Supplementary Material for the paper: Equivalence between non-Markovian and Markovian dynamics in epidemic spreading processes

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DERIVATION OF THE DIFFERENTIAL EQUATION FOR THE $n-$POINT CORRELATION FUNCTION

The average of Eq (1) of the main text given the state of the system at time t, $\mathbf{n}(t) \equiv (n_1(t), n_2(t), \cdots, n_N(t))$, can be written as

$$\langle n_i(t+dt)|\mathbf{n}(t)\rangle = n_i(t) + dtA_i(t), \qquad A_i(t) = -n_i(t)\delta[t_i(t)] + (1-n_i(t))\sum_j a_{ij}\lambda[\tau_{ji}(t)]n_j(t). \tag{1}$$

Let us consider a set of n nodes $\mathcal{I} \equiv \{i_1, i_2, \cdots i_n\}$. The correlation function between these n nodes reads

$$\dot{\rho}_{i_1\cdots i_n}(t) = \frac{1}{dt}\left\langle \left[\prod_{i\in\mathcal{I}}\langle n_i(t+dt)|\mathbf{n}(t)\rangle - \prod_{i\in\mathcal{I}} n_i(t)\right]\right\rangle, \tag{2}$$

where the outer average is over the state of the system at time t. Notice also that the factorization in the first term of this equation is a direct consequence of the independence of the random variables ξ_i and η_i in Eq. (1) of the main text for different nodes. The first term in Eq (2) can be written, by means of Eq (1) as

$$\prod_{i\in\mathcal{I}}\langle n_i(t+dt)|\mathbf{n}(t)\rangle = \sum_{k=0}^{n}\sum_{\{\mathcal{I}_k\}}(dt)^k\prod_{i\in\mathcal{I}_k} n_i(t)\prod_{l\in\mathcal{I}_k} A_l(t), \tag{3}$$

where $\{\mathcal{I}_k\}$ is the set of all subsets of $\mathcal{I}$ containing k nodes. Because of the term dt^k, however, the expansion to linear order in dt is a reduced sum over $k<2$, and thus

$$\prod_{i\in\mathcal{I}}\langle n_i(t+dt)|\mathbf{n}(t)\rangle = \prod_{i\in\mathcal{I}} n_i(t) + dt\sum_{i\in\mathcal{I}} A_i(t)\prod_{k\in\mathcal{I}\setminus i} n_k(t). \tag{4}$$

Therefore the $n-$point correlation function reads

$$\dot{\rho}_{i_1\cdots i_n}(t) = \langle\sum_{i\in\mathcal{I}}(1-n_i(t))\sum_j a_{ij}\lambda[\tau_{ji}(t)]n_j(t)\prod_{k\in\mathcal{I}\setminus i} n_k(t) - \sum_{i\in\mathcal{I}} n_i(t)\delta(t_i(t))\prod_{k\in\mathcal{I}\setminus i} n_k(t)\rangle \tag{5}$$

$$= \sum_{i\in\mathcal{I}}\left\langle\left[(1-n_i(t))\sum_j a_{ij}\lambda[\tau_{ji}(t)]n_j(t) - n_i(t)\delta(t_i(t))\right]\prod_{k\in\mathcal{I}\setminus i} n_k(t)\right\rangle, \tag{6}$$

from which Eq (6) of the main text follows immediately.

TIME τ_{ij} OF ACTIVE LINK $i-j$ DOES NOT DEPEND ON THE STATES OF OTHER NODES DIFFERENT FROM i AND j

A critical step in our approach is to prove that

$$\text{Prob}(\tau_{ji}; t|n_i=0, n_j=1, \{n_k=1, k\in\mathcal{I}_i\}) = \text{Prob}(\tau_{ji}; t|n_i=0, n_j=1). \tag{7}$$

