

Thesis
Concurrency-induced transitions in epidemic
dynamics on temporal networks

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Publications

Some of the results from this thesis have appeared in the following peer reviewed publication.

- T. Onaga, J. P. Gleeson, and N. Masuda, “Concurrency-Induced Transitions in Epidemic Dynamics on Temporal Networks”, *Physical Review Letters* 119(10), 108301 (2017).

The following publications are published during the course of this thesis. As the work is not directly relevant to the thesis, they are not included in the main body of the thesis.

- T. Onaga and S. Shinomoto, “Bursting Transition in a Linear Self-Exciting Point Process”, *Physical Review E* 89(4), 042817 (2014).
- Y. Mochizuki, T. Onaga, et al., “Similarity in Neuronal Firing Regimes across Mammalian Species”, *Journal of Neuroscience* 36(21), 5736-5747 (2016).
- T. Onaga and S. Shinomoto, “Emergence of Event Cascades in Inhomogeneous Networks”, *Scientific Reports*, 6(1), 33321 (2016).

Contents

1	Introduction	7
1.1	Preliminaries for network science	8
1.1.1	Small-world networks	8
1.1.2	Scale-free networks	8
1.2	Epidemic spreading processes	8
1.2.1	Epidemic models	9
1.3	Analysis	9
1.3.1	Master equation	9
1.3.2	Mean-field approximation	10
1.3.3	Final states and epidemic transition	11
1.4	Epidemic dynamics on scale-free networks	12
1.5	Contents of the thesis	13
2	Epidemic dynamics on temporal networks	15
2.1	Temporal networks	15
2.2	The effect of concurrency on epidemic dynamics	17
2.2.1	Measures of concurrency	17
2.2.2	Previous studies of concurrency	19
3	Concurrency-induced transitions in epidemic dynamics on temporal networks	21
3.1	Model	21
3.2	Analysis	22
3.3	Discussion	26
4	Conclusion and Future Perspective	29
4.1	Conclusion	29
4.2	Future perspective	29
4.2.1	Concurrency	29
4.2.2	Social information spreading	30
4.2.3	Temporal networks	30

5	Appendix	33
5.1	Prevalence on the aggregate network	33
5.2	When the low-activity assumption is violated	33
5.3	Derivation of c_1 , c_2 , c_3 , c_4 , and c_5	34
5.4	Derivation of Eq. (3.8)	37
5.5	Convergence of the Maclaurin series	39
5.6	Epidemic threshold under the individual-based approximation . . .	40
5.7	Derivation of τ_* for general activity distributions	41
5.8	Derivation of m_c for general activity distributions	41
5.9	Activity-driven model with a reinforcement process	41
5.9.1	Stochastic m	42
5.10	Heterogeneous activity distributions	43
5.11	Temporal networks composed of cliques	44
5.12	Empirical activity distributions	46

Chapter 1

Introduction

A network is a system composed of a set of items, which we call nodes, with connections, which we call links. Such systems are ubiquitously found in various research fields. Some examples are social contact networks, friendships, networks of business relations between banks or companies, food webs, and neural networks [1, 2].

Networks had been studied in the form of graph theory since Euler proposed the solution of the Königsberg bridge problem in 1735. The study of graph theory focused on the analysis of single small graphs and the properties of individual vertices or edges. In recent years, researchers study networks from a different angle with focus on large-scale statistical properties and dynamics on networks [1, 3].

One of the dynamics on networks attracting researchers' attention is an epidemic spreading process [3]. The research of epidemic spreading by mathematical modeling was started by Daniel Bernoulli in 1760, has been studied for more than 200 years [4]. The standard models of epidemic spreading are susceptible-infected-susceptible (SIS) and susceptible-infected-recovered/removed (SIR) models proposed by Kermack and McKendrick [5] in 1927. The research has been developed by epidemiologists since then. Since 2000, with more and more available empirical data, the field is facing a golden age. At the same time, epidemic spreading has been also studied by network scientists using the characteristics of social contact networks, on which epidemic spreads. Recently, instead of a static-network approach, a temporal-network approach gathers researchers attention [6]. In this thesis, we focus on concurrency, which is a component of temporal networks, and report the effects of concurrency on epidemic spreading.

1.1 Preliminaries for network science

We introduce the basic concepts of network science.

1.1.1 Small-world networks

The research of coupled dynamical systems had been studied using regular networks (here we assume a regular ring lattice, in which each node is connected to k neighbors) or random networks, e.g. Erdős-Rényi model. However, many social, technological, and biological networks are characterized by high clustering coefficients, which is the ratio of the number of triangles to that of connected three nodes in the network, and small average path lengths, which is the average distance between nodes along links. Neither regular nor random networks can show both of them. The highly clustered networks with small average path length are called small-world networks. This indicates that regular networks and random networks are not a good model of the social contact networks. Small-world effect affects the dynamics on the network. For example, because the average time for disease to spread to the entire network is determined by the average path length, disease spreads faster in small-world networks than regular networks [7].

1.1.2 Scale-free networks

In many networks in the real-world (e.g., social contact networks, and internet), only a few nodes have numerous connections and others have a few connections. The number of connections of each node is called degree k . The distribution of degree k is called degree distribution $P(k)$. The heterogeneity among nodes is characterized by the long-tailed (power-law) degree distribution as opposed to the exponential decay of regular or random networks. As shown in 1.4, the long-tailed degree distributions also alter the dynamics on the network.

1.2 Epidemic spreading processes

The dynamics of spreading processes are studied in many fields. Some examples are epidemic spreading, opinion/information spreading, neuronal activity, and seismic activity. Here, we focus on one epidemic spreading processes. Note that the analytical method similar to that presented in this chapter can be used for other kinds of spreading processes with some assumptions on the network structure [8, 9], and that with some assumptions, the model is equivalent to models of neuronal and seismic activity [10].

1.2.1 Epidemic models

Here, we introduce the continuous-time susceptible-infected-susceptible (SIS) and susceptible-infected-recovered/removed (SIR) models, which is a variant of the model that Kermack and McKendrick introduced in 1927 [5].

SIS model

We suppose that people are classified into two categories: susceptible and infected categories. A susceptible person gets infected when he or she has contacts with other infected people at rate β per contact. An infected person recovers to susceptible person at rate μ . Here we assume that each person has connections with randomly chosen k people. Here, we assume that the dynamics is continuous, but we can also consider a discrete dynamics.

SIR model

We suppose that people are classified into three categories: susceptible, infected, and recovered/removed categories. A susceptible person gets infected when he or she has contacts with other infected people at rate β per contact. An infected person recovers and becomes a recovered person at rate μ .

1.3 Analysis

Now, we introduce the method to analyze SIS or SIR model. First, we derive the exact time-development equation. Second, we use a mean-field approximation to reduce the number of equations. Finally, we calculate the prevalence for SIS model and the final epidemic size for SIR model.

1.3.1 Master equation

SIS and SIR dynamics are continuous-time Markov processes. To analyzing SIS or SIR model exactly, we use a master equation. We denote the total number of people by N and the status of i -th person by $\sigma_i \in \{S, I, R\}$. The entire configuration of all people is represented by $\sigma_1 \sigma_2 \dots \sigma_N$. We obtain the transition rate from a state $\sigma_1 \sigma_2 \dots \sigma_N$ to another state $\sigma'_1 \sigma'_2 \dots \sigma'_N$:

$$A_{\{\sigma_1 \sigma_2 \dots \sigma_N\} \{\sigma'_1 \sigma'_2 \dots \sigma'_N\}} = \begin{cases} n\beta & (\sigma_i = S, \sigma'_i = I, \sigma_j = \sigma'_j \text{ for } j \neq i) \\ \mu & (\sigma_i = I, \sigma'_i = S, \sigma_j = \sigma'_j \text{ for } j \neq i) \\ 0 & (\text{otherwise}) \end{cases} \quad (1.1)$$

for SIS model and

$$A_{\{\sigma_1\sigma_2\ldots\sigma_N\}\{\sigma'_1\sigma'_2\ldots\sigma'_N\}} = \begin{cases} n_i\beta & (\sigma_i = S, \sigma'_i = I, \sigma_j = \sigma'_j \text{ for } j \neq i) \\ \mu & (\sigma_i = I, \sigma'_i = R, \sigma_j = \sigma'_j \text{ for } j \neq i) \\ 0 & (\text{otherwise}) \end{cases} \quad (1.2)$$

for SIR model, where n denotes the number of infected neighbor of i in states $\sigma_1\sigma_2\ldots\sigma_N$. Let P be the distribution of probability over states. The time-development of vector P is given by the transition rate matrix $\mathbf{A}$ as

$$\dot{P} = \mathbf{A}P. \quad (1.3)$$

Note that the disease-free state, in which $\sigma_i = S$ for all i , is an absorbing state.

1.3.2 Mean-field approximation

To analyze the dynamics further, we apply a mean-field approximation. We assume that the state of each person is independent and identical, yielding

$$\Pr(\sigma_1\sigma_2\ldots\sigma_N) \approx \prod_i \Pr(\sigma_i), \quad (1.4)$$

where

$$\Pr(\sigma_i = S) = s, \quad (1.5)$$

$$\Pr(\sigma_i = I) = 1 - s. \quad (1.6)$$

By substituting Eqs. (1.5) and (1.6), summing Eq. (1.3) over σ_j ($j \neq i$), and taking the limit as N goes to ∞ , we obtain the time-development equation for s :

$$\dot{s} = -\beta ks(1 - s) + \mu(1 - s). \quad (1.7)$$

Note that $1 - s$ is the fraction of infected nodes and called prevalence.

For SIR model, in stead of Eqs. (1.5) and (1.6), the distribution of the probability of each person over states is given by

$$\Pr(\sigma_i = S) = s, \quad (1.8)$$

$$\Pr(\sigma_i = I) = i, \quad (1.9)$$

$$\Pr(\sigma_i = R) = 1 - s - i. \quad (1.10)$$

The time-development equations are given by

$$\dot{s} = -\beta ksi, \quad (1.11)$$

$$\dot{i} = \beta ksi - \mu i. \quad (1.12)$$

1.3.3 Final states and epidemic transition

Here, we consider the final states of the dynamics using the above derived time-development equation. First, for SIS model, Eq. (1.7) has two stable solutions

$$s^* = 1, \frac{\mu}{\beta k}. \quad (1.13)$$

Because s is a probability, s must be less than 1. If $\mu > \beta k$, $s^* = 1$ is the only solution and stable. Otherwise, there are two solutions, and the solution $s^* = 1$ is unstable, while the other solution $s^* = \mu/(\beta k)$ is stable. Unless the initial state is the disease-free state, the final prevalence is given by

$$1 - \frac{\mu}{\beta k}. \quad (1.14)$$

Second, for SIR model, we derive the final states of the dynamics as follows. Let r be the fraction of recovered people:

$$r = 1 - s - i. \quad (1.15)$$

By changing variables from i to r , Eqs. (1.11) and (1.12) lead to

$$\dot{s} = \beta k s(1 - s - r) \quad (1.16)$$

$$\dot{r} = \mu(1 - s - r). \quad (1.17)$$

Thus, we obtain

$$\frac{\dot{s}}{s} = -\frac{\beta k \dot{r}}{\mu}. \quad (1.18)$$

By integrating over t , we obtain the final size of epidemic, i.e. the fraction of recovered people at $t \rightarrow \infty$, as the solution of a self-consistent equation

$$r^* = 1 - s_0 \exp\left(-\frac{\beta k r^*}{\mu}\right), \quad (1.19)$$

where s_0 denotes the initial fraction of susceptible people. We assume that s_0 is close to 1. Because r^* is a probability, r^* must be greater than or equal to 0. If $\mu > \beta k$, Eq.(1.19) has only one solution $r^* = 0$, while otherwise, it has a solution $r^* = 0$ and another solution $0 \leq r^* \leq 1$. If $\mu < \beta k$, the final epidemic size becomes finite.

