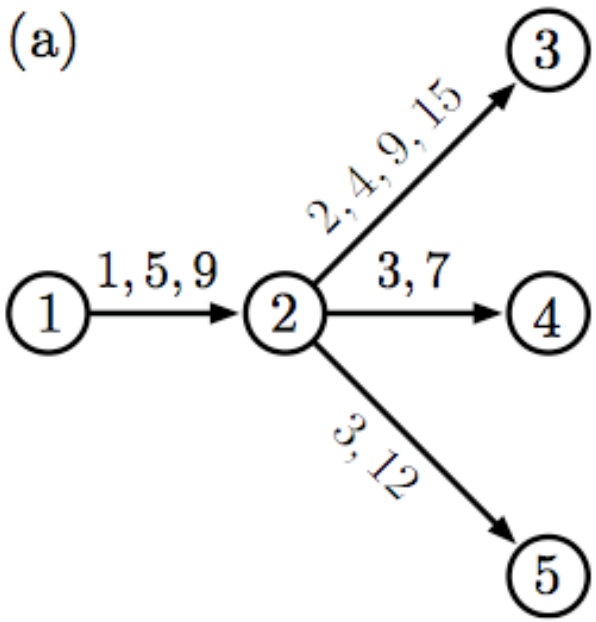
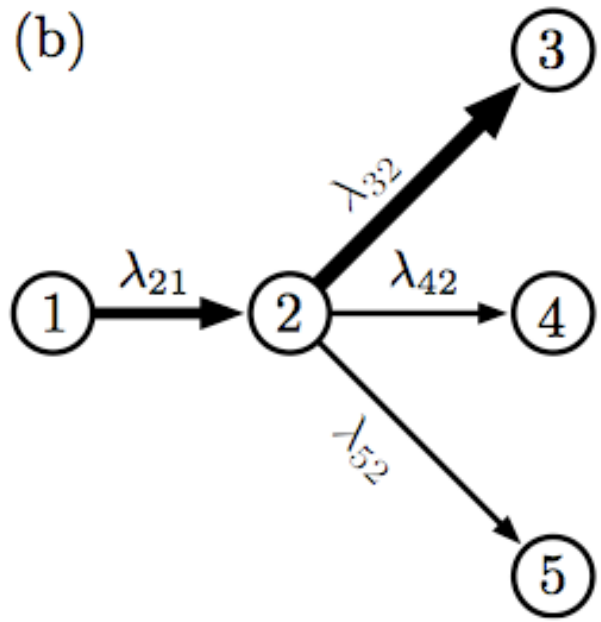
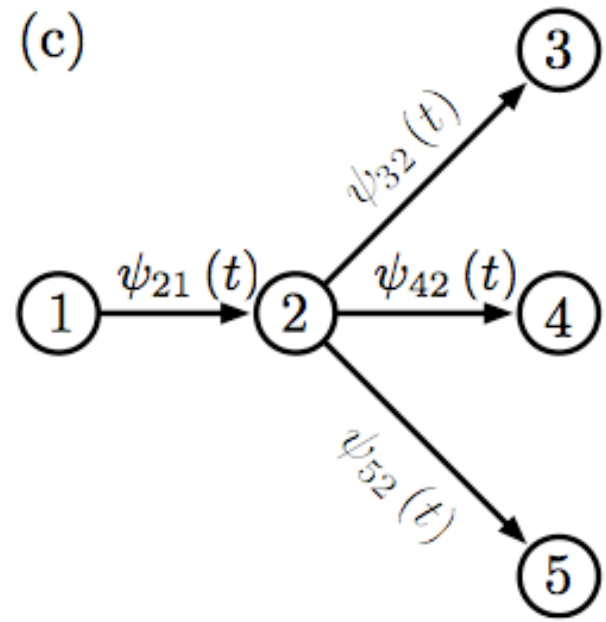


Temporal network, usually studied by means of computer simulations



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

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 independent, $\psi_{ij}(t)$ 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

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

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.