The probability in the left hand side of this equation can be written as

$$\text{Prob}(\tau_{ji}; t|n_i = 0, n_j = 1, \{n_k = 1, k \in \mathcal{I}_i\}) = \int \cdots \int \phi(\tau_i^R, \tau_j^I, \{\tau_k^I\}; t)\phi(\tau_{ji}|\tau_i^R, \tau_j^I, \{\tau_k^I\})d\tau_i^R d\tau_j^I \prod_{k\in\mathcal{I}_i} d\tau_k^I, \tag{8}$$

where $\phi(\tau_i^R, \tau_j^I, \{\tau_k^I\}; t)$ is the joint probability density, at time t, that given that node i is susceptible and nodes j and $\{k \in \mathcal{I}_i\}$ are infected, the time elapsed since i recovered is τ_i^R and the times elapsed since j and $\{k \in \mathcal{I}_i\}$ became infected are τ_j^I and $\{\tau_k^I\}$, respectively. By Bayes' rule, $\phi(\tau_{ji}|\tau_i^R, \tau_j^I, \{\tau_k^I\})$ is the probability density of the time τ_{ji} conditioned on the times $\tau_i^R, \tau_j^I, \{\tau_k^I\}$. However, it is easy to see that since infection events take place in active links independently, once τ_i^R and τ_j^I are fixed, τ_{ji} is totally independent of the elapsed times since nodes other than j became infected. Therefore,

$$\phi(\tau_{ji}|\tau_i^R, \tau_j^I, \{\tau_k^I\}) = \phi(\tau_{ji}|\tau_i^R, \tau_j^I), \tag{9}$$

which directly gives the result in Eq. (7).

GENERAL FORMALISM FOR λ_{eff}

Using the result in Eq. (9), at the steady state the probability density $\phi(\tau_{ji})$ of the time elapsed since the infection process of node j to node i started, given that node i is susceptible and node j is infected, can be written in general as

$$\phi(\tau_{ji}) = \int\int \phi(\tau_{ji}|\tau_j^I, \tau_i^R)\phi(\tau_j^I, \tau_i^R)d\tau_j^I d\tau_i^R, \tag{10}$$

where $\phi(\tau_j^I, \tau_i^R)$ is the joint probability that the time elapsed since j became infected is equal to τ_j^I and the time elapsed since i recovered is equal to τ_i^R. If we assume that the two process are uncorrelated, $\phi(\tau_j^I, \tau_i^R)$ can be factorized into $\phi(\tau_j^I, \tau_i^R) = \phi_I(\tau_j^I)\phi_R(\tau_i^R)$, and Eq. (10) reduces to

$$\phi(\tau_{ji}) = \int_0^\infty d\tau_j^I \phi_I(\tau_j^I) \int_0^\infty d\tau_i^R \phi_R(\tau_i^R)\Big\{\Theta(\tau_j^I - \tau_i^R)\phi(\tau_{ji}|\tau_i^R \le \tau_j^I) + \Theta(\tau_i^R - \tau_j^I)\phi(\tau_{ji}|\tau_i^R > \tau_j^I)\Big\}, \tag{11}$$

where $\Theta(t)$ is the Heaviside step function, $\phi_I(\tau_j^I)$ is the probability that the time elapsed since j became infected is equal to τ_j^I and $\phi_R(\tau_i^R)$ is the probability that the time elapsed since i recovered is equal to τ_i^R. The conditional probability $\phi(\tau_{ji}|\tau_i^R > \tau_j^I)$ is simply $\phi(\tau_{ji}|\tau_i^R > \tau_j^I) = \delta(\tau_{ji} - \tau_j^I)$, and

$$\phi(\tau_{ji}|\tau_i^R \le \tau_j^I) = \int_0^\infty \Theta(\tau_j^I - \tau_i^R - \tau)\delta(\tau_{ji} - (\tau_j^I - \tau))\Psi_I(\tau_j^I - \tau_i^R - \tau)\sum_{n=0}^\infty P_n(\tau)d\tau, \tag{12}$$

where n is the number of infection attempts of node j to node i, $P_n(\tau)$ is the probability that the time elapsed since node j became infected and the moment of his n-th fire is equal to τ, and $\Psi_I(\tau_j^I - \tau_i^R - \tau)$ is the probability that the time elapsed between the n-th fire and the $n+1$-th fire is greater than $\tau_{ji} - \tau_i^R$. As computed in Eq. (4) of the main text, the probability that the time elapsed since j became infected is equal to τ_j^I is simply

$$\phi_I(\tau_j^I) = \tilde{\delta}\Psi_R(\tau_j^I). \tag{13}$$

The survival probability $\Psi_R(\tau_j^I)$ of recovery events can be written as

$$\Psi_R(\tau_j^I) = \int_0^\infty \omega(u)e^{-u\tau_j^I}du = \widehat{\omega}(\tau_j^I), \tag{14}$$