Hence, both SIS and SIR model exhibits a transition at $\mu = \beta k$, above which epidemic is prevalent or affects a finite fraction of population.

1.4 Epidemic dynamics on scale-free networks

In the previous section, we showed that the epidemic threshold is given by

$$\frac{\beta}{\mu} > \frac{1}{k}. \quad (1.20)$$

The analysis of the previous section assumes that the number of nodes is infinite and the contacts between nodes are homogeneous. However, the contacts in the most real-world networks are heterogenous (section 1.1.2). In this section, we consider the epidemic threshold on heterogenous networks.

Here, we assume that the degree distribution is given by the power-law distribution with the exponent α :

$$P(k) = k^{-\alpha} \quad (k \geq 1). \quad (1.21)$$

and that there is no correlation between the degrees of neighboring nodes. This is achieved by the configuration model [11]. The configuration model is a model of a network characterized by the given distribution, but it is otherwise maximally random.

Let $\rho_k(t)$ be the probability that a node with degree k is infected at time t . Its time development is given by

$$\frac{d\rho_k(t)}{dt} = -\mu\rho_k(t) + [1 - \rho_k(t)]\{1 - [1 - \beta\pi_k(t)]^k\}, \quad (1.22)$$

where $\pi_k(t) = \sum_{k'} P(k'|k)i_{k'}(t)$ is the probability that its neighbor is infected. Note that a node with higher degree is more likely to become its neighbor than a node with lower degree. Therefore, $P(k)$ is given by

$$P(k'|k) = \frac{k'P(k')}{\langle k \rangle}. \quad (1.23)$$

Because we consider the condition near the epidemic threshold such that $\rho_k(t) \ll 1$, equation (1.22) leads to

$$\frac{d\rho_k(t)}{dt} = -\mu\rho_k(t) + \beta[1 - \rho_k(t)]k\pi_k(t). \quad (1.24)$$

Let $\theta(t)$ be $\sum_k kP(k)\rho_k(t)$. In the situation near the epidemic threshold, $\rho_k(t)$ and $\theta_k(t)$ are small enough to ignore their second-order terms. The time-development equation for $\theta(t)$ is given by

$$\dot{\theta}(t) = -\mu\theta(t) + \beta\frac{\langle k^2 \rangle}{\langle k \rangle}\theta(t). \quad (1.25)$$

Hence, the epidemic threshold is given by

$$\frac{\beta}{\mu} \geq \frac{\langle k \rangle}{\langle k^2 \rangle}. \quad (1.26)$$

This implies that in a network with a heavy-tailed degree distribution such that $\langle k^2 \rangle \rightarrow \infty$, the epidemic threshold vanishes. In most social contact networks, the exponent α is between 2 and 3, leading to $\langle k^2 \rangle \rightarrow \infty$ as $N \rightarrow \infty$. This indicates that once a emergent infectious disease appears, it hardly dies out.

1.5 Contents of the thesis

This thesis is composed of four chapters. Chapter 1 is the introduction as presented above. We introduce basic concepts of network science, describe the model of epidemic spreading, and review the recent result of epidemic spreading on networks as above. In chapter 2, we introduce the temporal-network approach for further understanding of epidemic spreading. We also review the previous studies of concurrency: its definition, how to measure, and its effects on epidemic dynamics. Chapter 3 is the main chapter of the thesis. In this chapter, we propose our novel approach using temporal networks and give the analytical understanding of the effect of concurrency. This chapter corresponds to the paper [T. Onaga, J. P. Gleeson, and N. Masuda, Phys. Rev. Lett. 119, 108301 (2017)] [12]. Chapter 4 is the conclusion and the future perspectives, in which we summarize the thesis and discuss the future direction of the research.

Chapter 2

Epidemic dynamics on temporal networks

2.1 Temporal networks

Since 2000, epidemic spreading on static networks, which we explained in the last section, has been studied. However, the real social contact networks changes its structure over time. This kind of networks is called temporal networks and is actively studied since 2010 [13]. The concept of temporal networks is important for the research of epidemic spreading, because the order of contacts determines the possible spreading path. For example, 3 people have contacts in the way indicated in Fig. 2.1. If we consider static networks (in this case, the network averaged over time), the disease can spread both from A to C and from C to A. However, the latter is not the case, because it does not follow the actual order. In addition, if two contacts from A to B and from B to C occur in a shorter time period, the disease can spread from A to C through B with higher probability, because B recovers before infecting C with lower probability. Static networks ignore these information.

Recent work showed that temporal networks can drastically change the spread-

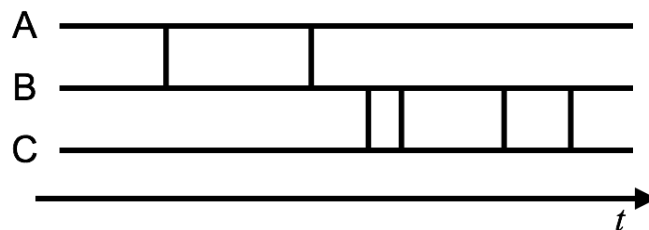


Figure 2.1: An example of a temporal network.

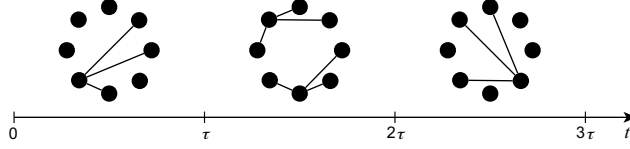


Figure 2.2: Schematic of an activity-driven network with $m = 3$.

ing dynamics on it, compared to static networks [6, 13, 14]. The factors that change the spreading dynamics are long-tailed distributions of inter-contact times, temporal and cross-edge correlation in inter-contact times, and entries and exits of nodes.

We consider the distribution of inter-contact times on each edge. If the inter-contact times follow the exponential distribution, each contact is drawn from a Poisson process. In this case, the dynamics on a temporal network coincides with that on the corresponding static network. In the real networks, inter-contact times follow the power-law distribution and is called bursty [15]. The bursty contact patterns may facilitate [16] or hinder [17] spreading processes.

In the last section of the previous chapter, we showed the lack of epidemic threshold in a heterogenous networks. However, in temporal networks, epidemic threshold does not always vanish. Here, as a model of temporal networks, we employ an activity-driven network. The activity-driven network is a model of temporally varying contacts (e.g., group conversation). We consider the following continuous-time susceptible-infected-susceptible (SIS) model on a discrete-time variant of activity-driven networks, which is a generative model of temporal networks [18–22]. The number of nodes is denoted by N . Each node i ($1 \leq i \leq N$) is assigned an activity potential a_i , drawn from a probability density $F(a)$ ($0 < a \leq 1$). Activity potential a_i is the probability with which node i is activated in a window of constant duration τ . If activated, node i creates m undirected links each of which connects to a randomly selected node (Fig. 2.2). If two nodes are activated and send edges to each other, we only create one edge between them. However, for large N and relatively small a_i , such events rarely occur.

The epidemic dynamics on this network is derived as follows. To derive the epidemic dynamics on a star-graph, we use the mean-field approximation. Note that if the epidemic dies out from a network with non-negligible probability, the mean-field approximation for epidemic dynamics breaks. The effect of dying out is negligible if the number of connected nodes is large enough or t is close enough to 0. Here, although the number of nodes of a star-graph is usually small, we consider the case in which the network changes fast enough to apply the mean-field approximation (in section 5.3 we discuss the extent to which this approximation

is valid). When $\tau \ll 1$, the time development of $\rho(a, t)$ is given by [21]

$$\begin{aligned} \rho(a, t + \tau) &= (1 - \mu\tau)\rho(a, t) + \beta\tau m[1 - \rho(a, t)]a \int da' \rho(a', t) \\ &+ \beta\tau[1 - \rho(a, t)] \int da' a' \rho(a', t). \end{aligned} \quad (2.1)$$

In the limit $\tau \rightarrow 0$, we obtain

$$\begin{pmatrix} \dot{\langle \rho \rangle} \\ \dot{\theta} \end{pmatrix} = \begin{pmatrix} -\mu + \beta m \langle a \rangle & \beta m \\ \beta m \langle a^2 \rangle & -\mu + \beta m \langle a \rangle \end{pmatrix} \begin{pmatrix} \langle \rho \rangle \\ \theta \end{pmatrix}, \quad (2.2)$$

where $\langle \rho(t) \rangle = \int da \rho(a, t)$ and $\theta(t) = \int da a \rho(a, t)$. Using the condition that the largest eigenvalue equals unity, we obtain the epidemic threshold

$$\beta_c = \frac{1}{m \left(\langle a \rangle + \sqrt{\langle a^2 \rangle} \right)}. \quad (2.3)$$

Even though an activity distribution is the power-law and the degree distribution of the aggregated static network is also the power-law distribution, the epidemic threshold stays finite. This is because in the case of static scale-free networks, the existence of nodes with extremely high degree mediate spreading with the high probability of getting infected and the high number of secondary infected nodes, and decreased the epidemic threshold [23], but in the case of activity-driven networks, there is no node like that at each time.

2.2 The effect of concurrency on epidemic dynamics

In the present study, we focus on a relatively neglected component of temporal networks, i.e., the number of concurrent contacts that a node has. Even if two temporal networks are the same when aggregated over a time horizon, they may be different as temporal networks due to different levels of concurrency. Concurrency is a long-standing concept in epidemiology, in particular in the context of monogamy/polygamy affecting sexually transmitted infections [24–26]. In this chapter, we introduce measures of concurrency, and review the studies of its effect on epidemic dynamics.