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?

Effect on spreading

Different temporal properties affect different spreading models, and different spreading properties:

Time ordering of events, how the probability mass of different probabilities are distributed:
- Biases in the trajectories of random walkers (*structural* effect on mixing time)
- Epidemic threshold in epidemic spreading

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

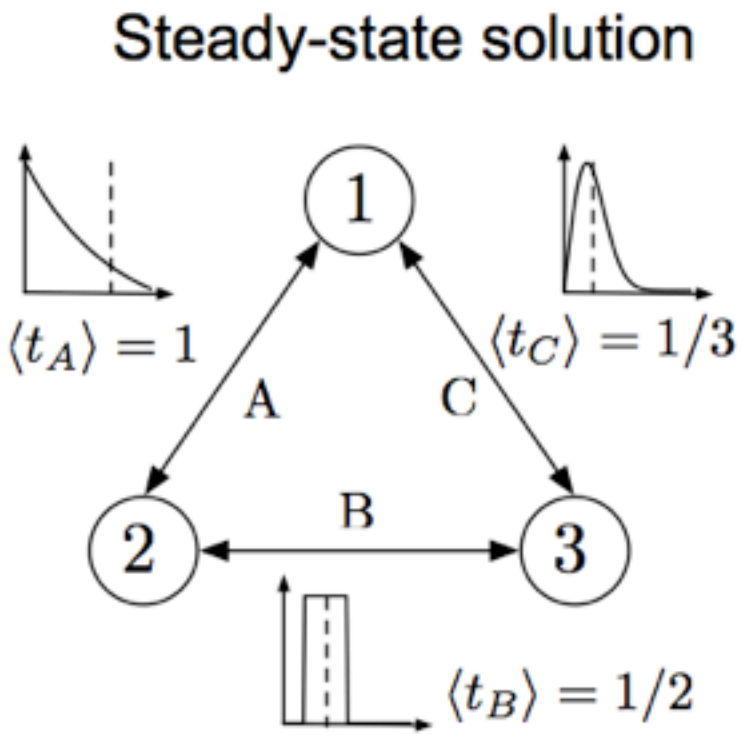
$$T_{ij}(t) = \psi_{ij}(t) \times \prod_{k \neq i} \chi_{kj}(t)$$

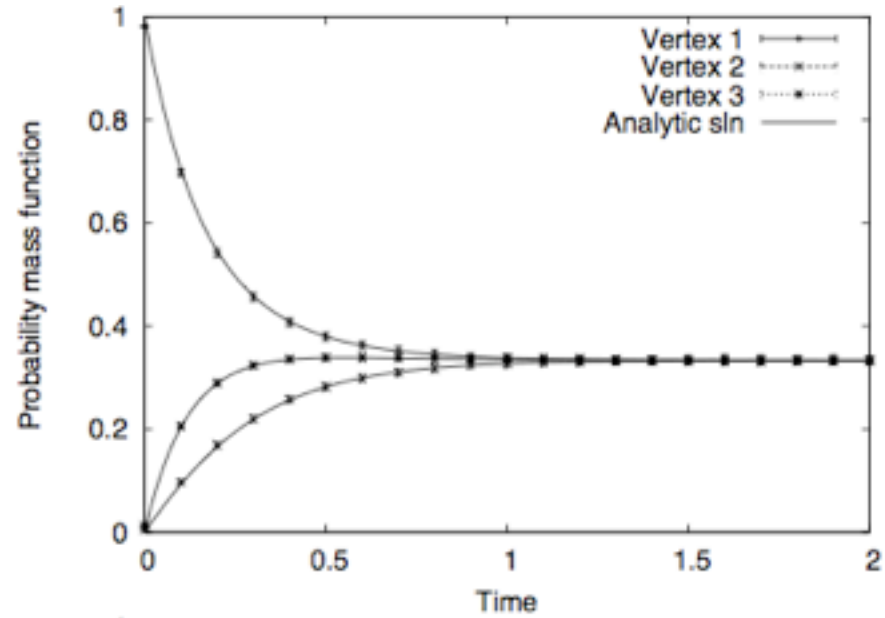
$$= \psi_{ij}(t) \times \prod_{k \neq i}\left(1 - \int_0^t \psi_{kj}(t')\,dt'\right).$$