where $\omega(u)$ is the inverse Laplace transform of $\Psi_R(\tau_j^I)$. In Laplace space, the probability distribution $P_n(\tau)$ has a convenient form, $\widehat{P}_n(u) = \left[\widehat{\psi}_I(u)\right]^n$, where $\widehat{\psi}_I(u)$ is the Laplace transform of $\psi_I(t)$. By inserting Eqs. (13) and (14) into Eq. (11) we obtain

$$\phi(\tau_{ji}) = \tilde{\delta}\int_0^\infty d\tau_i^R \phi_R(\tau_i^R)\int_0^\infty du\, e^{-u\tau_{ji}}\omega(u)\left\{\theta(\tau_i^R - \tau_{ji}) + \theta(\tau_{ji} - \tau_i^R)\Psi_I(\tau_{ji} - \tau_i^R)\frac{1}{1-\widehat{\psi}_I(u)}\right\}. \tag{15}$$

By inserting the form of $\phi(\tau_{ji})$ into Eq. (10) of the main text, we obtain an expression for the infection rate λ_{eff}

$$\lambda_{eff} = \int_0^\infty du\, \omega(u) \left\{ \widehat{[\lambda_I \Phi_R]}(u) + \widehat{[\lambda_I\, [\phi_R * \Psi_I]]}(u) \frac{1}{1-\widehat{\psi}_I(u)} \right\}, \tag{16}$$

where λ_I is the infection hazard rate, $\Phi_R(\tau_i^R)$ and $\Psi_I(t)$ are the survival probabilities of $\phi_R(\tau_i^R)$ and $\psi_I(t)$, respectively and $\phi_R * \Psi_I$ is the convolution between $\phi_R(\tau_i^R)$ and $\Psi_I(t)$. At this point, some ansatz regarding the form of $\phi_R(\tau_i^R)$, the probability that the time elapsed since i recovered is equal to τ_i^R, is needed to continue. We note that if one does not consider the state of node i in the probability $\phi_R(\tau_{ji})$, which corresponds to inserting $\phi_R(\tau_i^R) = \delta(\tau_i^R)$ into Eq. (15), one obtains

$$\lambda_{mf} = \tilde{\delta} \int_0^\infty \omega(u) \frac{\widehat{\psi}_I(u)}{1-\widehat{\psi}_I(u)} du. \tag{17}$$

This effective infection rate λ_{mf}, already found in Cator et al. [1] by using a mean field approximation, is now obtained within a more general formalism. A different possibility is to consider $\phi_R(\tau_i^R)$ equal to an exponential distribution,

$$\phi_R(\tau_i^R) = w e^{-w\tau_i^R}, \tag{18}$$

with rate w. The rate w can be written as a simple function of the prevalence ρ, $w = \delta\rho/(1-\rho)$. However, even if it were not possible to find a closed analytic form for the effective infection rate, one can resort to numerical simulation in order to compute λ_{eff}. One can see that the exponential ansatz for the form of $\phi_R(\tau_i^R)$ is correct for large values of the prevalence ρ, but it fails for low prevalence, thus close to the epidemic threshold.

APPROXIMATIONS TO λ_{app}

To find an infection rate which is accurate and analytically treatable close to the epidemic threshold, we follow a different approach. As stated in the main text, we consider here the probability density $\psi(\tau_{ji}) \equiv \lim_{t\to\infty} p(\tau_{ji}, n_i = 0; t | n_j = 1)$, which is the join probability that, given that node j is infected at the observation time, node i is susceptible and the time elapsed since the last infection attempt from j to i is equal to τ_{ji}. Our approximation consists of estimating the probability that node i is susceptible at the observation time t, which depends on the time instant at which node i became infected, this time instant being unknown in principle.

