Effect of self-excitement and behavioral factors on epidemics on activity driven networks

Effect of self-excitement and behavioral factors on epidemics on activity driven networks

Lorenzo Zino, Alessandro Rizzo, and Maurizio Porfiri

***Abstract*— In this paper, we deal with the problem of including real-world phenomena into a mathematically tractable framework for the spread of epidemics on time-varying networks. Specifically, we consider individual behavioral modifications of the node dynamics due to self-excitement mechanisms and activity reduction due to infection. We develop our model within the framework of activity driven networks, which have recently emerged as a powerful tool to study the co-evolution of a network and of a spreading processes on it. First, we present a recent model extension that allows for the inclusion of self-excitement mechanisms. Then, we extend the model by including activity reduction due to infection, and we study its effect on the network propensity to epidemic outbreaks. We determine that, depending on the relative strength of the two concurrent mechanisms (self-excitement and activity reduction due to infection), the network may favor or hinder the spread of an epidemic disease. We analytically characterize these two situations, depending on the model and network parameters. Numerical simulations are provided to support and extend our analytical findings.**

I. Introduction

The last decade has witnessed a dramatic advancement in the capability of collecting and processing large amount of data on real-world social systems. The analysis of these data has increased our understanding of the characteristics of interaction patterns between individuals in social communities, revealing the mechanisms behind their formation. One important observation that arises from many empirical studies is that the network of interactions typically evolves in time, as each individual dynamically generates and modifies his/her connections to the others [1], [2], [3], [4]. Time-varying features have also been revealed in the propensity of individuals to form connections, which often exhibit bursty patterns [5], thereby yielding temporal connections to cluster [6], [7], [8], [9].

Empirical evidence suggests that the link generation dynamics and the individual propensity to create links with others are intertwined through self-excitement mechanisms [10], [11], [12]. In fact, it has been observed that the more one individual is active in creating links and connections with others, the more he/she is motivated to further increase his/her social activity and, consequently, the propensity to connect to others. Hence, self-excitement contributes to the emergence of burstiness and temporal clustering of social interactions [11], [13], [14].

Besides self-excitement, individuals often tend to modify their propensity to connect to others as a consequence of their own dynamics. For example, infected individuals tend not to leave their houses, thereby reducing their ability to contact other individuals. This behavior can be even enforced by administrations, for example through quarantine and hospitalization [15].

Activity driven networks (ADNs) have emerged as a valuable framework to model the time-varying nature of the network of interactions of real-world systems [16]. In their original formulation, the temporal evolution of the connections between individuals in the system is modeled via a constant parameter, called activity. The activity of an individual represents his/her propensity to generate transitory connections with others. Then, the pattern of connections is generated in a probabilistic framework. This formalism has enabled the scientific community to understand how time-varying interactions influence the dynamics on networks unveiling, for instance, the effect of heterogeneity in the individuals' activity on epidemics spreading [17], [18], [19] and on the diffusion of innovation [20]. From their original formulation, many features have been included into the ADN framework, allowing for the representation of real-world phenomena, such as the presence of memory in the link generation mechanism [21], [22] and the changes in the individuals' activity caused by the co-evolution of the network and the spreading processes on it [23], [24], [25].

Here, we include a self-excitement mechanism in the node dynamics within the ADN paradigm by means of Hawkes processes [26]. Toward a more realistic modeling of social phenomena, Hawkes processes replace time-homogeneous activation of the original incarnation of ADNs [16], [19] with a bursty mechanism. Hawkes processes preserve the Markov property of ADNs and, consequently, allow for analytical tractability and for the use of the Gillespie algorithm for fast numerical estimations with Monte Carlo simulations [27]. Other significant attempts to include bursty behaviors in ADNs are based on the modification of the inter-event time distribution [28], [29], [30]. Even though these models allow for setting the level of burstiness in the system, the analytical tractability is hindered by the lack of the Markov

L. Zino is with the "G.L. Lagrange" Department of Mathematics, Politecnico di Torino, 10129 Torino, Italy lorenzo.zino@polito.it