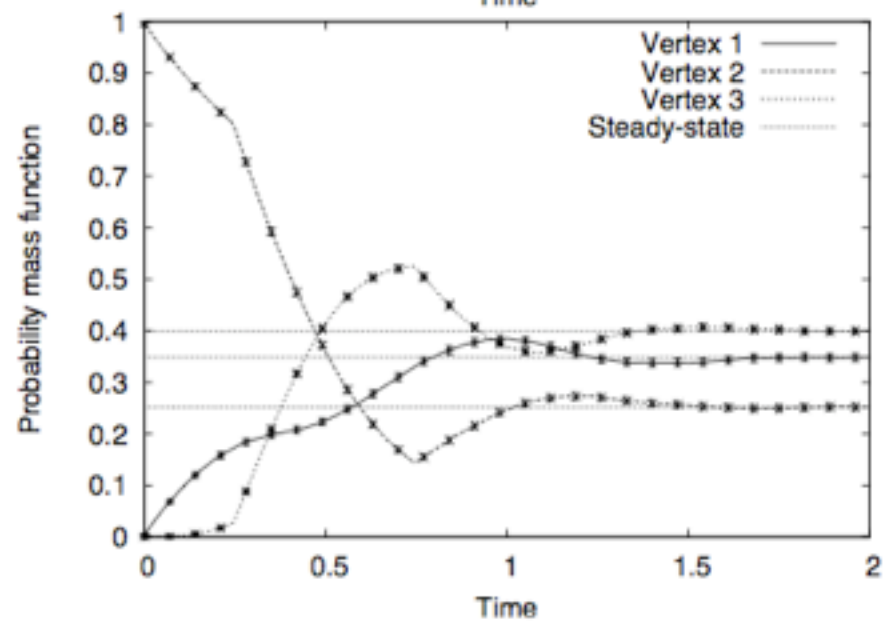
Time ordering

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

Effect on spreading: Random Walks







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

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

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.

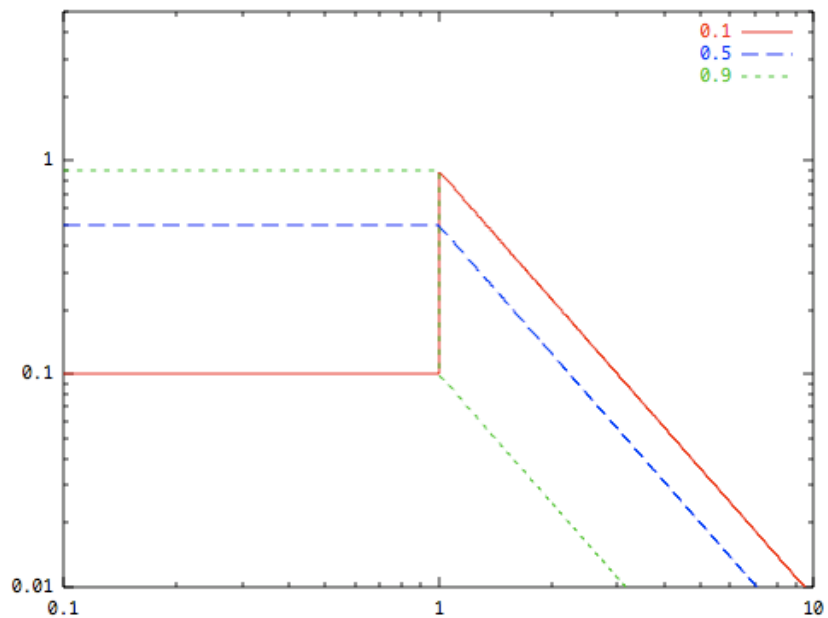
Burstiness and spreading on temporal networks, R.L., L. Tabourier and J.C. Delvenne, EPJB 2013