We first consider the case of low prevalence, $\rho^{st} \ll 1$. Then, let us consider separately the cases in which node j attempts at least once to infect node i, $n > 0$, and the case of no attempts, $n = 0$. In the first case, at the time of the last infection event from j to i, $t - \tau_{ji}$, node i either was already infected (in which case the infection attempt is ineffective) or it became infected by this event (see Fig. 1a). In both cases, we are certain that node i is in an infected state at time $t - \tau_{ji}$ and, thus, the probability that node i recovers before the observation time t is $1 - \Psi_R(\tau_{ji})$. If the prevalence is low, the probability that node i is subsequently infected by one of its neighbor (other than j) and then recovers before the observation time is also very low, and we assume it to be zero. With these assumption, the probability that node i is susceptible at time t is simply $1 - \Psi_R(\tau_{ji})$. If node j does not attempt to infect node i, we cannot know for certain the state of node i. However, given that node j became infected at time $t - \tau_j^I$, one of his neighbors must have infected him. Let us consider that node j has degree k. If the prevalence is low, it is very unlikely to find more than one neighbor of node j infected simultaneously and we assume that only one of his neighbors was infected and infected him. With probability $1/k$, such infected node is node i (See Fig. 1b), so that i is infected at time $t - \tau_j^I$, and the probability that node i is then susceptible at the observation time t is $1 - \Psi_R(\tau_j^I)$. With probability $1 - 1/k$, the infected node is a neighbor other than i and, thus, we assume that node i was susceptible at time $t - \tau_j^I$ and it will remain in this state until time t with probability equal to one. Summing up, if node j attempts at least once to infect node i, then the probability that it is susceptible at the observation time is $1 - \Psi_R(\tau_{ji})$. Instead, if node j does not attempt to infect node i, this probability reads $(1 - \Psi_R(\tau_j^I))/k + (k-1)/k = (k - \Psi_R(\tau_j^I))/k$. In the limit of low prevalence, we expect this approximation to be exact. In the following, we also approximate the value of k by the average degree, $\langle k \rangle$.

The probability density $\psi(\tau_{ji})$ can be written as

$$\psi(\tau_{ji}) = \int_0^\infty \psi(\tau_{ji} | \tau_j^I) \phi_I(\tau_j^I) d\tau_j^I, \tag{19}$$

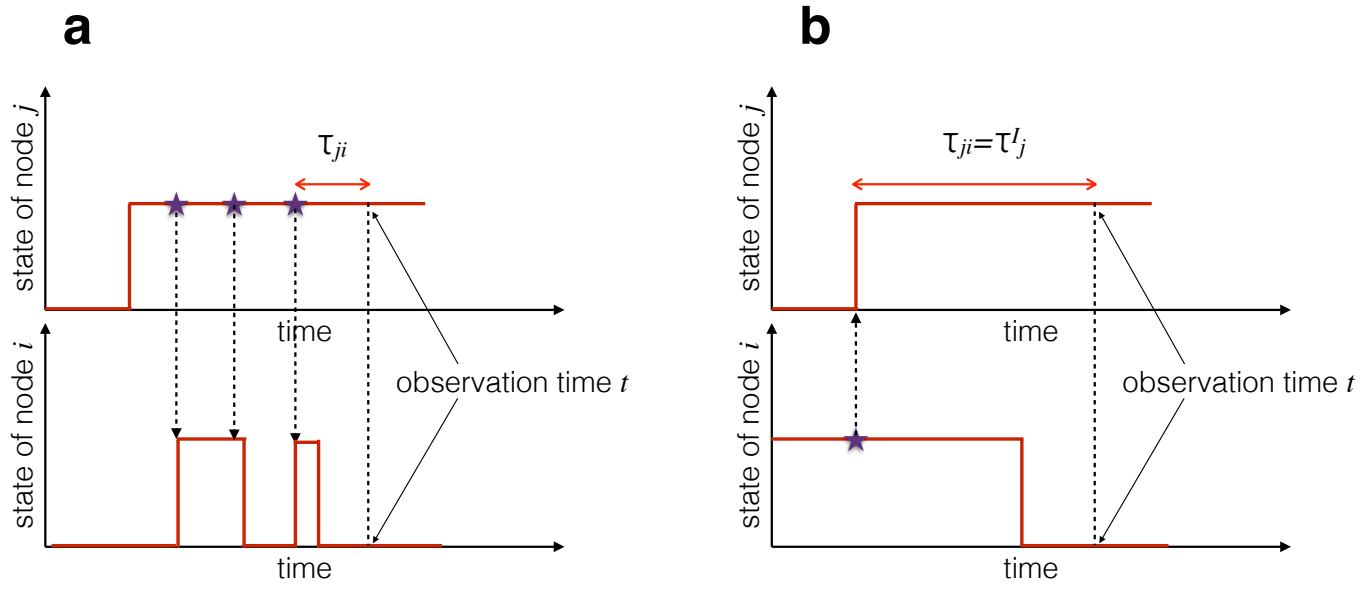

Figure 1: Sketch of two possible ways to obtain the time τ_{ji} and, simultaneously, node i infected at the observation time. In **a**, node j has attempted to infect i at least once at time $t-\tau_{ji}$. This implies that node i must necessarily be infected at that moment and, thus, it has to recover before the observation time. In **b**, node j has not attempted to infect i since it became infected by node i. In this case, we know that i was infected when j became infected and, again, it has to recover before the observation time.

