Spreading of infection on temporal networks: an edge-centered perspective

Andreas Koher, James P. Gleeson, Philipp Hövel

Abstract We discuss a continuous-time extension of the contact-based (CB) model, as proposed in [Koher et al. arXiv:1811.05809], for infections with permanent immunity on temporal networks. At the core of our methodology is a fundamental change to an edge-centered perspective, which allows for an accurate model on temporal networks, where the underlying time-aggregated graph has a tree structure. From the continuous-time CB model, we derive the infection propagator for the low prevalence limit and propose a novel spectral criterion to estimate the epidemic threshold. In addition, we explore the relation between the continuous-time CB model and the previously proposed edge-based compartmental model, as well as the message-passing framework.

Key words: Epidemic spreading, temporal networks, epidemic threshold, infection propagator, spectral radius, non-backtracking matrix

1 Introduction

The foundation of modern theoretical epidemiology was established at the beginning of the 20th century, mainly by health physicians such as Ross, Hamer, McKendrick, and Kermack who introduced the *compartment model* [1, 2, 3]. This ap-

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proach separates individuals within a population into epidemic categories or compartments, depending on their health status such as susceptible, infected, and recovered. Since the early years, development in the field of mathematical epidemiology has accelerated, not least due to the seminal works of Bailey [4], Anderson & May [5] and Hethocote [6]. Modern models include stochasticity [7, 8, 9, 10], non-Markovian dynamics [11, 12, 13, 14, 15], demographic structures, vaccinations, disease vectors and quarantine (see references in [6] for details). Thus, the field of research ranges from simple explanatory models that reveal hidden patterns and reproduce fundamental observations to elaborate numerical models that provide realistic predictions [16].

In recent years, we witnessed a second *golden age* [17] of epidemiological modeling. The driving forces behind this development are increasing computing power and an unprecedented amount of mobility data. The combination of both allows scientists to simulate the behavior of entire populations at the level of single individuals [18, 19, 20, 21, 22, 23, 24] and thus to advise policy makers by means of quantitative models.

One of the cornerstones of network-based disease models is the *individual-based* (IB) approach. It is a drastic simplification of the exact description using a master equation, because it assumes that the epidemiological states of neighboring nodes are statistically independent. Under this approximation, one can define a set of dynamic equations for the marginal probability to find a node in a given disease state [10, 25, 26, 27, 28, 29, 30, 31]. This method is widely employed, because it offers an intuitive and analytically tractable approach to integrate the underlying contact network. As a particularly important result, we mention that the largest eigenvalue of the adjacency matrix, which represents the topology of the network, determines the critical disease parameters that separate local and global outbreaks [28, 26].

Karrer & Newman substantially improved previous models of uni-directional diseases, such as the generic susceptible-infected-recovered (SIR) model, using the message-passing framework [13]. This approach dates back to the computer scientist Pearl [32], who formulated an exact inference algorithm on trees. Karrer & Newman proposed an integro-differential equation as a model for non-Markovian disease dynamics and improved previous estimates of the critical disease parameters on static networks [33]. A crucial conceptual difference to earlier works is that edges instead of nodes appear as central elements of the model. This idea has influenced considerably further research on network epidemiology [12, 34, 35, 36, 37].

The *dynamic message-passing* model [35] is a particularly application-oriented variant for Markovian SIR dynamics and has been extended recently to networks with time-varying topologies [37]. This novel approach for epidemics on temporal networks, termed the *contact-based* (CB) model, focuses on edge-based quantities that are updated in discrete time and thus allows for a seamless integration of temporal networks that are sampled at a constant rate. Importantly, the authors in [37] derive a critical condition that improves previous estimates of the epidemic threshold [26], which is an valuable risk measure for public health institutions.

Another important research branch in theoretical epidemiology focuses not on a single realization of a graph but on an ensemble of random networks. A particularly

accurate and compact model of epidemic spreading on this class of random networks has been proposed in [34] and is termed *edge-based compartmental* (EBC) model. The original work focused not only on the configuration model for static networks, but also on different classes of random graphs with time-varying topologies. Since then, several extensions have been proposed, such as non-Markovian recovery dynamics [12] and arbitrary initial conditions [38]. Moreover, studies in [39, 40] investigated links to other existing models such as *pair-approximations* [41], the *effective degree model* [42] and *message-passing* [13].

In this chapter, we will derive a continuous-time formulation of the CB model for temporal networks, analyze the low-prevalence limit and explore links to previously proposed models. To this end, we will briefly summarize in Sec. 2 the discrete-time version proposed in [37]. Then, we extend the dynamic equations to the continuous-time case in Sec. 3 and determine in Sec. 4 the epidemic threshold from a stability analysis of the disease-free fixed point. Moreover, we will link the continuous-time results to existing models and in particular to the *edge-based compartmental model* in Sec. 5 and the *message-passing* framework in Sec. 6.