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

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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 , as the system is above the epidemic threshold when α > 1/2, and below otherwise. important factor is instead the time-ordering of events,

Effect on spreading

Different temporal properties affect different spreading models, and different spreading

Time ordering of events, how the probability mass of different probabilities are distributed:
- biases in the trajectories of random walkers (*structural* effect on mixing time)
- Epidemic threshold in epidemic spreading

Variance, exponential cut-off and spectral gap:
- mixing time for random walker processes

Mixing time

We consider a strongly connected, acyclic underlying network

For discrete time random walks on static networks:

$$p(t+1) = p(t)P,$$

and its solution is given by $p(t) = P^t$.

For a Poisson, continuous-time random walks on static networks:

$$\dot{p}(t) = -p(t)\tau^{-1}(I - P),$$

where I is the identity matrix, and where the solution is given by $p(t) = \exp(-\tau^{-1}(I - P))$. $I - P$ is the normalised Laplacian of the underlying, possibly weighted and directed, network.

The second dominant eigenvalue (spectral gap) determines the characteristic time necessary for the process to reach stationarity

Mixing time

In the case of continuous-time random walks with (the same) arbitrary waiting-time distribution:

$$\mathbf{p}(s) = \frac{1-\rho(s)}{s}(I - \rho(s)P)^{-1}$$

Development in small s gives the long-time behaviour:

$$\tau_{mix} = \max(\tau_0, \tau_{tail})$$

$$\exp(-t/\tau_{tail})$$

$$\tau_0 \approx \frac{\tau}{\epsilon}(1+\beta\epsilon) = \tau(\epsilon^{-1}+\beta)$$

Characteristic time of the exponential tail (cut-off)

$$\beta = (\sigma^2 - \tau^2)/2\tau^2$$

Mixing time

$$\tau_0 \approx \frac{\tau}{\epsilon}(1+\beta\epsilon) = \tau(\epsilon^{-1}+\beta)$$

$$\beta = (\sigma^2 - \tau^2)/2\tau^2$$

Combination of temporal and structural information: burstiness slows down the walker more efficiently on networks with large epsilon: random networks that have no bottlenecks, such as the Erdos-Renyi and configuration models, or small diameter graphs with no communities.

This factor incorporates the variance of the waiting time, not that of the inter-event time !!!!!!

Mixing time

In Eq. 10, the approximation $\epsilon^{-1} + \beta \approx \max(\epsilon^{-1}, \beta)$ is valid, provided that the two terms are positive and dissimilar in order of magnitude. Under those conditions, the mixing time is given by

$$\tau_{mix} \approx \max(\ \tau/\epsilon,\ \tau\beta,\ \tau_{tail}). \qquad (11)$$

Eq. 11 highlights three competing factors regulating the mixing time. While τ/ϵ is essentially a topological factor, capturing the effect of the structural bottleneck, the second term captures the burstiness-driven slowdown (as in [3]) and τ_{tail} quantifies the 'fatness' of the tail of the distribution of waiting times [7, 9]. Burstiness

Either of the three factors may be dominant in real-life data.

Bottlenecks, burstiness, and fat tails regulate mixing times of non-Poissonian random walks, J.-C. Delvenne, Renaud Lambiotte and L. E. C. Rocha, arXiv:1309.4155

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Effect on spreading

Waiting time distribution

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- Epidemic threshold in epidemic spreading

Variance, exponential cut-off and spectral gap:
- mixing time for random walker processes