where again $\phi_I(\tau_j^I)$ is the probability that the time elapsed since j became infected is equal to τ_j^I. The conditional probability $\psi(\tau_{ji}|\tau_j^I)$ is

$$\psi(\tau_{ji}|\tau_j^I) = \delta(\tau_{ji}-\tau_j^I)\Psi_I(\tau_j^I)\left[\frac{\langle k\rangle - \Psi_R(\tau_j^I)}{\langle k\rangle}\right] + \int_0^{\tau_j^I} \delta(\tau_{ji}-(\tau_j^I-\tau))\left[1-\Psi_R(\tau_{ji})\right]\Psi_I(\tau_j^I-\tau)\sum_{n=1}^{\infty}P_n(\tau)d\tau \quad (20)$$

where the first term accounts for the case in which there are no infection attempts from j to i, $n=0$, while the second term accounts for the case $n>0$. By inserting Eq. (13) into Eq. (19) and integrating over τ_j^I, the probability $\psi(\tau_{ji})$ reads

$$\psi(\tau_{ji}) = \tilde{\delta}\Psi_I(\tau_{ji})\left\{\Psi_R(\tau_{ji})\left[\frac{\langle k\rangle - \Psi_R(\tau_{ji})}{\langle k\rangle}\right] + \left[1-\Psi_R(\tau_{ji})\right]\int_0^{\infty}\Psi_R(\tau_{ji}+\tau)\sum_{n=1}^{\infty}P_n(\tau)d\tau\right\}. \quad (21)$$

If we restrict to the case of Markovian recovery, we can use its memoryless property, $\Psi_R(\tau_{ji}+\tau)=\Psi_R(\tau_{ji})\Psi_R(\tau)$. By using the convenient Laplacian form of the probability distribution $P_n(\tau)$, one can obtain

$$\psi(\tau_{ji}) = \tilde{\delta}\frac{\Psi_I(\tau_{ji})\Psi_R(\tau_{ji})}{1-\widehat{\psi}_I(\tilde{\delta})}\left\{1-\Psi_R(\tau_{ji})\left[\langle k\rangle^{-1}\left(1-\widehat{\psi}_I(\tilde{\delta})\right)+\widehat{\psi}_I(\tilde{\delta})\right]\right\}. \quad (22)$$

The normalization of $\psi(\tau_{ji})$ reads

$$\mathcal{N} = \int_0^{\infty}\psi(\tau_{ji})d\tau_{ji} = \frac{\tilde{\delta}}{1-\widehat{\psi}_I(\tilde{\delta})}\left\{\widehat{\Psi}_I(\tilde{\delta}) - \widehat{\Psi}_I(2\tilde{\delta})\left[\langle k\rangle^{-1}\left(1-\widehat{\psi}_I(\tilde{\delta})\right)+\widehat{\psi}_I(\tilde{\delta})\right]\right\}, \quad (23)$$

therefore, by inserting Eq. (22) and Eq. (23) into Eq. (11) of the main text, one finally obtains the approximate infection rate λ_{app} presented in Eq. (12) of the main text.

In Fig. 2, we show the steady-state prevalence ρ^{st} as a function of the approximate effective infection rate λ_{app}, given by Eq. (12) of the main text, and the mean field effective rate λ_{mf} given by Eq. (17) for two extreme values of the exponent α_I controlling the interevent time infection distribution, $\alpha_I=0.25$ and $\alpha_I=10$. One can see that the curves for λ_{mf} do not collapse onto one another, especially for the lattice and SF network substrate.