2.2.1 Measures of concurrency

Concurrency is the concept that indicates the number of concurrent partnerships [27]. It is considered to be low for monogamy and high for polygamy. In

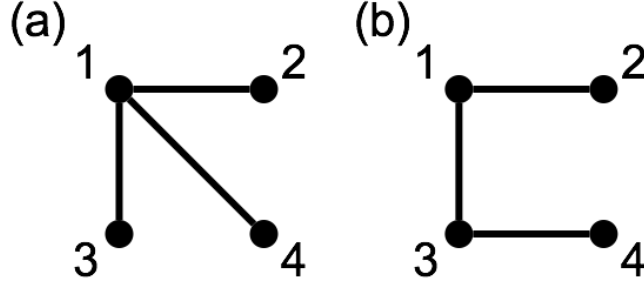


Figure 2.3: Two different contact networks with the same κ_1 values.

the real situations, the number of simultaneously active connections depends on the person and time. To measure the (average) level of concurrency, a couple of measures are proposed. Here, we introduce three measures of concurrency.

The first measure of concurrency is given by [24]

$$\kappa_1 = \frac{2L_1}{N_1^+}, \quad (2.4)$$

where N_1^+ denotes the number of non-isolate nodes in the network, and L_1 denotes the number of links. κ_1 is equal to 1 for monogamy. If there are multiple partnerships for some nodes, the number of non-isolate nodes is smaller than that for monogamy. κ_1 is larger than 1 for polygamy.

This measure captures the average degree of concurrent partnerships, but it is insensitive to the structure of concurrent partnerships in a network. For example, it cannot distinguish the difference in Fig. 2.3. From epidemiological perspective, (a) has more effect of concurrency, because infection transmission is much more rapid in situation (a) because the heterogeneity of degree is higher.

Line graph

To define a measure that is sensitive to these differences, we use the description of the contact graph. Partnerships in G are regarded as nodes in $L(G)$. If two partnerships in G share a common node, corresponding nodes in $L(G)$ has a link between them (Fig. 2.4). We call this graph $L(G)$ the line graph.

Although the number of edges in the line graph measures the number of concurrency, it increases with the number of nodes. We have two ways to normalize it with the number of nodes.

The first way is to divide the number of edges by the maximal number of edges [24]:

$$\kappa_2 = L_2 \left(\frac{N_2(N_2 - 1)}{2} \right)^{-1}, \quad (2.5)$$

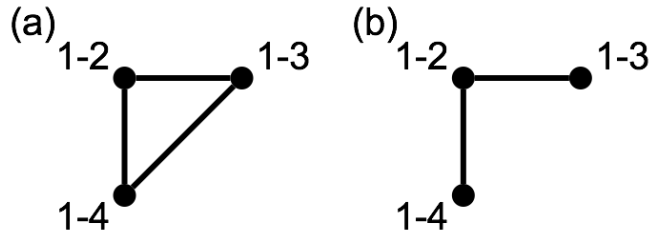


Figure 2.4: Line graphs for contact networks given in Fig. 2.3.

where N_2 and L_2 denotes the number of nodes and links in the line graph. κ_2 takes values between 0 and 1 [24].

The other way is to divide the number of edges by the number of nodes [25]:

$$\kappa_3 = \frac{L_2}{N_2}. \quad (2.6)$$

κ_3 is the mean number of concurrent partnerships per partnership.

2.2.2 Previous studies of concurrency

After the concept of concurrency was proposed in 1992 [27], its effects on HIV were studied numerically and analytically. Here, we review its effects on epidemic spreading with two studies.

C. Bauch and D. A. Rand numerically analyzed time evolution and the prevalence at the final state using a pair approximation [28]. A pair approximation is a method to incorporate the correlation of up to two nodes and ignore higher correlations. They modulate the value of κ_3 and calculate the effect. They found that concurrency increases the number of secondary infected people that a single infected people produce, and that it increases the final prevalence.

K. Y. Leung and M. Kretzschmar considered 6 scenarios with different speeds of network dynamics and different levels of the time-averaged contacts and numerically analyzed its effects on the number of secondary infected people (its effects on the epidemic threshold) [29]. They found that concurrency drives the system across epidemic threshold and dramatically enhances epidemics, and that this occurs at a low level of concurrency.

In previous studies, it is suggested that concurrency decreases epidemic threshold and increases the final prevalence. However, there is no solid theoretical understanding of its effect. First, some models increases the time-averaged number of contacts with an increase of concurrency [27, 30–32]. In this case, because the increase of the number of contacts obviously decrease the epidemic threshold [Eq. (1.20)] increase the prevalence, the effect of concurrency itself is not clear. The

effect should be analyzed with the time-averaged number of contacts fixed. Second, other studies with the fixed number of contacts are numerical [24, 25, 28, 29]. In these studies, because they fixed parameters (e.g., the speed of network dynamics) at a couple of values, we do not have exhaustive understanding. Note that there is a study that tackled these problems in the point of view of temporal networks [33]. However, their method cannot point out the drastic changes in the epidemic threshold and the final prevalence.

Chapter 3

Concurrency-induced transitions in epidemic dynamics on temporal networks

In the present study, we use the analytically tractable activity-driven model of temporal networks [18–22] to explicitly modulate the size of the concurrently active network with the structure of the aggregate network fixed. With this machinery, we carefully treat extinction effects, derive an analytically tractable matrix equation using a probability generating function for dynamical networks, and reveal non-monotonic effects of link concurrency on spreading dynamics. We show that the dynamics of networks can either enhance or suppress infection, depending on the amount of concurrency that individual nodes have. Note that analysis of epidemic processes driven by discrete pairwise contact events, which is a popular approach [6, 13, 14, 22, 34–38], does not address the problem of concurrency because we must be able to control the number of simultaneously active links possessed by a node in order to examine the role of concurrency without confounding with other aspects.

3.1 Model

We consider the following continuous-time susceptible-infected-susceptible (SIS) model on a discrete-time variant of activity-driven networks, which is a generative model of temporal networks [18–22].

For the SIS dynamics, each node takes either the susceptible or infected state. At any time, each susceptible node contracts infection at rate β per infected neighboring node. Each infected node recovers at rate μ irrespectively of the neighbors' states. Changing τ to $c\tau$ ($c > 0$) is equivalent to changing β and μ to β/c and

μ/c , respectively, while leaving τ unchanged. Therefore, we set $\mu = 1$ without loss of generality.

3.2 Analysis

We calculate the epidemic threshold as follows. First, we formulate SIS dynamics near the epidemic threshold on a static star graph, which is the building block of the activity-driven model, while explicitly considering extinction effects. Second, we convert the obtained set of linear difference equations into a tractable mathematical form with the use of a probability generating function of an activity distribution. Third, the epidemic threshold is obtained from an implicit function. For the sake of the analysis, we assume that star graphs generated by an activated node, which we call the hub, are disjoint from each other. Because a star graph with hub node i overlaps with another star graph with probability $\approx m \sum_{j \neq i} a_j (m + 1)/N \propto m^2 \langle a \rangle$, where $\langle a \rangle \equiv \int da F(a) a$ is the mean activity potential, we impose $m^2 \langle a \rangle \ll 1$. (However, our method works better than the so-called individual-based approximation even when $m^2 \langle a \rangle = 0.5$, as shown in section 5.2.) We denote by $\rho(a, t)$ the probability that a node with activity a is infected at time t . The fraction of infected nodes in the entire network at time t is given by $\langle \rho(t) \rangle \equiv \int da F(a) \rho(a, t)$. Let c_1 be the probability with which the hub in an isolated star graph is infected at time $t + \tau$, when the hub is the only infected node at time t and the network has switched to a new configuration right at time t . Let c_2 be the probability with which the hub is infected at $t + \tau$ when only a single leaf node is infected at t . The probability that a hub with activity potential a is infected after the duration τ of the star graph, denoted by ρ_1 , is given by

$$\rho_1(a, t + \tau) = c_1 \rho(a, t) + c_2 m \langle \rho(t) \rangle. \quad (3.1)$$

In deriving Eq. (3.1), we considered the situation near the epidemic threshold such that at most one node is infected in the star graph at time t [and hence $\rho(a, t), \langle \rho(t) \rangle \ll 1$]. The probability that a leaf with activity potential a that has a hub neighbor with activity potential a' is infected after time τ is analogously given by

$$\rho_2(a, a', t + \tau) = c_3 \rho(a, t) + c_4 \rho(a', t) + c_5 (m - 1) \langle \rho(t) \rangle, \quad (3.2)$$

where c_3 , c_4 , and c_5 are the probabilities with which a leaf node with activity potential a is infected after duration τ when only that leaf node, the hub, and a different leaf node is infected at time t , respectively. We derive formulas for c_i ($1 \leq i \leq 5$) in section 5.3. The probability that an isolated node with activity potential a is infected after time τ is given by $e^{-\tau} \rho(a, t)$. By combining these

contributions, we obtain

$$\begin{aligned} \rho(a, t + \tau) &= a\rho_1(a, t + \tau) + \int da' F(a') ma' \rho_2(a, a', t + \tau) \\ &+ (1 - a - m\langle a \rangle) e^{-\tau} \rho(a, t). \end{aligned} \quad (3.3)$$

To analyze Eq. (3.3) further, we take a generating function approach. With this approach, one trades a probability distribution for a probability generating function whose derivatives provide us with useful information about the distribution such as its moments. Furthermore, it often makes analysis easier, in particular linear analysis. By multiplying Eq. (3.3) by z^a and averaging over a , we obtain

$$\begin{aligned} \Theta(z, t + \tau) &= c'_1 \Theta^{(1)}(z, t) + c'_2 \Theta(1, t) g^{(1)}(z) + c'_3 \Theta(z, t) \\ &+ [c'_4 \Theta^{(1)}(1, t) + c'_5 \Theta(1, t)] g(z), \end{aligned} \quad (3.4)$$

where $c'_1 \equiv c_1 - e^{-\tau}$, $c'_2 \equiv mc_2$, $c'_3 \equiv e^{-\tau} + m\langle a \rangle(c_3 - e^{-\tau})$, $c'_4 \equiv mc_4$, $c'_5 \equiv m(m - 1)\langle a \rangle c_5$, $g(z) \equiv \int da F(a) z^a$ is the probability generating function of a , $\Theta(z, t) \equiv \int da F(a) \rho(a, t) z^a$, and throughout the paper the superscript (n) represents the n th derivative with respect to $\ln z$. Although Eq. (3.3) is an infinite dimensional system of linear difference equations, Eq. (3.4) is a single difference equation of $\Theta(z, t)$ and its derivative [39].