A. Rizzo is with the Department of Electronics and Telecommunications, Politecnico di Torino, Torino, Italy. He is also with the Office of Innovation, New York University Tandon School of Engineering, Brooklyn NY 11201, USA, alessandro.rizzo@polito.it

M. Porfiri is with the Department of Mechanical and Aerospace Engineering and the Department of Biomedical Engineering, New York University Tandon School of Engineering, Brooklyn NY 11201, USA, mporfiri@nyu.edu

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property. Our model, based on the combination of ADNs and Hawkes processes (ADN+HP), has been preliminary proposed in [31]. Therein, some analytical properties of the model have been studied, including the asymptotic distribution of the activities and the effect of self-excitement on the epidemic threshold of a Susceptible–Infected–Susceptible (SIS) model embedded on the temporal network. Specifically, it has been shown that self-excitement tends to decrease the epidemic threshold, favoring the inception of the epidemics.

In this paper, we extend the analysis of the effect of self-excitement in the spread of epidemics by investigating the concurrent effect of self-excitement due to activation and activity reduction of infected individuals. Our study unveils the key role of the superimposition of the two mechanisms on the epidemic threshold of the SIS model. Specifically, our main result consists of the analytical characterization of two regions of the parameter space: one where the effect of self-excitement overpowers the activity reduction due to infection, and the network increases its propensity to the inception of an outbreak; the other, in a distinct region, where the activity reduction is the dominant phenomenon and the network is less prone to the spread of the disease. Then, by means of Monte Carlo simulations, we investigate the effect of the duration of the incubation period of the disease on the network propensity to the inception of an outbreak. Our numerical results suggest that, for diseases with long incubation periods, the effect of the activity reduction due to infection tends to vanish.

The rest of the paper is organized as follows. In Section II, we present the mathematical preliminaries on Hawkes processes. In Section III, we present the mathematical model. Section IV summarizes our results for the SIS epidemic model. In Section V, we introduce and study the SIS model in the presence of activity reduction due to infection. Section VI concludes the paper and outlines our future research.

II. Mathematical preliminaries

A. Notation

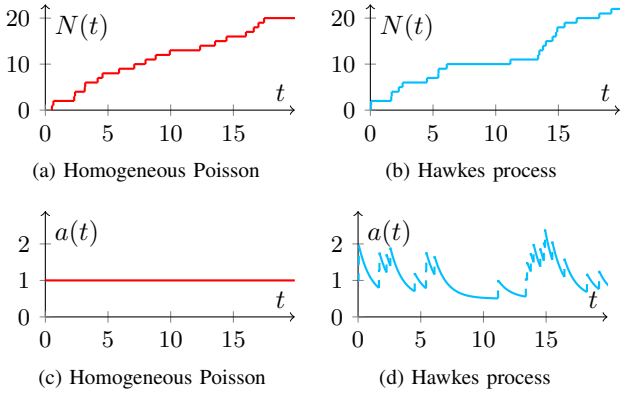
We gather here the notation used throughout this paper. We denote by $\mathbb{R}_{\geq 0} := \{x \in \mathbb{R} : x \geq 0\}$ ($\mathbb{R}_{>0} := \{x \in \mathbb{R} : x > 0\}$) the set of nonnegavive (strictly positive) real numbers and by $\mathbb{N}$ the set of nonnegative integer numbers. Given a random variable X, $\mathbb{E}[X]$ denotes its expected value.

B. Hawkes processes

Point processes are a family of stochastic processes, often used to model the occurrences of events in a probabilistic framework [32]. They can be conveniently represented by means of their counting process $N(t) \in \mathbb{N}$, which counts the number of occurrences of the corresponding event up to time t. Poisson processes are the most used point processes. In the following, we briefly recall their formal definition.

Definition 1: Given a locally integrable nonnegative function $a(t)$ called *intensity*, for any $\Delta t \in \mathbb{R}_{>0}$, a *Poisson point process* verifies $N(0) = 0$, and

$$\mathbb{P}[N(t+\Delta t) - N(t) = 1] = \int_t^{t+\Delta t} a(s)ds + o(\Delta t). \quad (1)$$



(a) Homogeneous Poisson (b) Hawkes process (c) Homogeneous Poisson (d) Hawkes process

Fig. 1: Comparison between sample path (a) and intensity (c) of a homogeneous Poisson process, and the corresponding quantities (b), (d) in a Hawkes process. The intensity of the Hawkes process presents a bursty behavior, and its events tend to cluster in correspondence of the peaks of the intensity.