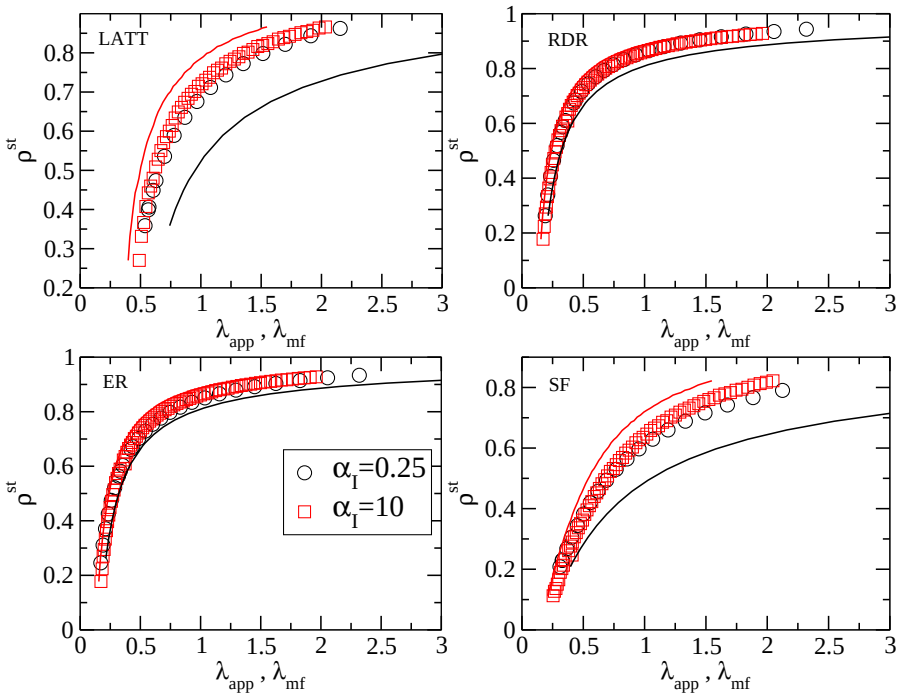

Figure 2: Prevalence ρ as a function of the approximate effective infection rate λ_{app} (points) and the mean field effective rate λ_{mf} (continuous line), for different values of the exponent α_I and different network substrate.

NUMERICAL SIMULATIONS OF THE NON-MARKOVIAN SIS DYNAMICS

To check the validity of the effective infection rates λ_{eff} and λ_{app}, we run extensive numerical simulations of the non-Markovian SIS dynamics. For each value of the average infection time $\langle t_I \rangle$ and fixed average recovery time $\langle t_R \rangle = \tilde{\delta}^{-1} = 1$, we simulate the non-Markovian SIS dynamics by implementing an algorithm based on a queue of infection and recovery events.

At time $t = 0$, all nodes are in a susceptible state, and a set of fN randomly chosen nodes, with $f = 0.5$, change their state to the infected one. In the algorithm, whenever a node i changes his state from susceptible to infected at time t, he first randomly extracts his recovery time t_R from the distribution $\psi_R(t)$, and pushes his recovery event at time $t + t_R$ to the queue. He also starts k independent infection processes to his k neighbors. In each infection process to a neighbor j, an infection event from node i to node j is scheduled at time $t + t_I^1$, where t_I^1 is randomly extracted from the distribution $\psi_I(t)$, only if $t_I^1 < t_R$, that is if node i is still infected at time $t + t_I^1$. A second infection event from node i to node j is scheduled at time $t + t_I^1 + t_I^2$, where t_I^2 is randomly extracted from the distribution $\psi_I(t)$, only if $t_I^1 + t_I^2 < t_R$, and so on until n (with possibly $n = 0$) infection events are generated and pushed to the queue.

The queue is pulled by following the time order of the events. If the pulled event is the recovery of node i, i changes his state from infected to susceptible. If the pulled event is an infection event from node i to node j, and j is already in a infected state, nothing happens, otherwise node j changes his state from susceptible to infected and schedules his recovery and infection events, pushing them to the queue. The queue is pulled until either no more events are left (and so all nodes are susceptible) or the time reaches a time T_{max}, set conveniently. In order to measure the prevalence in the steady state ρ^{st} and the effective infection rate λ_{eff}, we sample $N_s = 10^4$ time instants uniformly chosen in $[T_{min}, T_{max}]$, with T_{min} chosen such that the stationary state is reached long before it. For each time instant, we measure the prevalence and the values of τ_{ij} for each active link between nodes i and j, so as to calculate λ_{eff} by means of Eq. (10) of the main text.

We have double checked our event queue algorithm by simulating the non-Markovian SIS dynamics with a non-Markovian Gillespie algorithm [2], which is much slower, and we obtained identical results for the prevalence and the effective infection rate.