We expand $\rho(a, t)$ as a Maclaurin series as follows:

$$\rho(a, t) = \sum_{n=1}^{\infty} w_n(t) a^{n-1}. \quad (3.5)$$

Using this polynomial basis representation (the convergence is proven in the Supplemental Material), we can consider the differentiations in Eq. (3.4) (i.e., $\Theta^{(1)}(z, t)$ and $g^{(1)}(z)$) as an exchange of bases and convert Eq. (3.4) into a tractable matrix form. Let p_0 be the fraction of initially infected nodes, which are selected uniformly at random, independently of a . We represent the initial condition as $\mathbf{w}(t = 0) \equiv (w_1(0), w_2(0), \dots)^\top = (p_0, 0, 0, \dots)^\top$. Epidemic dynamics near the epidemic threshold obey linear dynamics given by

$$\mathbf{w}(t + \tau) = \mathbf{T}(\tau) \mathbf{w}(t). \quad (3.6)$$

By substituting $\Theta(z, t) = \sum_{n=1}^{\infty} w_n(t) g^{(n-1)}(z)$ and $g^{(n-1)}(1) = \langle a^{n-1} \rangle$ in Eq. (3.4), we obtain

$$\mathbf{T} = \begin{pmatrix} c'_3 + \langle a \rangle c'_4 + c'_5 & \langle a^2 \rangle c'_4 + \langle a \rangle c'_5 & \langle a^3 \rangle c'_4 + \langle a^2 \rangle c'_5 & \langle a^4 \rangle c'_4 + \langle a^3 \rangle c'_5 & \langle a^5 \rangle c'_4 + \langle a^4 \rangle c'_5 & \dots \\ c'_1 + c'_2 & \langle a \rangle c'_2 + c'_3 & \langle a^2 \rangle c'_2 & \langle a^3 \rangle c'_2 & \langle a^4 \rangle c'_2 & \dots \\ 0 & c'_1 & c'_3 & 0 & 0 & \dots \\ 0 & 0 & c'_1 & c'_3 & 0 & \dots \\ 0 & 0 & 0 & c'_1 & c'_3 & \dots \\ \vdots & \vdots & \vdots & \vdots & \vdots & \ddots \end{pmatrix}. \quad (3.7)$$

A positive prevalence $\langle \rho(t) \rangle$ (i.e., a positive fraction of infected nodes in the equilibrium state) occurs only if the largest eigenvalue of $\mathbf{T}(\tau)$ exceeds 1, because in this situation the probability of being infected grows in time, at least in the linear regime. Therefore, we get the following implicit function for the epidemic threshold, denoted by β_c :

$$\begin{aligned} f(\tau, \beta_c) &\equiv \frac{(1-r)(1-s) - (1+q)u}{S(q)} \\ &- qr - qs + qrs - q^2u - rs = 0, \end{aligned} \quad (3.8)$$

where $S(q) \equiv \sum_{n=0}^{\infty} (\langle a^{n+2} \rangle / \langle a \rangle^{n+2}) q^n = (1/\langle a \rangle^2) \{ \langle (a^2) / [1 - (a/\langle a \rangle)q] \rangle \}$, $q \equiv \langle a \rangle c'_1 / (1 - c'_3)$, $r \equiv \langle a \rangle c'_2 / (1 - c'_3)$, $s \equiv \langle a \rangle c'_4 / (1 - c'_3)$, and $u \equiv c'_5 / (1 - c'_3)$ (see section 5.4 for the derivation). Note that f is a function of β ($= \beta_c$) through q , r , s , and u , which are functions of β . In general, we obtain β_c by numerically solving Eq. (3.8), but some special cases can be determined analytically.

In the limit $\tau \rightarrow 0$, Eq. (3.8) gives $\beta_c = \left[m \left(\langle a \rangle + \sqrt{\langle a^2 \rangle} \right) \right]^{-1}$, which coincides with the epidemic threshold for the activity-driven model derived in the previous studies [18, 21]. In fact, this β_c value is the epidemic threshold for the aggregate (and hence static) network, whose adjacency matrix is given by $A_{ij}^* \approx m(a_i + a_j)/N$ [13, 33], as demonstrated in Fig. 5.1.

For general τ , if all nodes have the same activity potential a , and if $m = 1$, we obtain β_c as the solution of the following implicit equation:

$$\begin{aligned} &2ae^{\frac{(\beta_c-1)\tau}{2}} \left[\cosh\left(\frac{\kappa_c\tau}{2}\right) + \frac{1+3\beta_c}{\kappa_c} \sinh\left(\frac{-\kappa_c\tau}{2}\right) \right] \\ &- e^\tau + 1 - 2a = 0, \end{aligned} \quad (3.9)$$

where $\kappa_c = \sqrt{\beta_c^2 + 6\beta_c + 1}$.

The theoretical estimate of the epidemic threshold [Eq. (3.8); we use Eq. (3.9) in the case of $m = 1$] is shown by the solid lines in Figs. 3.1(a) and 3.1(b). It is compared with numerically calculated prevalence values for various τ and β values shown in different colors. Equations (3.8) and (3.9) describe the numerical results fairly well. When $m = 1$, the epidemic threshold increases with τ and diverges at $\tau \approx 0.1$ [Fig. 3.1(a)]. Furthermore, slower network dynamics (i.e., larger values of τ) reduce the prevalence for all values of β . In contrast, when $m = 10$, the epidemic threshold decreases and then increases as τ increases [Fig. 3.1(b)]. The network dynamics (i.e., finite τ) impact epidemic dynamics in a qualitatively different manner depending on m , i.e., the number of concurrent neighbors that a hub has. Note that the estimate of β_c by the individual-based approximation ([33], see section 5.6 for the derivation), which may be justified when $m \gg 1$, is consistent with the numerical results and our theoretical results only at small τ [a dashed line

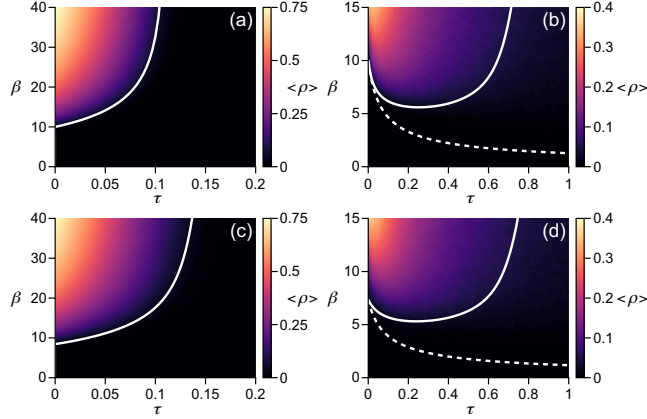


Figure 3.1: Epidemic threshold and the numerically-simulated prevalence when $m = 1$ (a) and (c) and $m = 10$ (b) and (d). In (a) and (b), all nodes have the same activity potential value a . The solid lines represent the analytical estimate of the epidemic threshold [Eq. (3.8); we plot Eq. (3.9) instead in (a)]. The dashed lines represent the epidemic threshold obtained from the individual-based approximation (section 5.6). The color indicates the prevalence. In (c) and (d), the activity potential ($\epsilon \leq a_i \leq 0.9$, $1 \leq i \leq N$) obeys a power-law distribution with exponent 3. In (a)–(d), we set $N = 2000$ and adjust the values of a and ϵ such that the mean degree is the same ($\langle k \rangle = 0.1$) in the four cases. We simulate the stochastic SIS dynamics using the quasistationary state method [40], as in [33], and calculate the prevalence averaged over 100 realizations after discarding the first 15 000 time steps. We set the step size $\Delta t = 0.002$. Qualitatively similar results are obtained for the variant of the activity-driven model with a reinforcement mechanism of link creation [41] (Fig. 5.2).

in Fig. 3.1(b)]. Qualitatively similar results are found, when the activity potential a is power-law distributed [Figs. 3.1(c) and 3.1(d)].

To illuminate the qualitatively different behaviors of the epidemic threshold as τ increases, we determine a phase diagram for the epidemic threshold. We focus our analysis on the case in which all nodes share the activity potential value a , noting that qualitatively similar results are also found for power-law distributed activity potentials [Fig. 3.2(b)]. We calculate the two boundaries partitioning different phases as follows. First, we observe that the epidemic threshold diverges at $\tau = \tau_*$. In the limit $\beta \rightarrow \infty$, infection starting from a single infected node in a star graph immediately spreads to the entire star graph, leading to $c_i \rightarrow 1$ ($1 \leq i \leq 5$). By substituting $c_i \rightarrow 1$ in Eq. (3.8), we obtain $f(\tau_*, \beta_c \rightarrow \infty) = 0$, where

$$\tau_* = \ln \frac{1 - (1 + m)a}{1 - (1 + m)^2 a}. \quad (3.10)$$

When $\tau > \tau_*$, infection always dies out even if the infection rate is infinitely large. This is because, in a finite network, infection always dies out after a sufficiently long time due to stochasticity [42–44]. Second, although β_c eventually diverges as τ increases, there may exist τ_c such that β_c at $\tau < \tau_c$ is smaller than the β_c value at $\tau = 0$. Motivated by the comparison between the behavior of β_c at $m = 1$ and $m = 10$ (Fig. 3.1), we postulate that $\tau_c (> 0)$ exists only for $m > m_c$. Then, we obtain $d\beta_c/d\tau = 0$ at $(\tau, m) = (0, m_c)$. The derivative of Eq. (3.8) gives $\partial f/\partial\tau + (\partial f/\partial\beta_c)(d\beta_c/d\tau) = 0$. Because $d\beta_c/d\tau = 0$ at $(\tau, m) = (0, m_c)$, we obtain $\partial f/\partial\tau = 0$, which leads to

$$m_c = \frac{3}{1 - 4a}. \quad (3.11)$$

When $m < m_c$, network dynamics (i.e., finite τ) always reduce the prevalence for any τ [Figs. 3.1(a) and 3.1(c)]. When $m > m_c$, a small τ raises the prevalence as compared to $\tau = 0$ (i.e., static network) but a larger τ reduces the prevalence [Figs. 3.1(b) and 3.1(d)]. τ_* and m_c for general activity distributions are given in sections 5.7 and 5.8, respectively.

The phase diagram based on Eqs. (3.10) and (3.11) is shown in Fig. 3.2(a). The β_c values numerically calculated by solving Eq. (3.8) are also shown in the figure. It should be noted that the parameter values are normalized such that β_c has the same value for all m at $\tau = 0$. We find that the dynamics of the network may either increase or decrease the prevalence, depending on the number of connections that a node can simultaneously have, extending the results shown in Fig. 3.1.

These results are not specific to the activity-driven model. The phase diagram is qualitatively similar for randomly distributed m (Fig. 5.5), for different distributions of activity potentials (Fig. 5.7), and for a different model in which an activated node induces a clique instead of a star (Fig. 5.3), modeling a group conversation event as some temporal network models do [45–47].