Homogeneous Poisson processes where $a(t) = a \in \mathbb{R}_{>0}$ is constant for any $t \in \mathbb{R}_{\geq 0}$, are often used in the literature [19]. Here, instead, we consider Hawkes processes [26], which are a class of nonhomogeneous Poisson point processes, characterized by a time-varying intensity $a(t)$ that is ruled by two contrasting dynamics: a self-excitement and a forgetting mechanism. We define a Hawkes process as follows.

Definition 2: Given three parameters $J \in \mathbb{R}_{\geq 0}$, $\hat{a}, \gamma \in \mathbb{R}_{>0}$, a *Hawkes process* is a Poisson point process whose intensity $a(t)$ follows the stochastic differential equation [33]

$$da = \gamma\,(\hat{a} - a)\,dt + J dN. \quad (2)$$

Three parameters define a Hawkes process: i) the *jump* $J \in \mathbb{R}_{\geq 0}$, which measures the increase of the intensity after each occurrence; ii) the *forgetting rate* $\gamma \in \mathbb{R}_{>0}$, which determines the velocity of the forgetting process; and iii) the *background intensity* $\hat{a} \in \mathbb{R}_{>0}$, which is the (asymptotic) intensity of the process in the absence of self-excitement. It has been proved that Hawkes processes satisfy the Markov property [26], [34]. In Fig. 1, we compare the evolution of the counting process and the intensity of a homogeneous Poisson process (red) and a Hawkes process (light blue). The events in the sample path of the Hawkes process tend to cluster, differently from the ones of the homogeneous process. This evidence suggests that Hawkes processes could be used to model the occurrences of events that presents burstiness and temporal clustering, as studied in [5], [6], [7], [8], [9].

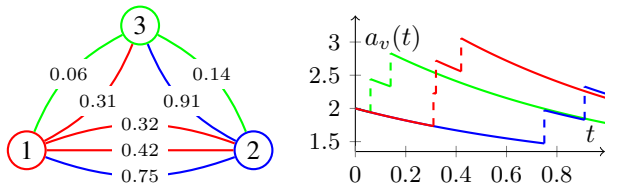
We conclude this preliminary section by reporting an important result on the asymptotic behavior of Hawkes process, whose proof can be found in [34].

Lemma 1: Let $(N(t), a(t))$ be a Hawkes process such that $J/\gamma < 1$. Then, $a(t)$ converges to an asymptotic invariant distribution $a(\infty)$ with

$$\mathbb{E}[a(\infty)] = \frac{\hat{a}}{1 - J/\gamma}, \quad \mathbb{E}[a^2(\infty)] = \frac{\hat{a}^2 + \dfrac{J^2\hat{a}}{2\gamma}}{(1 - J/\gamma)^2}. \quad (3)$$

III. Model

Each individual is identified by a node on a time-varying graph $\mathcal{G} = (\mathcal{V}, \mathcal{E}(t))$, where $\mathcal{V} = \{1, \ldots, n\}$ is the node set and $\mathcal{E}(t)$ is a time-varying link set. Links are generated



(a) Links and time-stamps (b) Nodes' activity

Fig. 2: Simulation of an ADN+HP with $n = 3$ nodes for $T = 1$. In (a), the occurrences of links are plotted separately, along with their time-stamps. The color of the link denotes the node that has generated it. In (b), the evolution of the activities of the three nodes is represented. Parameters are $J = 0.5$, $\gamma = 1$, $\hat{a}_v = 1$, and $a_v(0) = 2$, $\forall\, v \in \mathcal{V}$.