EPIDEMIC THRESHOLD AND CRITICAL EXPONENTS

We run extensive numerical simulations of the non-Markovian SIS dynamics in order to evaluate its behavior close to the epidemic threshold. We consider $\alpha_I = 0.5$ and $\alpha_I = 2$, and two different network substrates, 2D lattice and RDR network. We address the critical properties by means of the lifespan method [3], in which the infection starts with a single infected node. In the lifespan method, each realization is characterized by its lifetime, T, and its coverage, C, defined as the number of distinct nodes that have become infected at least once. We let each realization run until either the coverage C reaches a certain threshold C^* (and we consider it endemic), or the realization dies out, and we measure its lifetime T. We set $C^* = \Theta N$, with $\Theta = 0.9$. We then measure the probability of having an endemic realization P, the average lifetime $\langle T \rangle$ and average square lifetime $\langle T^2 \rangle$ over a number of runs N_{run}, as a function of the average infection time $\langle t_I \rangle$ (corresponding to an effective rate λ_{app}, hereafter λ for brevity) close to the epidemic threshold, for different sizes N. We set $N_{run} = 10^5$ for lattice, $N_{run} = 10^6$ for RDR networks. For each value of N, λ, α_I and network substrate we fit the curves of $\langle T \rangle$ and $\langle T^2 \rangle$ to find the peaks $\langle T \rangle_p$ and $\langle T^2 \rangle_p$ and their corresponding values of λ_p^1 and λ_p^2. We set λ_p as the average of λ_p^1 and λ_p^2, provided that λ_p falls within the λ_p^1 and λ_p^2 standard errors. The corresponding endemic probability at the peak P_p is interpolated from the data.

The set of equations we used to evaluate the critical point λ_c and critical exponents $\beta, \delta, \nu_\perp$ are

$$P(\lambda_c, N) \sim N^{-\beta/\nu_\perp} \tag{24}$$

$$P_p(N) \sim N^{-\beta/\nu_\perp} \tag{25}$$

$$|\lambda_c - \lambda_p(N)| \sim N^{1/\nu_\perp}. \tag{26}$$

$$\langle T^n \rangle_p(N) \sim N^{\gamma_n/\nu_\perp} \tag{27}$$

$$\tag{28}$$

We first evaluate the critical threshold λ_c by plotting the endemic probability $P(\lambda, N)$ as a function of N, for several values of λ close to λ_c, see the first row of Fig. 3. Through Eq (24), we estimate the value of λ_c to be the one which produces the best fit of $P(\lambda_c, N)$ as a power-law. We then plot the endemic probability at the peak P_p as a function of the size N, see the second row of Fig. 3, and the difference $|\lambda_c - \lambda_p|$ as a function of N, see the third row of Fig. 3. We estimate $\nu_\perp$ by means of Eq (26). By means of Equations (24) and (25) we estimate $\beta/\nu_\perp$ equal to the average of the fits of $P(\lambda_c, N)$ and $P_p(N)$, provided that $\beta/\nu_\perp$ falls within the standard errors of the two fits, and so we calculate β, knowing the value of $\nu_\perp$. Finally, we plot the height of the peaks $\langle T \rangle_p$ and $\langle T^2 \rangle_p$ as a function of N, see the fourth row of Fig. 3. We estimate γ_2 and γ_1 for lattices and γ_2 for RDR networks by means of Eq (27), knowing the value of $\nu_\perp$. For lattices, we calculate δ by means of the equivalence $\gamma_n \sim n - \delta$ while for RDR networks we first check that $\langle T \rangle_p$ diverges logarithmically as a function of N and then we calculate δ by means of the equivalence $\gamma_n = n - \delta$. The results are reported in Table 1 of the main text.

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[1] E. Cator, R. van de Bovenkamp, and P. Van Mieghem, Physical Review E **87**, 062816 (2013).
[2] M. Boguñá, L. F. Lafuerza, R. Toral, and M. A. Serrano, Phys. Rev. E **90**, 042108 (2014).
[3] M. Boguñá, C. Castellano, and R. Pastor-Satorras, Phys. Rev. Lett. **111**, 068701 (2013).