Correlations

Correlations between successive events: effect on the spectral gap

From data to models

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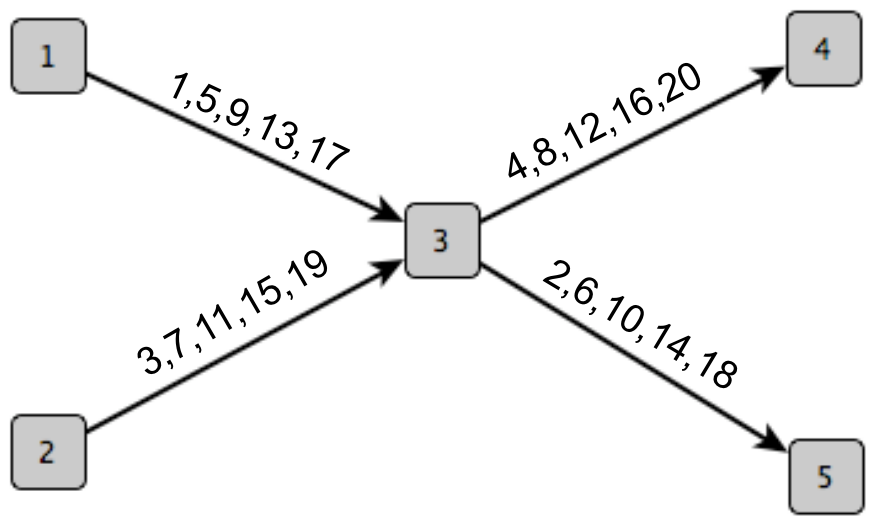
A.O. Allen. *Probability, Statistics, Queueing Theory: With Computer Science Applications*, 1990.

Memory affects mixing time

Correlations between the activation times of neighbouring edges

=> induces non-random pathways: where one goes to depends on where one comes from



Memory network
Betweenness preference

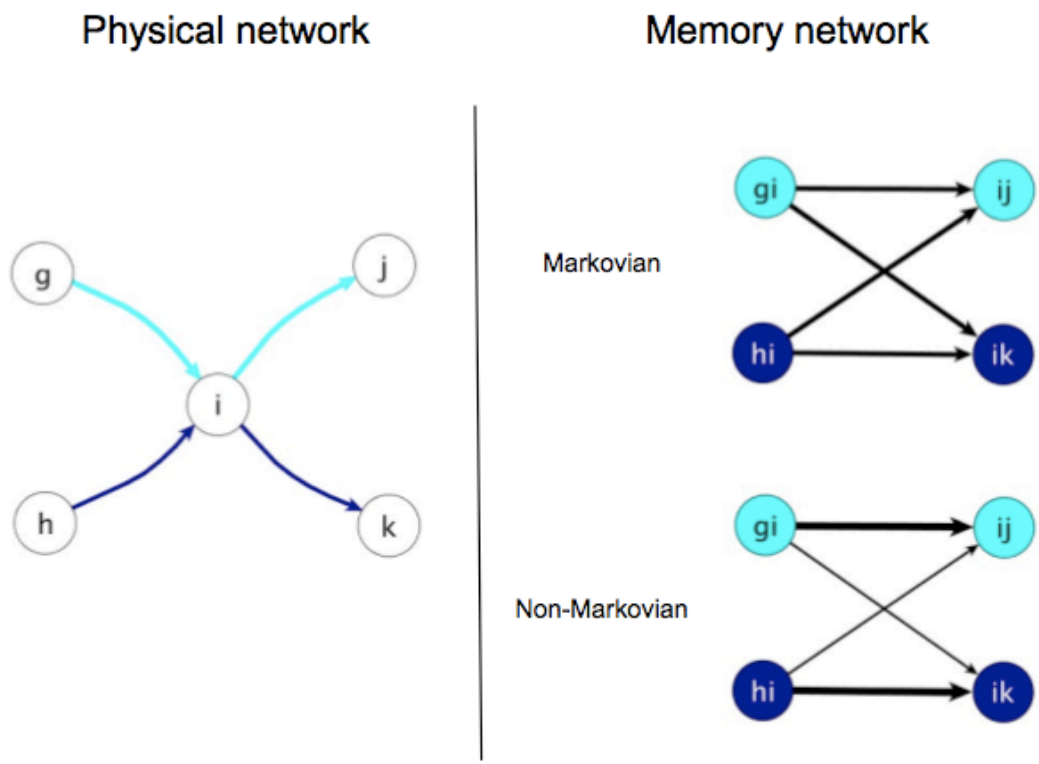
→ Second-order Markov

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

Slow-Down vs. Speed-Up of Information Diffusion in Non-Markovian Temporal Networks, I Scholtes, N Wider, R Pfitzner, A Garas, C Juan Tessone and F Schweitzer, arXiv:1307.4030

