A continuous-time discrete-distribution theory for activity-driven networks

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Our understanding of epidemic spreading has been deeply improved by the study of time-varying networks. Activity-driven networks (ADNs) are a very powerful paradigm to study these networks and, in particular, to model the co-evolution of the network of contacts and the spreading dynamics [4, 5, 9]. Despite significant advances, most of the studies on ADNs are based on extensive Monte Carlo simulations, and analytical results are only limited to linearized mean-field approximations that allow to obtain precise results on the epidemic threshold [3, 5, 7], revealing the presence of a phase transition between a condition in which the epidemic dies out fast and another where the epidemic spreads in the population, becoming endemic. However, the available analytical results are not able to forecast the dynamic evolution of the spreading, nor to estimate its endemic equilibrium. The latter is a metastable equilibrium point of the epidemic process with a constant (non zero) fraction of infected individuals.

Here, we propose an analytical framework that enables the study of the entire dynamics of the epidemic spreading at the population level. Differently from the original ADN formulation, where a discrete-time epidemic model is implemented with a continuous probability distribution for the nodes' activities, here we consider a continuous-time model with a discrete distribution. This change of perspective leads to a rigorous analytical treatment, which is also not prone to the confounds associated with the selection of the time step [6]. Our theory relies on a reduced number of parameters than traditional ADNs, making robust parameter identification from real-world data more robust and less prone to overfitting [8].

We consider a (large) population of n individuals, each one labeled with a natural number up to n and associated with a node of a time-varying graph $G(t) = (V, E(t))$, with $t \in \mathbb{R}^+$. We focus on a susceptible-infected-susceptible (SIS) process [1], being the simplest and most studied among the epidemic models [5]. Each node $v \in V$ is given a time-invariant activity rate $a_v \in \mathbb{R}^+$. Node v becomes active after a time sampled from an exponentially distributed random variable with rate a_v and contacts a node uniformly at random in V, generating an instantaneous undirected edge through which the epidemic spreads with fixed probability λ, if one of the two nodes is infected and

[5] Our theory can be easily extended to tackle more complex epidemic models (SIR, SEIS, etc.).

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the other one is susceptible. Infected nodes recover after a random time generated from an exponentially distributed random variable with parameter μ.

We propose a discrete power-law activity distribution in which individuals are partitioned into k equidistant activation classes $(a_1 < \cdots < a_k)$. We denote with n_i the number of nodes in the i-th class and we let $n_i \propto a_i^{-\gamma}$, where real-world observations [5] bound $\gamma \in [2,3]$. Due to central limit theorem, for $n \to \infty$, the fraction of nodes in each class converges to $\eta_i \propto a_i^{-\gamma}$, independently on n.

In the hydrodynamic limit $n \to \infty$, our formulation leads to a deterministic approximation of the dynamics, exponentially close to the original stochastic process [2]. In this approximation we model the evolution of the fraction of infected nodes ζ_i in each activation class through the following system of k ordinary differential equations (ODEs):

$$\dot{\zeta}_i = -\mu\zeta_i + \lambda(1-\zeta_i)(a_i x_1 + x_2), \tag{1}$$

where $x_1 = \sum \eta_h \zeta_h$ and $x_2 = \sum \eta_h a_h \zeta_h$. Notice that the macroscopic variable x_1 represents the fraction of infected individuals across all classes, while x_2 takes into consideration the activity rates of the infected nodes.

The main observable in the study of epidemics is x_1, whereas in real-world situations, the fraction of infected nodes in each class described by (1) cannot be observed. Therefore, being initial conditions for each class not available, system (1) cannot be directly applied to predict the evolution of epidemics. For this reason and to facilitate the mathematical treatment of (1), we rewrite it using exactly k ODEs in terms of macroscopic variables x_1, x_2 and, in general, $x_j = \sum \eta_h a_h^{j-1} \zeta_h$, where $j = 1, \ldots, k$.

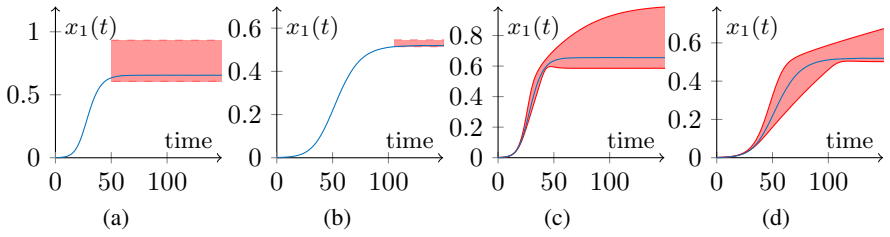

Fig. 1: Averaged Monte Carlo simulations of the epidemic process (blue) and theoretical bounds (red) from our low-dimensional continuous approximation. Time unit is a day. In (a) and (b), $k^* = 1$ is set to predict bounds on the endemic equilibrium. In (c) and (d), $k^* = 2$ is set to predict bounds on the transient evolutions. Simulations are averaged over 200 trials. The power-law exponent of the activity rate distribution is set as $\gamma = 2.2$ in (a) and (c) and $\gamma = 2.8$ in (b) and (d). Other model parameters are set as $n = 5000$, $\lambda = 0.3$, $\mu = 0.08$.

Such approach involves some issues in the computation of the quantities x_j's, for large values of j. In fact, on the one hand, it is practically difficult to compute initial conditions for such quantities. On the other hand, because of the power-law distribution of

the activation rates a_i, the quantities x_j's tend to blow up, harming the numerical integration of the ODEs. To overcome this limitation we consider a low-dimensional system of ordinary differential inclusions, comprising $k^* - 1 << k$ equations and one inclusion, using the following natural bounds on x_{k^*+1}: $a_1 x_{k^*} \leq x_{k^*+1} \leq a_k x_{k^*}$ and $x_{k^*+1} \leq \alpha_{k^*}$, where $\alpha_j = \sum \eta_h a_h^j$ is the j-th moment of the power-law distribution of the activation rates. The cases with $k^* = 1$ and $k^* = 2$ lead to very interesting results, illustrated in Fig. 1. Notably, using $k^* = 1$, two bounds for the fraction of infected nodes in the endemic equilibrium can be obtained, as illustrated in Figs. 1 (a)-(b). On the other hand, $k^* = 2$ leads to the definition of two bounds where the fraction of infected nodes x_1 during the transient evolution is confined, as illustrated in Figs. 1 (c)-(d). A sensible combination of these bounds can lead to predictions of the whole dynamics of the fraction of infected nodes x_1, from the inception of the epidemic spreading to its endemic equilibrium.

Predictions obtained using our approach can be drastically improved if a finite-time horizon is considered. Results are not shown here due to space constraints.

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