according to a stochastic mechanism, detailed in the following. Each node $v \in \mathcal{V}$ is associated with a Hawkes process $(N_v(t), a_v(t))$, each one independent of the others. For the sake of simplicity, we assume that all the Hawkes processes have the same jump $J \in \mathbb{R}_{\geq 0}$ and the same forgetting rate $\gamma \in \mathbb{R}_{>0}$. In [31], a scenario in which this assumption is removed is investigated. The background activity $\hat{a} \in \mathbb{R}^n_{>0}$ can differ from one node to another, allowing for modeling the system's heterogeneity. When the process $N_v(t)$ has a jump, then node v activates and generates a temporary link. We summarize the ADN+HP network generation algorithm as follows:

1) at time $t = 0$, we set $\mathcal{E}(t) = \emptyset$ and, for each node $v \in \mathcal{V}$, an independent Hawkes process $(N_v(t), a_v(t))$ is initialized with given initial intensity $a_v(0) \in \mathbb{R}_{\geq 0}$;
2) if the Hawkes process associated with node v has a jump at time t, then node v activates and generates an instantaneous undirected link by connecting to a node in $w \in \mathcal{V}$ chosen uniformly at random[1]. The undirected link $\{v, w\}$ is added to $\mathcal{E}(t)$; and
3) the instantaneous link is immediately removed from the link set, and the procedure is resumed to step 2).

Figure 2 shows a realization of an ADN+HP, depicting the temporal evolution of the nodes' activity. To sum up, the ADN+HP model is defined by: i) the *node set* $\mathcal{V} = \{1, \dots, n\}$; ii) the jump $J \in \mathbb{R}_{\geq 0}$ and iii) the *forgetting rate* $\gamma \in \mathbb{R}_{>0}$ of the Hawkes processes; iv) the *initial activity* vector $a(0) \in \mathbb{R}^n_{>0}$; and v) the *background activity* vector $\hat{a} \in \mathbb{R}^n_{>0}$. In this paper, we will make the following assumption, which allows us to use Lemma 1.

Assumption 1: The Hawkes processes have $J < \gamma$.

Remark 1: In the limit case with $J = 0$ and $a(0) = \hat{a}$, the ADN+HP model reduces to the standard continuous-time ADN model [19]. Therefore, the ADN+HP model encompasses and generalizes the original ADNs.

To study the evolution of the activity distribution, we define its moments as $M_{a^i(t)} := \frac{1}{n}\sum_{v\in\mathcal{V}} a_v^i(t)$, $i \in \mathbb{N}$. Then, we state the following result, whose proof is based on the Kolmogorov law of large numbers and on the application of Lemma 1. Details can be found in [31].

[1]Following this mechanism, self-loops may be generated. To avoid their presence, one may chose w from $\mathcal{V} \setminus \{v\}$.

Lemma 2: For $n \to \infty$, under Assumption 1, the first two moments of the activity distribution converge almost surely to

$$M_{a(\infty)} = \frac{M_{\hat{a}}}{1 - J/\gamma}, \quad M_{a^2(\infty)} = \frac{M_{\hat{a}^2} + \frac{J^2}{2\gamma} M_{\hat{a}}}{(1 - J/\gamma)^2}. \tag{4}$$

IV. SIS EPIDEMIC MODEL ON ADN+HP

Here, we study an epidemic outbreak on an ADN+HP using an SIS model. According to [19], we define the epidemic model as follows. Each individual $v \in \mathcal{V}$ is given a binary state $Y_v(t) \in \{0, 1\}$ to describe whether he/she is susceptible to the epidemic ($Y_v(t) = 0$) or infected ($Y_v(t) = 1$). The state of the nodes evolves according to two competing mechanisms: a *spreading* dynamics and a *recovery* process. When a link that connects a susceptible individual and an infected one is generated, the disease may propagate with a fixed probability $\lambda \in [0, 1]$. If it propagates, then both individuals become infected. Then, infected individuals recover according to independent homogeneous Poisson processes with intensity $\mu \in \mathbb{R}_{>0}$, becoming susceptible to the epidemic again.

This SIS model is characterized by the presence of a phase transition between a regime where the disease quickly extinguishes and one where it becomes endemic [35]. This phase transition occurs when the ratio between the two parameters, i.e., λ/μ, passes a specific value σ called *epidemic threshold*, which may depend on specific properties of the network of interactions among the individuals [36], [37]. In the thermodynamic limit, for $n \to \infty$, in the *fast extinction regime* all the trajectories of the process converge to the disease-free equilibrium, while in the *endemic regime*, those with initial condition different from the disease-free equilibrium do not converge to it for an exponentially long time, with high probability [37], [38].