3.3 Discussion

Our analytical method shows that the presence of network dynamics boosts the prevalence (and decreases the epidemic threshold β_c) when the concurrency m is large and suppresses the prevalence (and increases β_c) when m is small, for a range of values of the network dynamic time scale τ . This result lends theoretical support to previous claims that concurrency boosts epidemic spreading [18, 24, 25, 27–32, 48]. The result may sound unsurprising because a large m value implies that there exists a large connected component at any given time. However, our finding is not trivial because a large component consumes many edges such that other parts

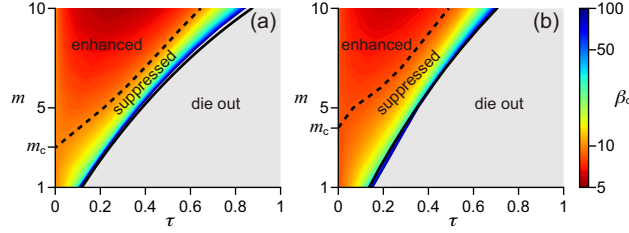


Figure 3.2: Phase diagrams for the epidemic threshold, β_c , when the activity potential is (a) equal to a for all nodes, or (b) obeys a power-law distribution with exponent 3 ($\epsilon \leq a_i \leq 0.9$). We set $\langle k \rangle = 0.1$ at $m = 1$ and adjust the value of a and ϵ such that β_c takes the same value for all m at $\tau = 0$. In the “die out” phase, infection eventually dies out for any finite β . In the “suppressed” phase, β_c is larger than the β_c value at $\tau = 0$. In the “enhanced” phase, β_c is smaller than the β_c value at $\tau = 0$. The solid and dashed lines represent τ_* [Eq. (3.10)] and τ_c , respectively. The color bar indicates the β_c values. In the gray regions, $\beta_c > 100$.

of the network at the same time or the network at other times would be more sparsely connected as compared to the case of a small m . We confirmed that qualitatively similar results are found when the activity potentials were constructed from two empirical social contact networks (Fig. 5.4). Our results confirm that a monogamous sexual relationship or a small group of people chatting face to face, as opposed to polygamous relationships or large groups of conversations, hinders epidemic spreading, where we compare like with like by constraining the aggregate (static) network to be the same in all cases. For general temporal networks, immunization strategies that decrease concurrency (e.g., discouraging polygamy) may be efficient. Restricting the size of the concurrent connected component (e.g., size of a conversation group) may also be a practical strategy.

Another important contribution of the present study is the observation that infection dies out for a sufficiently large τ , regardless of the level of concurrency. As shown in Figs. 3.2 and 5.3, the transition to the “die out” phase occurs at values of τ that correspond to network dynamics and epidemic dynamics having comparable time scales. This is a stochastic effect and cannot be captured by existing approaches to epidemic processes on temporal networks that neglect stochastic dying out, such as differential equation systems for pair formulation-dissolution models [25, 28–30, 32] and individual-based approximations [33, 49, 50]. Our analysis methods explicitly consider such stochastic effects, and are therefore expected to be useful beyond the activity-driven model (or the clique-based temporal networks analyzed in section 5.11) and the SIS model.

Chapter 4

Conclusion and Future Perspective

4.1 Conclusion

In this work, to elucidate the effect of concurrency, we carefully treat the effect of stochastic dying-out and propose the novel mathematical approach, which allows us to use arbitrary distributions under a few assumptions. We show that the dynamics of networks can either enhance or suppress infection, depending on the amount of concurrency that individual nodes have. We added the analytical understanding of the non-monotonous effect of concurrency in terms of the parameter region of importance.

4.2 Future perspective

We discuss farther development of this research and the future directions of the study of this field as follows.

4.2.1 Concurrency

First, we discuss the future work of concurrency. Thus far, there is no common understanding of the measure of concurrency. Many recent studies use a couple of proposed definitions. To achieve a consensus on its measure, we mention two important points. First, the measure should reflect its effect on epidemic spreading. Second, although most of the previously proposed measures are a single value, but more than one value (e.g., the moments or cumulants of the distribution of the number of concurrent partners) may be a good measure and capture more

information because the number of partners changes considerably depending on person and time.

In this work, we focus on the case in which all star-graphs disappear and new star-graphs appear at the same time. In the real situations, star-graphs disappear and appear at different times (group conversations) or changes its shape gradually (sexual contacts). We expect that if τ is small, this effect is not important and if τ is too large, the epidemic dies out any way. In the middle region of τ value, this may influence the phase diagram.

In this work, we focus on the epidemic threshold. Although the epidemic threshold is the most important parameter in epidemic dynamics because it determines whether epidemic spreads or dies out, epidemic dynamics has other important things (e.g. the prevalence at the final state, the speed of spreading, the efficient way to immunize people). They warrant future work.

4.2.2 Social information spreading

We consider the epidemic spreading in this work. We believe that the main findings of this work can be applied beyond the models that we use here. Particularly important application is social information spreading. Social opinion and information gathers huge interest from academic and industrial researchers with various applications, e.g. viral marketing, the design of the capacity of servers of social networking services. As opposed to simple contagion of epidemic spreading, in which the probability of infection is proportional to the number of contacts, the spreading of opinions or information is considered as complex contagion because the number of infection of a piece of opinion or information increases when the person hear the same one from a different person or group. We believe that our findings may be observed in the model of social information spreading, but it warrants future study.

4.2.3 Temporal networks

Finally, we discuss the future direction of the analytical work on the epidemic dynamics on temporal networks. In most of the previous work, temporal structure and spatial structure were considered separately [50]. However, they are highly correlated. Recently, a method to predict epidemic dynamics on empirical temporal contact networks using a message-passing algorithm is proposed [50]. This method is an efficient algorithm and can calculate the dynamics on complex temporal networks with relatively small amount of computation. With the increasing availability of data of contact networks, we believe that this method is promising.

Current message-passing methods are based on the mean-field approximation [50, 51]. However, the social contact networks in the real-world consist of

not connected small networks (usually several people or tens of people, up to hundreds of people) at each time. Therefore, we believe that a method incorporating stochastic dying-out is necessary for precise prediction.

Chapter 5

Appendix

5.1 Prevalence on the aggregate network

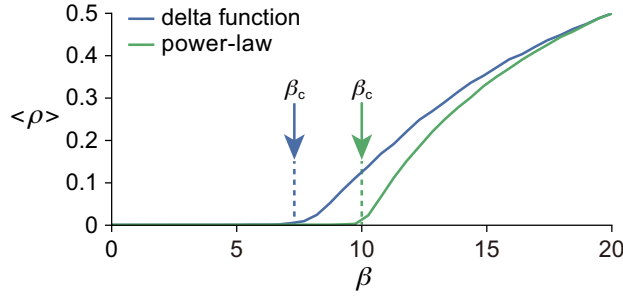


Figure 5.1: Prevalence on the aggregate (hence static) network whose adjacency matrix is given (in the limit $N \rightarrow \infty$) by $A_{ij}^* = m(a_i + a_j)/N$ [13, 33]. The lines represent the numerical results for the delta function (i.e., all nodes have same activity potential) and power-law activity distributions. The arrows indicate $\beta_c = \left[m \left(\langle a \rangle + \sqrt{\langle a^2 \rangle} \right) \right]^{-1}$. We set $m = 5$ and $\langle a \rangle = 0.01$.

5.2 When the low-activity assumption is violated

Here we consider the situation in which the low-activity assumption $m^2 \langle a \rangle \ll 1$ is violated. When $m \ll N$, the expected number of star graphs that a star graph overlaps with is given by

$$p = N \langle a \rangle \left[1 - \left(1 - \frac{m+1}{N-1} \right)^m \right] \approx m(m+1) \langle a \rangle. \quad (5.1)$$

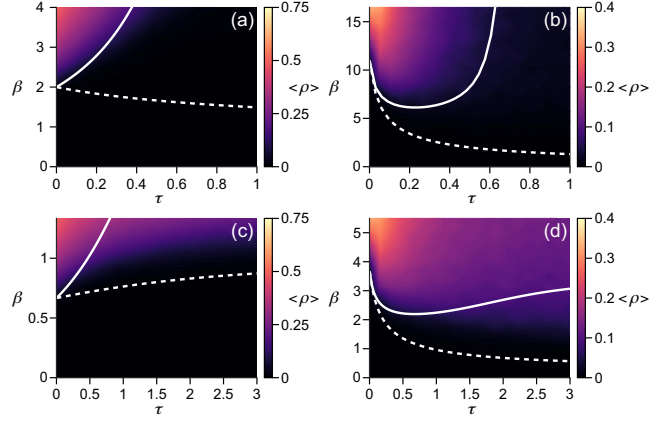


Figure 5.2: Epidemic threshold and numerically calculated prevalence when the low-activity assumption is violated. We set $m = 1$ in (a) and (c), $m = 10$ in (b) and (d), $p = 0.5$ in (a) and (b), and $p = 1.5$ in (c) and (d). The solid and dashed lines represent the epidemic threshold obtained from Eq. (3.8) and that obtained from the individual-based approximation, respectively. All nodes are assumed to have the same activity potential $a = 0.25$ in (a), $a = 0.0045$ in (b), $a = 0.75$ in (c), and $a = 0.0136$ in (d). We calculated the prevalence averaged over 100 simulations after discarding the first 15000 time steps of each simulation. We set $N = 1000$ and $\Delta t = 0.002$.

If $p \ll 1$ is violated, a star graph would overlap with others such that the actual concurrency is larger than m . In the extreme case of $p \geq 1$, almost all star graphs overlap with each other such that the concurrency is not sensitive to m . In this situation, our results overestimate the epidemic threshold because our analysis does not take into account infections across different star graphs. If $p \geq 1$, the individual-based approximation describes the numerical results more accurately than our method does [Figs. 5.2(c) and 5.2(d)]. However, even at a moderately large value of p ($= 0.5$), our method is more accurate than the individual-based approximation [Figs. 5.2 (a) and 5.2(b)].

5.3 Derivation of c_1 , c_2 , c_3 , c_4 , and c_5

We consider SIS dynamics on a star graph with m leaves and derive c_1 , c_2 , c_3 , c_4 , and c_5 . Let us denote the state of the star graph by $\{x, y, z\}$ ($x, y \in \{S, I\}$, $0 \leq z \leq m-1$), where x and y are the states of the hub and a specific leaf node, respectively, and z is the number of infected nodes in the other $m-1$ leaf nodes. Although a general network with $m+1$ nodes allows 2^{m+1} states, using this notation, we can describe SIS dynamics on a star graph by a continuous-time Markov process with

$4m$ states [43].