2 Discrete-time description

The dynamic equations of the contact-based model appeared first in [35] and [37] for static and temporal networks, respectively. In the following, we will briefly rederive the discrete-time model, which will then serve as the starting point for a continuous-time formulation in the main part of this chapter.

We begin by introducing our notational convention and consider a network $G(t)$, whose topology can change at any time $t \in [0,T]$. Next we sample T_s snapshots of the graph with a constant interval Δt. The resulting sequence $[G_0, G_1, ..., G_{T_s-1}]$ is an approximation of the continuous-time network, which approaches the exact representation in the limit $\Delta t \to 0$.

Let us denote with $\mathscr{N}$ and $\mathscr{C} \subset \mathscr{T} \times \mathscr{N} \times \mathscr{N}$ the set of all nodes ($|\mathscr{N}| = N$) and time-resolved contacts, respectively, where $\mathscr{T} = \{0, 1, \ldots, T_s - 1\}$ represents the set of sampling times. To emphasize the difference between temporal and static elements, we will refer to edges as static links $(k,l) \in \mathscr{E} \subset \mathscr{N} \times \mathscr{N}$ of the time-aggregated graph and denote the number of edges with $E = |\mathscr{E}|$. In other words, an edge exists if and only if at least one contact was recorded between the corresponding nodes. We assume a directed network and hence represent a potential undirected contact through two reciprocal elements. Finally, it is helpful to define an indicator function that returns whether or not a contact from k to l exists at sampling time t:

$$a_{k\to l}(t) = \begin{cases} 1, & \text{if } (t,k,l) \in \mathscr{C} \\ 0, & \text{otherwise.} \end{cases} \tag{1}$$

Here we use the notation $k \to l$ to denote quantities defined on the set of edges $\mathscr{E}$, thus preventing potential confusion with node-based elements.

As a model for disease spreading, we consider the generic susceptible-infected-recovered (SIR) dynamic as a paradigmatic model for infections that lead to permanent immunity. In this modeling framework, a susceptible node (S) contracts the disease from an infected neighbor (I) with a constant and uniform rate β. The transition to the recovered state (R) follows with a likewise universal rate μ.

After this formal introduction we can now start with the actual modeling and to this end, we begin with the marginal probability $P_l^S(t)$ that node l is susceptible at time t. We observe that l is susceptible if it has been so already at the beginning $t = 0$ (with a corresponding probability of z_l) and hence did not contract the infection from any of its neighbors up to the observation time t. We denote the probability of the latter event with $\Phi_l(t)$, which leads to the following relation:

$$P_l^S(t) = z_l \Phi_l(t). \tag{2}$$

In order to determine the central quantity $\Phi_l(t)$, we make the simplifying assumption that the undirected, time-aggregated graph has a tree structure. In other words, ignoring the directionality, the static backbone does not contain loops and hence all branches emanating from the root node l can be considered independently of each other.

In order to factorize the probability $\Phi_l(t)$, we also need to introduce a concept that is sometimes referred to as *test node* [34], *cut-vertex* [43], or *cavity state* [13, 35]. To understand why this concept is helpful, imagine the case that a disease appears in one branch and hence may spread into another branch via the root node l. As a consequence, the probability that l will be infected by either of the two infected neighbors is clearly correlated and therefore cannot factorize. However, this case requires l to be already infected and hence appears as an artefact. In order to exclude this event we remove (virtually) all edges emanating from l, which prevents a disease transmission from one branch to another. We refer to vertex l as being in the *cavity state* or simply a *cavity node*. This intervention does not change the dynamics of l, as the node can still be infected and once it is, it recovers regardless of the network structure. Furthermore, we call this modification *virtual* because we restore the topological modification as soon as we focus on the dynamics of another node. This method ensures that $\Phi_l(t)$ factorizes and thus we arrive at

$$P_l^S(t) = z_l \prod_{k \in \mathscr{N}_l} \theta_{k \to l}(t). \tag{3}$$

Here, the product iterates through all neighbors $k \in \mathscr{N}_l$ of node l and with $\theta_{k\to l}(t)$ we denote the probability that cavity node l has not contracted the disease from k up to the observation time.

The conceptual change from a node-based to an edge-based modeling approach requires new auxiliary dynamic variables. Besides $\theta_{k\to l}(t)$, we will introduce additional quantities that are defined on the set of edges $\mathscr{E}$ and following our convention, we use the index notation *source* $\to$ *target*. In order to avoid repetition, we also note that in all cases the target node is considered to be in the cavity state.