The evolution of the system is governed by an n-dimensional Markov process on the state space $\{0, 1\}^{\mathcal{V}}$, whose size grows exponentially with n, hindering its analysis for large-scale systems. Following [39], [40], we consider a continuous relaxation of the dynamics in which, instead of the evolution of the individuals' healthy state, we study the evolution of the probability for each individual to be infected, denoted by $y_v(t) := \mathbb{E}[Y_v(t)]$, which is governed by the ODE

$$\dot{y}_v = -\mu y_v + (1 - y_v)\left(a_v(t)\frac{1}{n}\sum_{v\in\mathcal{V}} y_w + \frac{1}{n}\sum_{v\in\mathcal{V}} a_w(t) y_w\right). \tag{5}$$

The evolution of the probability that node $v \in \mathcal{V}$ is infected is governed by three terms. The first one is the contribution of the recovery process, which is negative. The other two terms, instead, are positive. One is the contribution of links to infected nodes generated by v, the other is the contribution of infected nodes that generate connections toward v.

We compute the epidemic threshold of the model by studying the local linear stability of the disease-free equilibrium for the continuous relaxation. We observe that, the evolution of the epidemic prevalence (i.e., the fraction of infected individuals) for the stochastic process can be approximated arbitrarily well using its continuous relaxation [41], [42].

Hence, the epidemic threshold computed using (5) is very accurate for large-scale systems, specifically from population sizes larger than 1000 individuals, as shown in the simulations in [40]. The stability analysis of (5), performed in [31], can be summarized in the following result.

Theorem 1: Under Assumption 1, the epidemic threshold of an SIS epidemic model on an ADN+HP is

$$\sigma_{HP} = \frac{1 - J/\gamma}{M_{\hat{a}} + \sqrt{M_{\hat{a}^2} + \frac{J^2}{2\gamma} M_{\hat{a}}}}. \tag{6}$$

Specifically, for $\sigma < \sigma_{HP}$, the system is in the fast extinction regime while, for $\sigma > \sigma_{HP}$, it is in the endemic regime.

Remark 2: In the absence of self-excitement, i.e., for $J = 0$, the epidemic threshold in (6) reduces to the one of an SIS on a standard ADNs [16].

The proof is based on the change of variables $x_i(t) = \frac{1}{n}\sum_{v\in\mathcal{V}} a_v^{i-1}(t) y_v(t)$, $i \in \mathbb{N}$, and on the stability analysis of the origin in the new set of variables, which reduces to the stability analysis of

$$\begin{cases} \dot{x}_1 &= (\lambda M_{a(\infty)} - \mu) x_1 + \lambda x_2, \\ \dot{x}_2 &= \lambda M_{a^2(\infty)} x_1 + (\lambda M_{a(\infty)} - \mu) x_2, \end{cases} \tag{7}$$

where $x_1(t)$ and $x_2(t)$ are the fraction of infected nodes, and the average activity of the infected individuals at time t, respectively. All the details can be found in [31].

To understand the effect of self-excitement on the network propensity to the inception of an epidemic outbreak, the threshold of the ADN+HP model in Theorem 1 should be compared with the one of the standard ADNs with time-invariant activity [16], [19]. To perform a fair comparison, for any $v \in \mathcal{V}$, we set the activity of the node in the ADN with time-invariant activity equal to $\hat{a}_v/(1 - J/\gamma)$, that is the expected asymptotic activity of the node in the ADN+HP model, so that corresponding nodes in the two models have the same expected activity. The epidemic threshold in the presence of self-excitement is reduced by a parameter

$$\frac{\sigma_{HP}}{\sigma_{ADN}} = \frac{M_{\hat{a}} + \sqrt{M_{\hat{a}^2}}}{M_{\hat{a}} + \sqrt{M_{\hat{a}^2} + \frac{J^2}{2\gamma} M_{\hat{a}}}} < 1. \tag{8}$$

Thus, ADN+HP has a higher propensity toward the inception of epidemic outbreaks than the corresponding standard ADN. The dependence on self-excitement is governed by the ratio J^2/γ: the larger this ratio, the more the epidemic threshold decreases, favoring the spread of the disease.