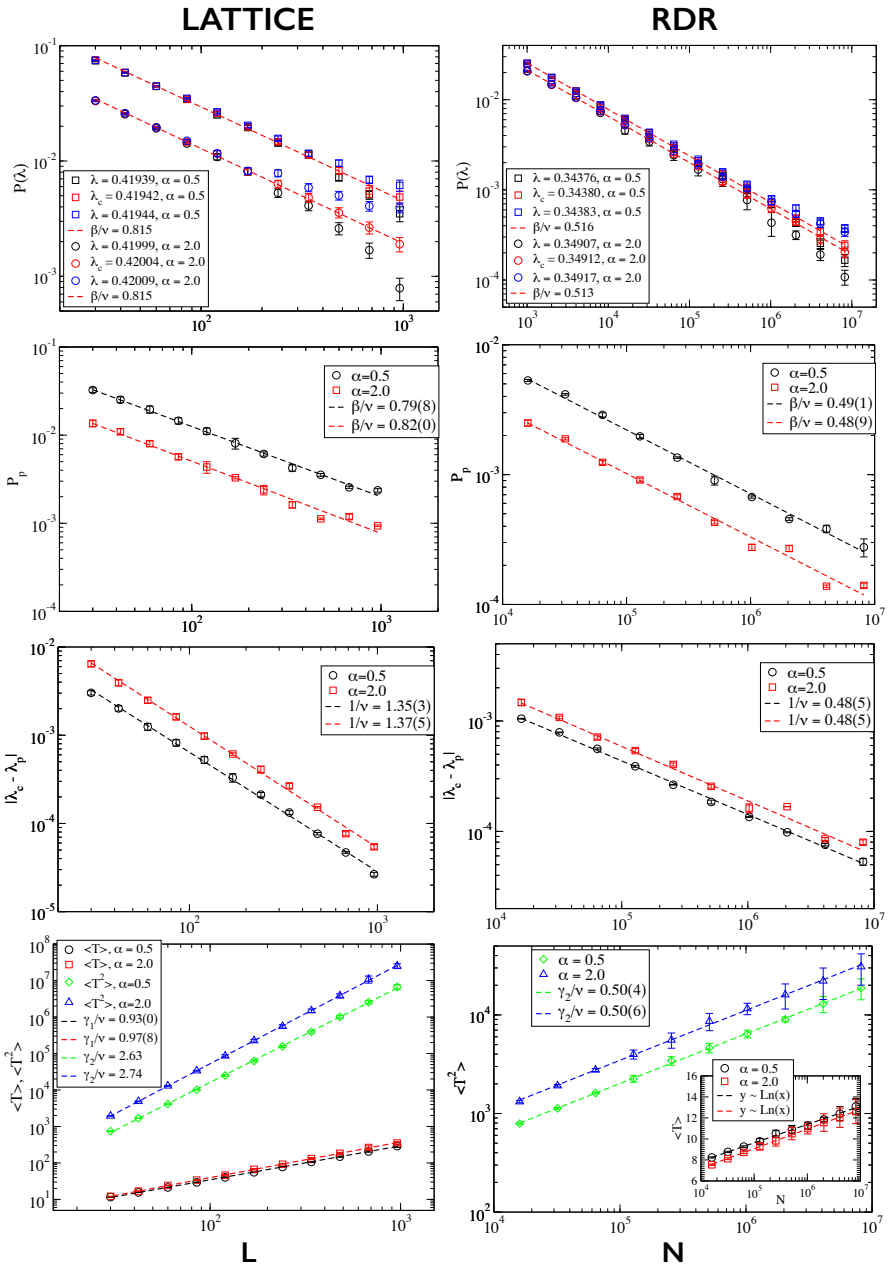

Figure 3: Finite size scaling of a non-Markovian SIS dynamics with $\alpha_I = 0.5$ and $\alpha_I = 2$, on 2D lattice (on the left) and RDR network (on the right). Symbols represent the results of numerical simulations, dashed lines represent power-law (or logarithmic, in the case of $\langle T \rangle$ for RDR) fits. In this Figure, we refer to λ_{app} as to λ for brevity. Notice that, to compare with the values found in the literature, in the case of the lattice we use the side of the lattice L instead of the number of nodes $N = L^2$. Plots show, from first to last row: (1) Probability that an outbreak is endemic for different values of λ, $P(\lambda)$, as a function of the size N. (2) Probability that an outbreak is endemic, $P(\lambda_p)$, as a function of the size N, for λ_p corresponding to the peak of $\langle T^2 \rangle$. (3) Difference $|\lambda_c - \lambda_p|$ as a function of N, for λ_p corresponding to the peak of $\langle T^2 \rangle$. (4) Peak of $\langle T \rangle$ and $\langle T^2 \rangle$ as a function of N.