We denote the transition rate matrix of the Markov process by $\mathbf{M}$. Its element $M_{\{x',y',z'\},\{x,y,z\}}$ is equal to the rate of transition from $\{x,y,z\}$ to $\{x',y',z'\}$. The diagonal elements are given by

$$M_{\{x,y,z\},\{x,y,z\}} = - \sum_{\{x',y',z'\} \neq \{x,y,z\}} M_{\{x',y',z'\},\{x,y,z\}}. \quad (5.2)$$

The rates of the recovery events are given by

$$M_{\{S,y,z\},\{I,y,z\}} = 1, \quad (5.3)$$

$$M_{\{x,S,z\},\{x,I,z\}} = 1, \quad (5.4)$$

$$M_{\{x,y,z-1\},\{x,y,z\}} = z \quad (z \geq 1). \quad (5.5)$$

The rates of the infection events are given by

$$M_{\{I,S,z\},\{S,S,z\}} = z\beta, \quad (5.6)$$

$$M_{\{I,I,z\},\{S,I,z\}} = (z+1)\beta, \quad (5.7)$$

$$M_{\{I,I,z\},\{I,S,z\}} = \beta, \quad (5.8)$$

$$M_{\{I,y,z+1\},\{I,y,z\}} = (m-1-z)\beta \quad (z \leq m-2). \quad (5.9)$$

The other elements of $\mathbf{M}$ are equal to 0. Let $p_{\{x,y,z\}}(t)$ be the probability for a star graph to be in state $\{x,y,z\}$ at time t . Because

$$\dot{\mathbf{p}}(t) = \mathbf{M}\mathbf{p}(t), \quad (5.10)$$

where $\mathbf{p}(t)$ is the $4m$ -dimensional column vector whose elements are $p_{\{x,y,z\}}(t)$, we obtain

$$\mathbf{p}(t) = \exp(\mathbf{M}t)\mathbf{p}(0). \quad (5.11)$$

Note that c_1 and c_2 are the probabilities with which $x = I$ at time τ , when the initial state is $\{I, S, 0\}$ and $\{S, I, 0\}$, respectively, and that c_3 , c_4 , and c_5 are the probabilities that $y = I$ at time τ , when the initial state is $\{S, I, 0\}$, $\{I, S, 0\}$, and $\{S, S, 1\}$, respectively. Therefore, we obtain

$$\begin{pmatrix} c_1 \\ c_2 \\ c_3 \\ c_4 \\ c_5 \end{pmatrix} = \begin{pmatrix} \sum_{y,z} [\exp(\mathbf{M}\tau)]_{\{I,y,z\},\{I,S,0\}} \\ \sum_{y,z} [\exp(\mathbf{M}\tau)]_{\{I,y,z\},\{S,I,0\}} \\ \sum_{x,z} [\exp(\mathbf{M}\tau)]_{\{x,I,z\},\{S,I,0\}} \\ \sum_{x,z} [\exp(\mathbf{M}\tau)]_{\{x,I,z\},\{I,S,0\}} \\ \sum_{x,z} [\exp(\mathbf{M}\tau)]_{\{x,I,z\},\{S,S,1\}} \end{pmatrix}. \quad (5.12)$$

When $m = 1$, Eq. (5.12) yields

$$c_1 = c_3 = \frac{e^{-\tau}}{2} \left[e^{-\beta\tau} + e^{-\frac{1+\beta}{2}\tau} \left(\cosh \frac{\kappa\tau}{2} + \frac{1+3\beta}{\kappa} \sinh \frac{\kappa\tau}{2} \right) \right], \quad (5.13)$$

$$c_2 = c_4 = \frac{e^{-\tau}}{2} \left[-e^{-\beta\tau} + e^{-\frac{1+\beta}{2}\tau} \left(\cosh \frac{\kappa\tau}{2} + \frac{1+3\beta}{\kappa} \sinh \frac{\kappa\tau}{2} \right) \right], \quad (5.14)$$

where $\kappa = \sqrt{\beta^2 + 6\beta + 1}$, and c_5 is not defined.

When $m \gg 1$, we can apply an individual-based approximation [4, 33, 49]. We assume that the state of each node is statistically independent of each other, i.e.,

$$p_{\{x,y,z\}} \approx P(x)P(y)P(z), \quad (5.15)$$

where $P(x)$, for example, is the probability that the hub takes state x . We have suppressed t in Eq. (5.15). Under the individual-based approximation, x and y obey Bernoulli distributions with parameters p_1^{MF} and p_2^{MF} , respectively, and z obeys a binomial distribution with parameters $m - 1$ and p_3^{MF} , where $\mathbf{p}^{\text{MF}} \equiv (p_1^{\text{MF}}, p_2^{\text{MF}}, p_3^{\text{MF}})^{\top}$ is given by

$$\mathbf{p}^{\text{MF}} = \begin{pmatrix} P(x = I) \\ P(y = I) \\ \frac{\langle z \rangle}{m-1} \end{pmatrix} = \begin{pmatrix} \sum_{y,z} p_{\{I,y,z\}} \\ \sum_{x,z} p_{\{x,I,z\}} \\ \frac{1}{m-1} \sum_{x,y,z} z p_{\{x,y,z\}} \end{pmatrix}. \quad (5.16)$$

By substituting Eq. (5.10) in the time derivative of Eq. (5.16), we obtain

$$\dot{\mathbf{p}}^{\text{MF}} = \begin{pmatrix} -p_1^{\text{MF}} + \beta p_2^{\text{MF}} + (m-1)\beta p_3^{\text{MF}} \\ \beta p_1^{\text{MF}} - p_2^{\text{MF}} \\ \beta p_1^{\text{MF}}(1 - p_3^{\text{MF}}) - p_3^{\text{MF}} \end{pmatrix}. \quad (5.17)$$

If $p_3^{\text{MF}} \ll 1$, $\mathbf{p}^{\text{MF}}$ obeys linear dynamics given by

$$\dot{\mathbf{p}}^{\text{MF}} \approx \mathbf{M}^{\text{MF}} \mathbf{p}^{\text{MF}} \quad (5.18)$$

where

$$\mathbf{M}^{\text{MF}} = \begin{pmatrix} -1 & \beta & (m-1)\beta \\ \beta & -1 & 0 \\ \beta & 0 & -1 \end{pmatrix}. \quad (5.19)$$

In a similar fashion to the derivation of Eq. (5.12), we obtain

$$\begin{aligned}
\begin{pmatrix} c_1 \\ c_2 \\ c_3 \\ c_4 \\ c_5 \end{pmatrix} &\approx \begin{pmatrix} [\exp(\mathbf{M}^{\text{MF}} \tau)]_{11} \\ [\exp(\mathbf{M}^{\text{MF}} \tau)]_{12} \\ [\exp(\mathbf{M}^{\text{MF}} \tau)]_{22} \\ [\exp(\mathbf{M}^{\text{MF}} \tau)]_{21} \\ \frac{1}{m-1} [\exp(\mathbf{M}^{\text{MF}} \tau)]_{23} \end{pmatrix} \\
&= e^{-\tau} \begin{pmatrix} \cosh(\beta \sqrt{m} \tau) \\ \frac{1}{\sqrt{m}} \sinh(\beta \sqrt{m} \tau) \\ 1 + \frac{\cosh(\beta \sqrt{m} \tau) - 1}{m} \\ \frac{1}{\sqrt{m}} \sinh(\beta \sqrt{m} \tau) \\ \frac{1}{m} (\cosh(\beta \sqrt{m} \tau) - 1) \end{pmatrix}. \tag{5.20}
\end{aligned}$$

We estimate the extent to which Eq. (5.20) is valid as follows. First, we need $m \gg 1$, because the initial condition $p_3^{\text{MF}} = 1/(m-1)$ should satisfy $p_3^{\text{MF}} \ll 1$. Second, p_3^{MF} must satisfy

$$p_3^{\text{MF}}(\tau) \leq \beta(1 - e^{-\tau}) + p_3^{\text{MF}}(0)e^{-\tau} \tag{5.21}$$

because $p_1^{\text{MF}} \leq 1$ in Eq. (5.17). To satisfy $p_3^{\text{MF}} \ll 1$, we need $\tau < 1/\beta$. This condition remains unchanged by re-scaling (τ, β) to $(c\tau, \beta/c)$. These two conditions are sufficient for this approximation to be valid. If $m \gg 1$ is violated, the individual-based approximation significantly underestimates the epidemic threshold for any finite τ because it ignores the effect of stochastic dying-out. If $\tau < 1/\beta$ is violated, the approximation [dashed lines in Fig. 3.1(b) and (d)] underestimates the epidemic threshold because dynamics on the star graph deviate from the linear regime. In particular, the epidemic threshold obtained from the approximation [Eq. (5.48)] remains finite even in the limit $\tau \rightarrow \infty$, whereas analytical [Eq. (3.8)] and numerical (Fig. 3.1) results diverge at a finite τ .

5.4 Derivation of Eq. (3.8)

At the epidemic threshold, the largest eigenvalue of $\mathbf{T}$ is equal to unity. Let $\mathbf{v} = (v_1, v_2, \dots)^\top$ be the corresponding eigenvector of $\mathbf{T}$. We normalize $\mathbf{v}$ such that $\sum_{j=1}^{\infty} v_j = 1$. By substituting Eq. (3.7) in $\mathbf{v} = \mathbf{T}\mathbf{v}$, we obtain the system of

equations

$$v_1 = c'_3 v_1 + c'_4 \sum_{n=1}^{\infty} \langle a^n \rangle v_n + c'_5 \sum_{n=1}^{\infty} \langle a^{n-1} \rangle v_n, \quad (5.22)$$

$$v_2 = c'_1 v_1 + c'_3 v_2 + c_2 \sum_{n=1}^{\infty} \langle a^{n-1} \rangle v_n, \quad (5.23)$$

$$v_j = c'_1 v_{j-1} + c'_3 v_j \quad (j \geq 3). \quad (5.24)$$

Equation (5.24) gives

$$v_j = \frac{q}{\langle a \rangle} v_{j-1} \quad (j \geq 3), \quad (5.25)$$

where

$$q \equiv \frac{\langle a \rangle c'_1}{1 - c'_3}. \quad (5.26)$$

By combining Eqs. (5.23) and (5.25), we obtain

$$(q + r)v_1 = \langle a \rangle [1 - (1 + qS)r] v_2, \quad (5.27)$$

where

$$r \equiv \frac{\langle a \rangle c'_2}{1 - c'_3}, \quad (5.28)$$

$$S(q) \equiv \sum_{n=0}^{\infty} \frac{\langle a^{n+2} \rangle}{\langle a \rangle^{n+2}} q^n = \frac{1}{\langle a \rangle^2} \left\langle \frac{a^2}{1 - \frac{a}{\langle a \rangle} q} \right\rangle. \quad (5.29)$$

Because $\mathbf{v}$ is normalized, we obtain

$$\mathbf{v} = \begin{pmatrix} \frac{[\langle a \rangle - q][1 - (1 + qS)r]}{r + \langle a \rangle + (1 + qS)[q - \langle a \rangle]r} \\ \frac{[1 - \frac{q}{\langle a \rangle}](q + r)}{r + \langle a \rangle + (1 + qS)[q - \langle a \rangle]r} \\ \frac{\frac{q}{\langle a \rangle}[1 - \frac{q}{\langle a \rangle}](q + r)}{r + \langle a \rangle + (1 + qS)[q - \langle a \rangle]r} \\ \frac{(\frac{q}{\langle a \rangle})^2[1 - \frac{q}{\langle a \rangle}](q + r)}{r + \langle a \rangle + (1 + qS)[q - \langle a \rangle]r} \\ \vdots \end{pmatrix}. \quad (5.30)$$

Equation (5.22) leads to

$$[1 - s - u]v_1 = \langle a \rangle [sS + (1 + qS)u] v_2, \quad (5.31)$$

where,

$$s \equiv \frac{\langle a \rangle c'_4}{1 - c'_3}, \quad (5.32)$$

$$u \equiv \frac{c'_5}{1 - c'_3}. \quad (5.33)$$

By substituting Eq. (5.30) in Eq. (5.31), we obtain

$$\begin{aligned} f(\tau, \beta_c) &\equiv \frac{(1-r)(1-s) - (1+q)u}{S(q)} \\ &- qr - qs + qrs - q^2u - rs = 0, \end{aligned} \quad (5.34)$$

which is Eq. (3.8) in the main text. If all nodes have the same activity potential a , Eq. (5.34) is reduced to

$$f(\tau, \beta_c) = 1 - q - r - s - u = 0. \quad (5.35)$$

5.5 Convergence of the Maclaurin series

We derive the condition under which the Maclaurin series in Eq. (5.41) converges for any t when $\beta \leq \beta_c$. First, at $t = 0$, the series converges because $\mathbf{w}(0) = (p_0, 0, 0, \dots)^\top$.