Memory affects mixing time

Second-order Markov: transitions from directed edges to directed edge (memory node)
Memory may induce biases in the transition between memory nodes



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Memory affects mixing time

Does memory accelerate or slow-down diffusion?

Scholtes et al. show numerically that the spectral gap can either increase or decrease in different real-life and artificial systems
Rosvall et al. show that modularity increases in empirical systems (=> slows down diffusion)

We look for an explicit expression for the effect of memory on the spectral gap (and hence on the mixing time)

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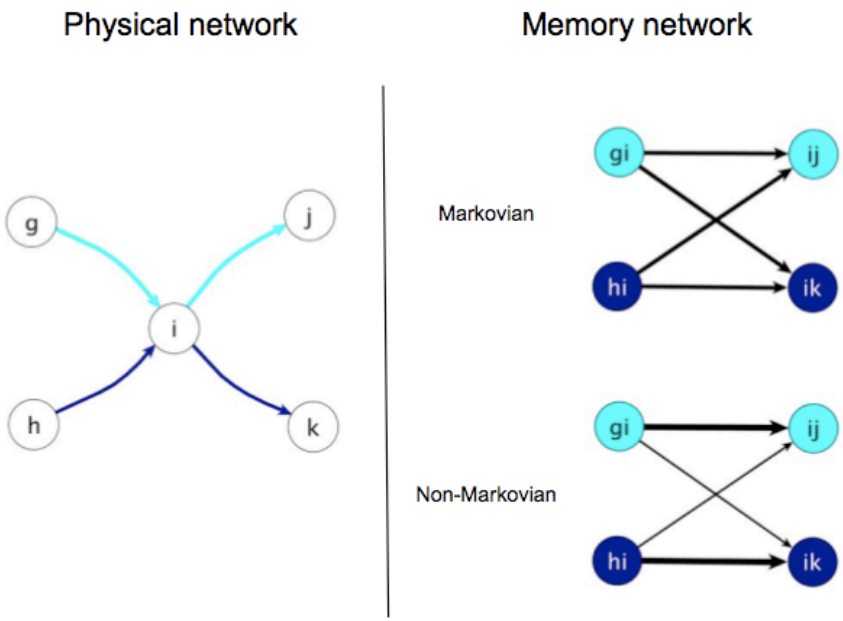


Random walk on the memory network

$$P(\beta;t+1)=\sum_{\alpha}P(\alpha;t)T_{\alpha\beta}$$

If the dynamics is memoryless, uniform transition:

$$T^{M}_{\alpha\beta}=\begin{cases}1/k^{\text{out}}_{\alpha} & \text{for } \beta\in\sigma^{\text{out}}_{\alpha},\\ 0 & \text{otherwise,}\end{cases}$$

(Left and right) eigenvectors of the spectral gap associated to the best bi-partition of the network (Fiedler)

$$\mathrm{uT}=\lambda_2\mathrm{u}$$

Small deviation to the Markovian case and perturbation analysis:

$$T=T^{M}+\Delta T$$

Memory affects mixing time

$$\Delta\lambda_2 = \frac{\sum_{\alpha\beta} u_\alpha^M \Delta T_{\alpha\beta} v_\beta^M}{\sum_\alpha u_\alpha v_\alpha}$$

Interplay between memory and the (dominant) bi-modular structures:

- if memory enhances flows inside communities => slowing down of diffusion

- if memory enhances flows across communities => acceleration of diffusion

Memory affects mixing time

Physical network

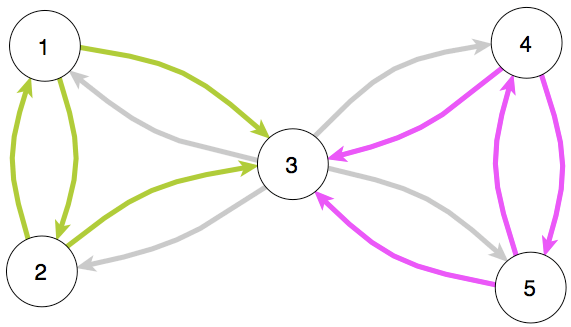


FIG. 2: Illustration of the bow tie network studied in detail in the main text. When the process is Markovian and that the transitions between memory nodes are uniform, the second dominant (left) eigenvector, of eigenvalue $1/2$, is represented by the color code: 1 for green memory nodes, -1 for pink ones, and 0 for grey ones.

Memory network

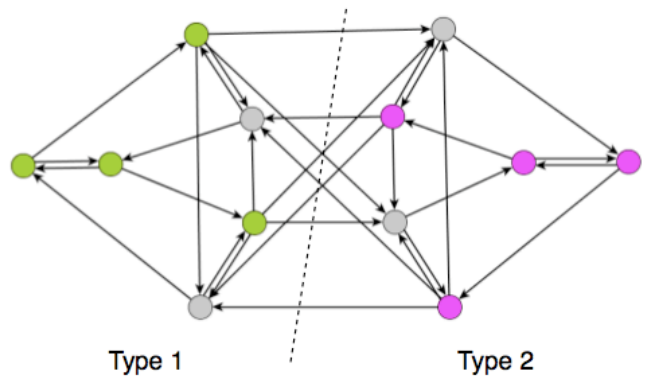
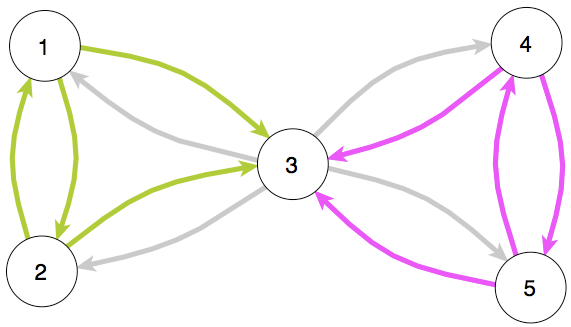


FIG. 3: Representation of the memory network associated to Fig. 2. The same color code has been used. The non-Markovian process is defined by partitioning the network into two groups, and by assigning different types of transitions across and within groups.

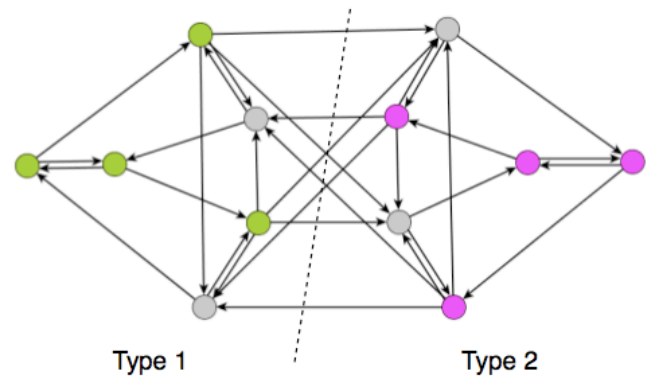
The non-Markovian dynamics is modelled as follows: memory nodes are partitioned into two groups. The weight of a transition between nodes of the same type (different types) is 1+ε (1-ε).