The following example stresses the importance of including self-excitement in epidemic models. Imagine that, after a few cases of a disease have been reported, we developed a mathematical model to predict its spread. If we adopted a standard ADN with time-invariant activities, thereby neglecting any self-excitement, we would overestimate the epidemic threshold, underestimating the danger of the outbreak, with potential catastrophic consequences for the whole population. Figure 3 illustrates this hypothetical scenario. The simulation of the SIS model on a standard ADN (red dotted curve) suggests a fast extinction of the disease, while the inclusion of self-excitement yields to the inception of an outbreak.

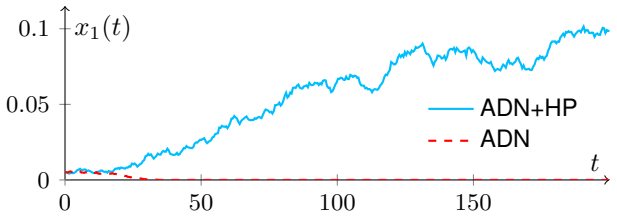


Fig. 3: Sample paths of an SIS model on an ADN+HP (cyan solid) and the corresponding ADN with time-invariant activity (red dashed). Model parameters are $n = 10000$, $\lambda = 0.43$, $\mu = 0.4$, $J = 0.4$, $\gamma = 0.8$, $\hat{a}_v = 0.225$, and $x_1(0) = 0.005$. The ADN+HP is in the endemic regime ($\lambda/\mu > \sigma_{HP} \approx 1.1054$), while the ADN with time-invariant activity is in the fast extinction one ($\lambda/\mu < \sigma \approx 1.1111$).

V. Behavioral changes due to infection

Besides the effects of self-excitement, individuals tend to modify their activity as a consequence of their health state. Here, we investigate the effect of the co-existence of this phenomenon and self-excitement on the propagation of an epidemic disease on a network. We assume that individuals reduce their activity because of the infection. Following [23], we model this phenomenon by introducing a parameter $\rho \in [0, 1]$ that measures the activity reduction of an infected individual. Hence, the activity of an infected node $v \in \mathcal{V}$ is reduced by the parameter ρ. Formally, the activations of node $v \in \mathcal{V}$ are still governed by a Hawkes process $(N_v(t), a_v(t))$ but, if the individual is infected and becomes active, then he/she generates a connection with probability ρ while, with probability $1 - \rho$, no links are generated. This probabilistic mechanism induces a split Poisson process [32]. We observe that the evolution of the processes $Y(t)$ depends only on the state of the process itself (since the activity reduction of $v \in \mathcal{V}$ depends on the state $Y_v(t)$), so Markov property is preserved. Therefore, the continuous relaxation adopted in Section IV to analyze the standard SIS model can be also used in this scenario, yielding an accurate approximation of its evolution in the thermodynamic limit [41], [42]. Hence, the epidemic threshold can be computed by studying the local linear stability of the origin for the system of ODEs

$$\dot{y}_v = -\mu y_v + (1 - y_v)\left(a_v(t)\frac{1}{n}\sum_{v\in\mathcal{V}} y_w + \frac{\rho}{n}\sum_{v\in\mathcal{V}} a_w(t) y_w\right), \tag{9}$$

for $v \in \mathcal{V}$. We observe that the only difference with respect to (5) is in the last term, which is the contribution to the disease propagation that comes from infected individuals that create connections to node v. This term is reduced by the parameter ρ as an effect of the activity reduction of infected nodes. Then, we state the following result.