Second, consider a finite t . It should be noted that the series is only defined at t that is a multiple of τ . Because $T_{ij} = 0$ ($i \geq j + 2$) in Eq. (3.7), we obtain

$$w_n(t) = 0 \quad \text{for } n \geq 1 + \frac{t}{\tau}. \quad (5.36)$$

Therefore, the series converges.

Third, we consider the limit $t \rightarrow \infty$. If $\beta < \beta_c$, because

$$\lim_{t \rightarrow \infty} \langle \rho \rangle = 0, \quad (5.37)$$

we obtain

$$\lim_{t \rightarrow \infty} w_n(t) = 0 \quad \text{for } n \geq 1. \quad (5.38)$$

Therefore, the series converges. For $\beta = \beta_c$, we consider the convergence of the series when

$$\lim_{t \rightarrow \infty} \mathbf{w}(t) = b\mathbf{v}, \quad (5.39)$$

where $\mathbf{v}$ is the eigenvector of $\mathbf{T}$ given by Eq. (5.30), and b is a constant. Because Eq. (5.30) yields

$$\lim_{j \rightarrow \infty} \frac{v_{j+1}}{v_j} = \frac{q}{\langle a \rangle}, \quad (5.40)$$

the radius of convergence is equal to $\langle a \rangle / q$. To ensure convergence, we require that

$$\max_i (a_i) < \frac{\langle a \rangle}{q}. \quad (5.41)$$

Because c_i ($1 \leq i \leq 5$) are probabilities, we obtain

$$c_1 \leq 1, \quad (5.42)$$

$$c_3 \leq 1. \quad (5.43)$$

By substituting Eqs. (5.42) and (5.43) in the definitions of c'_1 and c'_2 , we obtain

$$c'_1 \leq 1 - e^{-\tau}, \quad (5.44)$$

$$c'_3 \leq e^{-\tau} + m\langle a \rangle(1 - e^{-\tau}). \quad (5.45)$$

By substituting Eqs. (5.44) and (5.45) in Eq. (5.26), we obtain

$$q \leq \frac{\langle a \rangle}{1 - m\langle a \rangle}. \quad (5.46)$$

Inequalities (5.42)–(5.46) hold with equality in the limit $\beta \rightarrow \infty$. Hence, a sufficient condition for convergence is given by

$$\max_i (a_i) < 1 - m\langle a \rangle. \quad (5.47)$$

Equation (5.47) holds true in practical situations because the assumption $m^2\langle a \rangle \ll 1$ guarantees that $m\langle a \rangle \ll 1$ and a_i is a probability.

5.6 Epidemic threshold under the individual-based approximation

When $m \gg 1$, the epidemic threshold can be obtained by the individual-based approximation [4, 33, 49]. We assume that all nodes have the same activity potential a . By substituting Eq. (5.20) in Eq. (5.35), we obtain

$$\beta_c \approx \frac{1}{\sqrt{m\tau}} \ln \left(1 + \frac{e^\tau - 1}{2\sqrt{ma}} \right). \quad (5.48)$$

Equation (5.48) agrees with the value derived in [33]. Note that this approximation is valid only for small τ ($\tau < 1/\beta_c$).

5.7 Derivation of τ_* for general activity distributions

In the limit $\beta \rightarrow \infty$, we obtain $c_i \rightarrow 1$ ($1 \leq i \leq 5$). For general activity distributions, $f(\tau_*, \beta_c \rightarrow \infty) = 0$ leads to

$$\tau_* = -\ln \left(1 - \frac{b + \sqrt{b^2 + 4d}}{2} \right), \quad (5.49)$$

where

$$\begin{aligned} b &= m\langle a \rangle^2 [1 - m\langle a \rangle]^{-3} [2 - (m+1)\langle a \rangle] S\left(\frac{\langle a \rangle}{1 - m\langle a \rangle}\right) \\ &+ m\langle a \rangle [1 - m\langle a \rangle]^{-2} [m+1 - (m^2+1)\langle a \rangle], \end{aligned} \quad (5.50)$$

$$\begin{aligned} d &= m^2\langle a \rangle^2 [1 - m\langle a \rangle]^{-3} [1 - (m+1)\langle a \rangle] S\left(\frac{\langle a \rangle}{1 - m\langle a \rangle}\right) \\ &- m^2\langle a \rangle^2 [1 - m\langle a \rangle]^{-2}. \end{aligned} \quad (5.51)$$

5.8 Derivation of m_c for general activity distributions

At $m = m_c$, an infinitesimal increase in τ from 0 to $\Delta\tau$ does not change the β_c value. For general activity distributions, by setting $\partial f / \partial \tau = 0$ for f given by Eq. (5.34), we obtain

$$m_c = \frac{1 + 2\frac{\sqrt{\langle a^2 \rangle}}{\langle a \rangle}}{1 - 2\sqrt{\langle a^2 \rangle} - 2\frac{\langle a^2 \rangle}{\langle a \rangle}}. \quad (5.52)$$

5.9 Activity-driven model with a reinforcement process

We carried out numerical simulations for an extended activity-driven model in which link dynamics are driven by a reinforcement process [41]. The original activity-driven model is memoryless [18]. In the extended model, an activated node i connects to a node j that i has already contacted with probability $1/(n_i + c)$ and to a node j that i has not contacted with probability $c/(n_i + c)$, where n_i denotes the number of nodes that node i has already contacted.

The numerically calculated prevalence is compared between the original model [Figs. 5.3(a) and 5.3(b)] and the extended model with $c = 1$ [Figs. 5.3(c) and

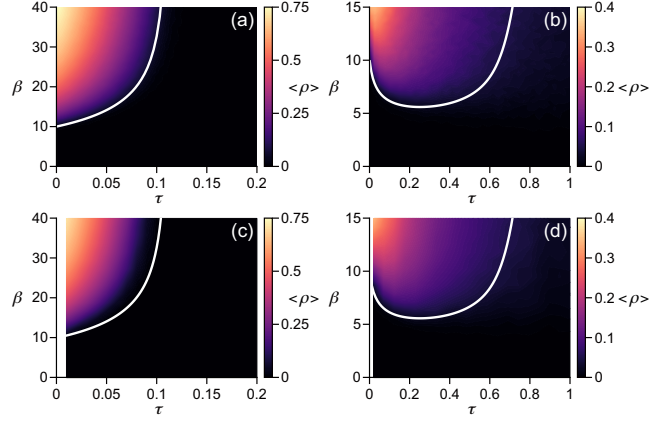


Figure 5.3: Epidemic threshold and numerically calculated prevalence for the activity-driven model with link dynamics driven by a reinforcement process [41]. We set $m = 1$ in (a) and (c), and $m = 10$ in (b) and (d). We used the original activity-driven model in (a) and (b) and the extended model with $c = 1$ in (c) and (d). The solid lines represent the epidemic threshold obtained from Eq. (3.8). All nodes have $a_i = 0.05$ ($1 \leq i \leq N$) in (a) and (c), and $a_i = 0.005$ ($1 \leq i \leq N$) in (b) and (d). We calculated the prevalence averaged over 100 simulations after discarding the first 15000 time steps in each simulation. We set $N = 2000$ and $\Delta t = 0.002$.

5.3(d)]. We replicate Figs. 3.1(a) and 3.1(b) in the main text as Figs. 5.3(a) and 5.3(b) as reference. All nodes are assumed to have the same activity potential $a_i = 0.05$ ($1 \leq i \leq N$) in (a) and (c) and $a_i = 0.005$ ($1 \leq i \leq N$) in (b) and (d). Figure 5.3 indicates that the extended model only slightly changes the epidemic threshold.

5.9.1 Stochastic m

We consider the case in which the strength of concurrency, m , is not constant. To analyze this case, we change the definitions of c'_1 , c'_2 , c'_3 , c'_4 , and c'_5 to

$$c''_1 = E[c_1 - e^{-\tau}], \quad (5.53)$$

$$c''_2 = E[mc_2], \quad (5.54)$$

$$c''_3 = E[e^{-\tau} + m\langle a \rangle(c_3 - e^{-\tau})], \quad (5.55)$$

$$c''_4 = E[mc_4], \quad (5.56)$$

$$c''_5 = E[m(m-1)\langle a \rangle c_5], \quad (5.57)$$

where $E[\cdot]$ is the expectation with respect to the distribution of m . The mean degree is given by $\langle k \rangle = 2aE[m]$. Using Eqs. (5.53)–(5.57) instead of c'_i ($1 \leq i \leq 5$),