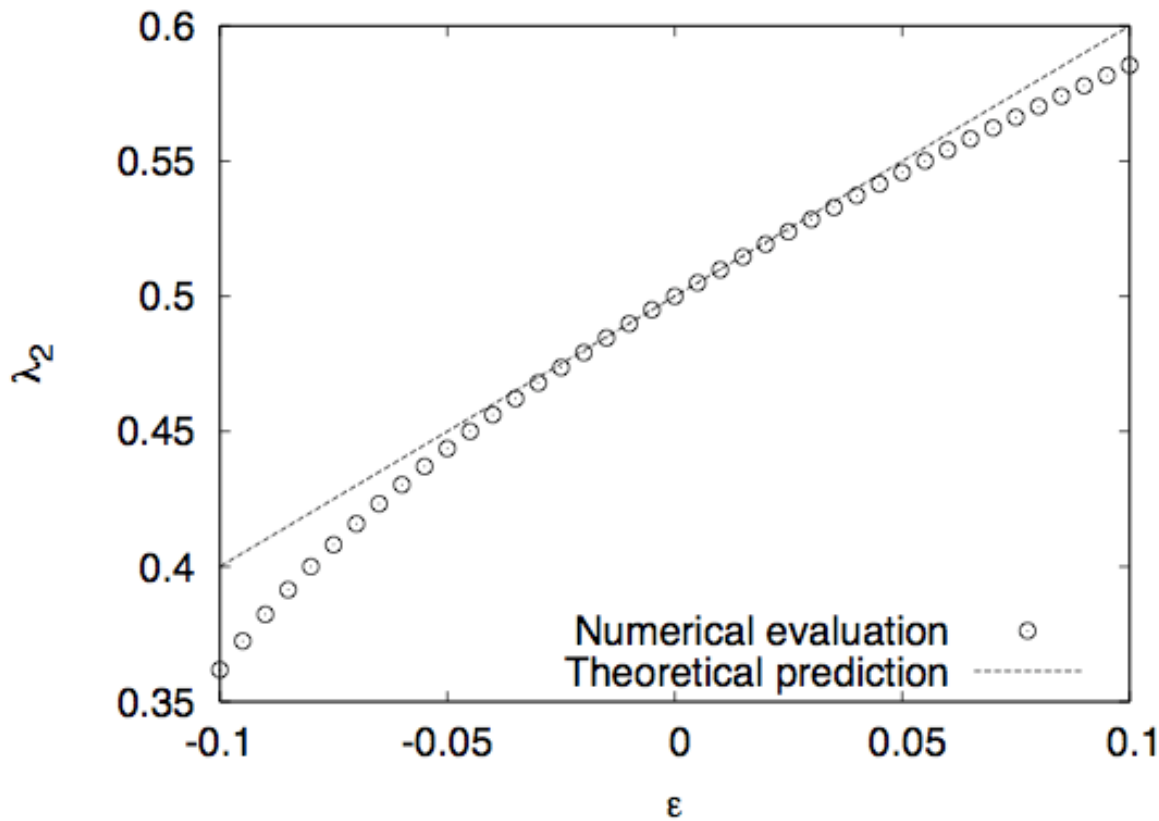
Memory affects mixing time

Physical network



Memory network





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.

Dynamics affect diffusion in different ways:

Importance of the **time-ordering** of events to define the **importance of an edge** => leads to biases for random walks and modifies the epidemic threshold (the bature of the tail of the distribution only has an indirect effect)

Continuous-time random walks: **mixing time** is determined by a combination of three factors: the spectral gap (structural bottlenecks, the second moment of the waiting time distribution, and its exponential tail. Mixing time is more sensitive to temporal bursts in the absence of topological bottlenecks.

Second-order Markov process: temporal **correlations** modify flows of probability. Memory either slows down or accelerates diffusion depending on the rearrangement of flows across the **modular** structure of the system

Thanks to:

Till Hoffmann and Mason Porter (Oxford): temporal networks

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

J.C. Delvenne (Louvain), Luis Rocha (Louvain) and Lionel Tabourier (Namur): diffusion

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More info: http://xn.unamur.be