Theorem 2: Under Assumption 1, the epidemic threshold of an SIS epidemic model on an ADN+HP with activity reduction due to infection with parameter $\rho \in [0, 1]$ is

$$\tilde{\sigma}_{HP} = \frac{1 - J/\gamma}{\frac{1+\rho}{2} M_{\hat{a}} + \sqrt{\frac{(1-\rho)^2}{4} M_{\hat{a}}^2 + \rho M_{\hat{a}^2} + \rho \frac{J^2}{2\gamma} M_{\hat{a}}}}. \tag{10}$$

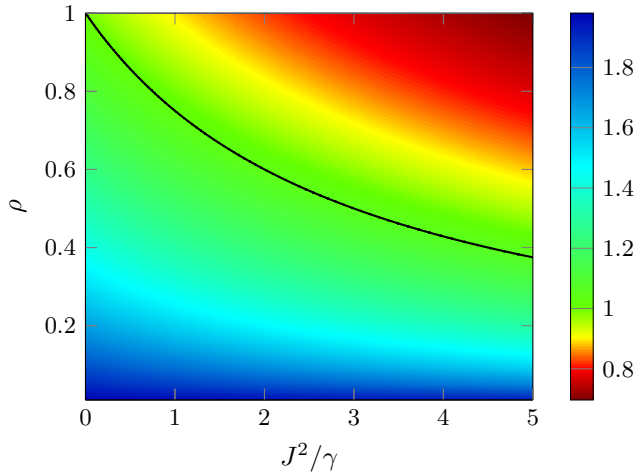


Fig. 4: Variation of the epidemic threshold in the presence of self-excitement and activity reduction due to infection, depending on J^2/γ and ρ. The black curve from (16) separates the region of the parameter space where the threshold increases (above) and decreases (below). Background activities are $\hat{a}_v = 1$, $\forall\, v \in \mathcal{V}$.

Proof: Following the same argument used to study the stability of the origin in the standard SIS model [31], we reduce the problem to the stability analysis of the system of equations that govern the evolution of x_1 and x_2, linearized about the origin, that is

$$\begin{cases} \dot{x}_1 &= (\lambda M_{a(\infty)} - \mu)x_1 + \lambda\rho x_2, \\ \dot{x}_2 &= \lambda M_{a^2(\infty)} x_1 + (\lambda\rho M_{a(\infty)} - \mu)x_2, \end{cases} \tag{11}$$

whose Jacobian matrix is

$$J = \begin{bmatrix} -\mu + \lambda\dfrac{M_a}{1 - J/\lambda} & \lambda\rho \\ \lambda\left(\dfrac{M_{\hat{a}^2} + \frac{J^2}{2\gamma}M_{\hat{a}}}{(1 - J/\gamma)^2}\right) & -\mu + \lambda\rho\dfrac{M_a}{1 - J/\lambda} \end{bmatrix}. \tag{12}$$

The eigenvalues of J are equal to

$$-\mu + \frac{1+\rho}{2}\lambda\frac{M_a}{1 - \frac{J}{\lambda}} \pm \lambda\sqrt{\frac{(1-\rho)^2}{4}M_{\hat{a}}^2 + \rho M_{\hat{a}^2} + \rho\frac{J^2}{2\gamma}M_{\hat{a}}}. \tag{13}$$

The largest one is negative if

$$\frac{\lambda}{\mu} < \frac{1 - J/\gamma}{\frac{1+\rho}{2}M_{\hat{a}} + \sqrt{\frac{(1-\rho)^2}{4}M_{\hat{a}}^2 + \rho M_{\hat{a}^2} + \rho\frac{J^2}{2\gamma}M_{\hat{a}}}}, \tag{14}$$

which provides the linear stability condition for the origin. ■

Remark 3: In the absence of activity reduction, i.e., for $\rho = 1$, the epidemic threshold in (10) coincides with (6).