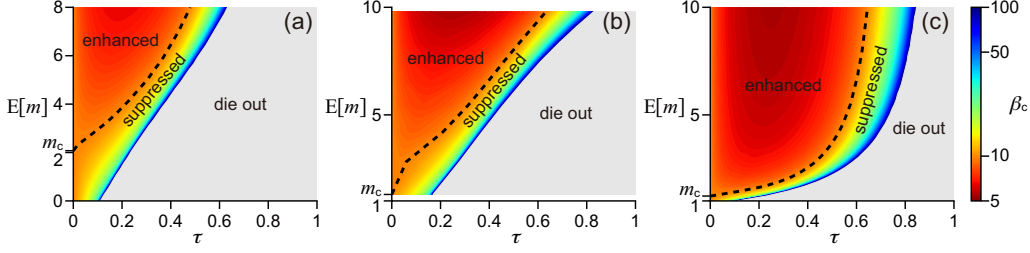


Figure 5.4: Phase diagram of the epidemic threshold when m is stochastic; m obeys (a) a truncated Poisson distribution ($0 \leq m \leq m_{\max}$) and (b) a power-law distribution with exponent 3 ($m_{\min} \leq m \leq m_{\max}$), and (c) a bimodal distribution in which m takes m_1 and m_2 with probabilities $\tilde{p}$ and $1 - \tilde{p}$, respectively. In (a) and (b), we set $m_{\max} = 11$. In (a), we truncated a Poisson distribution with varying the mean between 0.01 and 8 to modulate $E[m]$. In (b), We vary $m_{\min}$ between 1 to 9. In (c), we set $(m_1, m_2) = (10, 1)$ and varied $\tilde{p}$ to modulate $E[m]$. We set $\langle k \rangle = 0.1$. The dashed line represents τ_c . In the gray regions, $\beta_c > 100$.

we derived the epidemic threshold in the same manner as the derivation of Eq. (3.8). The phase diagrams of the epidemic threshold when m obeys a truncated Poisson distribution and a power-law distribution are shown in Figs. 5.4(a) and 5.4(b), respectively. We obtain $\beta_c = 1/\langle k \rangle$ at $\tau = 0$. We set the activity potential of all nodes $a = \langle k \rangle / (2E[m])$ such that the epidemic threshold is the same for all $E[m]$ at $\tau = 0$. We numerically calculated m_c at which $\tau_c = 0$. For the power-law distribution of m , we cannot make $E[m]$ smaller than m_c because the distribution does not have a probability mass at $m = 0$ by definition. However, the phase diagrams in the case of both the truncated Poisson and power-law distributions of m are qualitatively similar to the case of constant m .

To gain analytical insights, we calculated the phase diagrams when m is equal to m_1 and m_2 with probabilities $\tilde{p}$ and $1 - \tilde{p}$, respectively. We varied $\tilde{p}$ between 0 and 1. Here again, we set the activity potential of all nodes $a = \langle k \rangle / (2E[m])$ such that the epidemic threshold is the same for all $E[m]$ at $\tau = 0$. The phase diagram [Fig. 5.4(c)] is again qualitatively similar to that found in the case of constant m .

5.10 Heterogeneous activity distributions

We analyzed the phase diagram for different distributions of activity potentials to confirm the robustness of the results shown in the main text. We consider an exponential distribution and a power-law distribution with exponent 2.5. We numerically calculate the epidemic threshold by solving Eq. (3.8) and derive τ_* and m_c from Eqs. (5.49) and (5.52), respectively. The phase diagrams for the

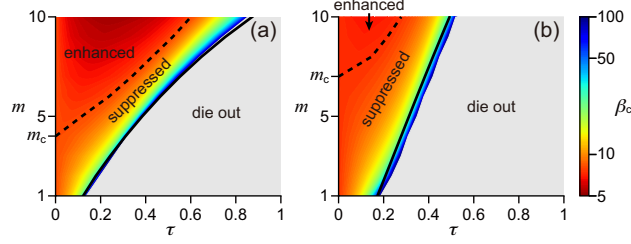


Figure 5.5: Phase diagram of the epidemic threshold when the activity potential obeys (a) an exponential distribution with a rate parameter λ ($0 \leq a_i \leq 0.9$) and (b) a power-law distribution with exponent 2.5 ($\epsilon \leq a_i \leq 0.9$). We set $\langle k \rangle = 0.1$ at $m = 1$ and adjust the value of λ and ϵ such that β_c takes the same value for all m at $\tau = 0$. The solid and dashed lines represent τ_* and τ_c , respectively. In the gray regions, $\beta_c > 100$.

exponential and power-law distributions are shown in Figs. 5.5(a) and 5.5(b), respectively. These results are qualitatively similar to those found when all nodes have the same activity potential value.

5.11 Temporal networks composed of cliques

We consider the case in which an activated node creates a clique (a fully-connected subgraph) with m randomly chosen nodes instead of a star graph. This situation models a group conversation among $m + 1$ people. We only consider the case in which all nodes have the same activity potential a . The mean degree for a network in a single time window is given by $\langle k \rangle = m(m + 1)a$. The aggregate network is the complete graph. We impose $m^2 a \ll 1$ so that cliques in the same time window do not overlap.

As in the case of the activity-driven model, we denote the state of a clique by $\{x, y, z\}$ ($x, y \in \{S, I\}, 0 \leq z \leq m - 1$), where x and y are the states of the activated node and another specific node, respectively, and z is the number of infected nodes in the other $m - 1$ nodes. The transition rate matrix of the SIS dynamics on this temporal network model is given as follows. The rates of the recovery events are given by Eqs. (5.3), (5.4), and (5.5). The rates of the infection

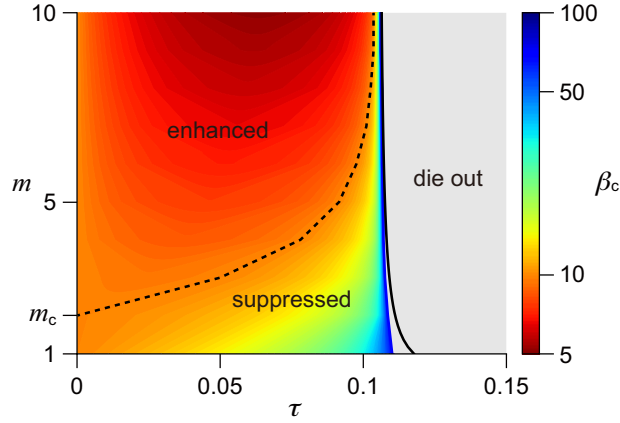


Figure 5.6: Phase diagram of the epidemic threshold for temporal networks composed of cliques. The solid and dashed lines represent τ_* [Eq. (5.67)] and τ_c , respectively. All nodes are assumed to have the same activity potential given by Eq. (5.69). We set $\langle k \rangle = 0.1$.

events are given by

$$M_{\{I,S,z\},\{S,S,z\}} = z\beta, \quad (5.58)$$

$$M_{\{S,I,z\},\{S,S,z\}} = z\beta, \quad (5.59)$$

$$M_{\{I,I,z\},\{S,I,z\}} = (z+1)\beta, \quad (5.60)$$

$$M_{\{I,I,z\},\{I,S,z\}} = (z+1)\beta, \quad (5.61)$$

$$M_{\{S,S,z+1\},\{S,S,z\}} = z(m-1-z)\beta \quad (z \leq m-2), \quad (5.62)$$

$$M_{\{I,S,z+1\},\{I,S,z\}} = (z+1)(m-1-z)\beta \quad (z \leq m-2), \quad (5.63)$$

$$M_{\{S,I,z+1\},\{S,I,z\}} = (z+1)(m-1-z)\beta \quad (z \leq m-2), \quad (5.64)$$

$$M_{\{I,I,z+1\},\{I,I,z\}} = (z+2)(m-1-z)\beta \quad (z \leq m-2). \quad (5.65)$$

We obtain c_i ($1 \leq i \leq 5$) from $\mathbf{M}$ in the same fashion as in the case of the activity-driven model. Because of the symmetry inherent in a clique, we obtain $c_1 = c_3$ and $c_2 = c_4 = c_5$. Therefore, Eq. (5.35) is reduced to

$$f(\tau, \beta_c) = 1 - q - (m+1)r = 0. \quad (5.66)$$

Calculations similar to the case of the activity-driven model lead to

$$\tau_* = \ln \frac{1 - (1 + m)a}{1 - (1 + m)^2 a} \approx \langle k \rangle, \quad (5.67)$$

$$m_c = 2. \quad (5.68)$$

The phase diagram shown in Fig. 5.6 is qualitatively the same as those for the activity-driven model (Fig. 3.2). Note that, in Fig. 5.6, we selected the activity potential value a to force β_c to be independent of m at $\tau = 0$, i.e.,

$$a = \frac{\langle k \rangle}{m(m + 1)}. \quad (5.69)$$

Although Eq. (5.67) coincides with the expression of τ_* for the activity-driven model [Eq. (3.10)], τ_* as a function of m is different between the activity-driven model [a solid line in Fig. 3.2(a)] and the present clique network model (a solid line in Fig. 5.6). This is because the values of a are different between the two cases when $m \geq 2$.

5.12 Empirical activity distributions

The epidemic threshold and prevalence when $F(a)$ is constructed from empirical contact data at a workplace, obtained from the SocioPatterns project [52], are shown in Figs. 5.7(a) and 5.7(b) for $m = 1$ and $m = 10$, respectively. The results for $F(a)$ constructed from email communication data at a research institution, obtained from the Stanford Network Analysis Platform [53], are shown in Figs. 5.7(c) and 5.7(d) for $m = 1$ and $m = 10$, respectively. These results are qualitatively similar to those shown in Fig. 3.1.

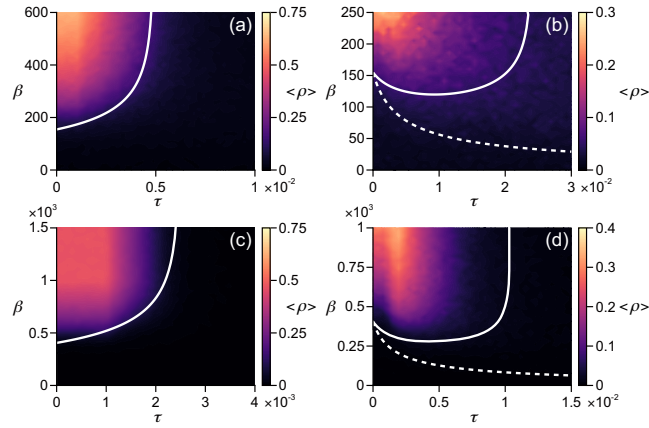


Figure 5.7: Results for activity potentials derived from empirical data. The epidemic threshold and numerically simulated prevalence are shown for $m = 1$ (a),(c) and $m = 10$ (b),(d). In (a) and (b), the activity potential is constructed from contact data obtained from the SocioPatterns project [52]. This data set contains contacts between pairs of $N = 92$ individuals measured every 20 seconds. In (c) and (d), the activity potential is constructed from email communication data at a research institution, obtained from the Stanford Network Analysis Platform [53]. Although the original edges are directed, we treat them as undirected. We assume that each email exchange event corresponds to a one-minute contact. We calculate the degree of each node per minute averaged over time, denoted by $\langle k_i \rangle$, and define the activity potential as $a_i = [\langle k_i \rangle - \langle k \rangle / 2] / m$. In (c) and (d), we used $N = 439$ individuals satisfying $a_i > 0$ (some individuals exchanged few emails such that $a_i < 0$). We set $\Delta t = 0.001$.

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