The concurrent effect of self-excitement and activity reduction due to infection is studied by comparing the epidemic threshold in Theorem 2, with the one of a standard SIS on an ADN [16] with the nodes' activity equal to the expected asymptotic activity of the corresponding nodes in the ADN+HP model, i.e., $\hat{a}_v/(1 - J/\gamma)$, $\forall\, v \in \mathcal{V}$. We obtain

$$\frac{\tilde{\sigma}_{HP}}{\sigma_{ADN}} = \frac{M_{\hat{a}} + \sqrt{M_{\hat{a}^2}}}{\frac{1+\rho}{2}M_{\hat{a}} + \sqrt{\frac{(1-\rho)^2}{4}M_{\hat{a}}^2 + \rho M_{\hat{a}^2} + \rho\frac{J^2}{2\gamma}M_{\hat{a}}}}. \tag{15}$$

Differently from the scenario with only self-excitement, where the epidemic threshold for the SIS model on an ADN+HP is always reduced, the behavior of (15) cannot be deduced easily from its expression. Depending on the relative strength of the two concurrent mechanisms, the epidemic threshold may increase or decrease, unveiling a nontrivial relation between the model parameters, as shown in Fig. 4. From the analysis of (15), it can be proved that the reduction in the threshold caused by self-excitement overpowers its increase due to activity reduction of infected individuals if

$$\frac{J^2}{\gamma} \geq \frac{(1-\rho)}{\rho}\left[2\frac{M_{\hat{a}^2}}{M_{\hat{a}}} + \sqrt{M_{\hat{a}^2}}\right]. \tag{16}$$

In many epidemics, individuals are not immediately aware of having contracted the disease. In fact, there is often an incubation period that could last from a couple of days to several months or years, in which the individual has no symptoms, but he/she is already infectious. This is the case of measles, smallpox, and HIV. In this period, individuals still generate connections according to their usual activity, without any reduction. We conclude the section by investigating the effect of the duration of such an incubation period on the epidemic threshold by means of Monte Carlo simulations of the epidemic model in which we add a delay between the contraction of the epidemics and of the activity reduction. To estimate the epidemic threshold of the SIS model, we adopt the method proposed in [25]: the epidemic thresholds coincides with the value of λ/μ that maximizes the variance of the number of infected nodes after a fixed amount of time, over a set of Monte Carlo simulations. The results of our numerical simulations, shown in Fig. 5, support the intuition that, when the incubation period becomes longer, the effect of the activity reduction due to infection tends to diminish and, eventually, vanishes, decreasing the epidemic threshold toward the value computed in (6). The establishment of rigorous analytical results might be pursued by using an SEIS model [40] and will be part of our future research.

VI. Conclusion

In this paper, within the framework of ADNs, we have developed a model for epidemics on temporal networks that includes self-excitement mechanisms and activity reduction due to infection, which is amenable to rigorous analytical studies. We have unveiled a nontrivial behavior of the epidemic model, depending on the relative strength of the two mechanisms: if self-excitement overpowers the activity reduction, then the spread of the disease is favored and the network is more prone to the inception of epidemic outbreaks. In the opposite scenario, the system is less inclined to the spread of epidemics. We have analytically characterized these two regimes, depending on the model parameters, and carried out preliminary investigations on the role of a possible asymptomatic but infectious incubation period.

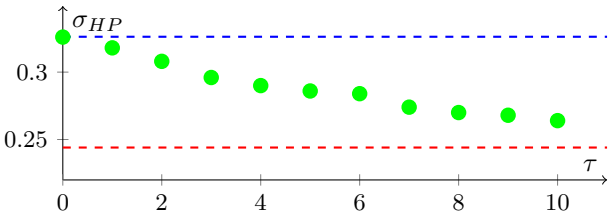


Fig. 5: Monte Carlo estimation (over 200 simulations) of the epidemic threshold of an SIS model with activity reduction due to infection and incubation period of duration τ on an ADN+HP for increasing values of τ. Model parameters are $n = 1000$, $J = 0.4$, $\gamma = 0.8$, $\mu = 0.14$, $\hat{a}_v = 1$, $\forall v \in \mathcal{V}$. The dashed horizontal lines is the epidemic threshold in the absence of activity reduction due to infection (red) and in its presence (blue) from Theorems 1 and 2, respectively. As the duration of the incubation period increases, the effect of activity reduction vanishes, and the epidemic threshold approaches the one of a standard SIS model on an ADN+HP.

In our future research, we will deepen the analysis of the effect of the incubation period, which in this paper has been studied by means of numerical simulations. We believe that the inclusion of real-world phenomena in analytically tractable epidemic models is a crucial point toward the development of techniques to produce accurate predictions of the evolution of a disease and, possibly, to control it.

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