To set up a dynamic equation for $\theta_{k\to l}(t)$, we observe that the value can only decrease precisely when (i) a contact indicated by $a_{k\to l}(t)$ exists and (ii) the source node k is infected and has not yet transmitted the disease to l. We denote the corresponding probability for event (ii) with $I_{k\to l}(t)$. Together with the probability $\beta\Delta t$ to contract the disease within the time step Δt, we obtain our first, discrete-time dynamic equation:

$$\theta_{k\to l}(t+\Delta t) = \theta_{k\to l}(t) - \beta\Delta t a_{k\to l}(t) I_{k\to l}(t). \tag{4}$$

As the initial condition we choose $\theta_{k\to l}(t) = 1$ for all edges $(k,l) \in \mathscr{E}$.

Next, we investigate $I_{k\to l}(t)$ and observe that the value can change due to three independent events: (i) Node k recovers, with probability $\mu\Delta t$; (ii) Node l contracts the disease from k upon a contact, with probability $\beta\Delta t$, whereby both events, i.e. (i) and (ii), can also occur simultaneously with probability $\beta\mu(\Delta t)^2$; (iii) Source node k is newly infected by one of its incident neighbors, excluding the cavity node l with probability $-\Delta S_{k\to l}(t) = S_{k\to l}(t+\Delta t) - S_{k\to l}(t)$. Here, $S_{k\to l}(t)$ denotes the probability to find k in the susceptible state. Balancing all probabilities, we obtain the following dynamic equation:

$$I_{k\to l}(t+\Delta t) = (1-\mu\Delta t)[1-\beta\Delta t a_{k\to l}(t)] I_{k\to l}(t) - \Delta S_{k\to l}(t). \tag{5}$$

The initial condition is given by $I_{k\to l}(0) = 1 - z_k$ for all edges.

We determine the probability $S_{k\to l}(t)$ in the same manner as Eq. (2), i.e., we find that k is susceptible if (i) it has been initially with probability z_k and (ii) with probability $\Phi_{k\to l}$ no pathogens were transmitted from one of its neighbors $j \in \mathscr{N}_k \setminus \{l\}$, excluding the cavity node l. Hence, we find $S_{k\to l}(t) = z_k \Phi_{k\to l}$. Moreover, the authors in [44] demonstrated that $\Phi_{k\to l}$ factorizes under the assumption of a tree topology and thus, similar to Eq. (3), we obtain:

$$S_{k\to l}(t) = z_k \prod_{j\in\mathscr{N}_k\setminus\{l\}} \theta_{j\to k}(t). \tag{6}$$

We can now substitute Eq. (6) into Eq. (5) and together with Eq. (4) we thus obtain a closed system of $2E$ dynamical equations that determine the disease progression.

Finally, we return to node-centric quantities. To this end, we follow [13] and note first that Eq. (3) determines already the probability $P_l^S(t)$ that node l is susceptible at the time t. Then, we obtain the corresponding probability $P^I(t)$ for the infected state from the conservation condition, i.e., a node can assume only one of the three possible states $X \in \{S,I,R\}$:

$$P_l^I(t) = 1 - P_l^S(t) - P_l^R(t). \tag{7}$$

The remaining marginal probability $P^R(t)$ can only increase due to a transition from the infected to the recovered state, which is given by $\mu\Delta t P^I(t)$. Hence, the third node-centric equation reads

$$P_l^R(t+\Delta t) = P_l^R(t) + \mu \Delta t P_l^I(t). \tag{8}$$

After this brief review of the discrete-time case that has been first derived in [37], we will elaborate on a continuous-time version next.

3 Continuous-time description

In the continuous-time limit $\Delta t \to 0$, Eq. (4) leads to

$$\frac{\mathrm{d}}{\mathrm{d}t}\theta_{k\to l}(t) = -\beta a_{k\to l}(t) I_{k\to l}(t). \tag{9}$$

We focus on $S_{k\to l}(t+\Delta t)$ from Eq. (6) and using the definition of $\theta_{j\to k}(t+\Delta t)$ (cf. Eq. (4)), we obtain:

$$S_{k\to l}(t+\Delta t) = z_k \prod_{j\in\mathcal{N}_k\setminus\{l\}} \theta_{j\to k}(t+\Delta t) \tag{10a}$$

$$= z_k \prod_{j\in\mathcal{N}_k\setminus\{l\}} \left[\theta_{j\to k}(t) - \beta\Delta t a_{k\to l}(t) I_{j\to k}(t)\right]. \tag{10b}$$

For a sufficiently small sampling interval Δt, such that $\theta_{j\to k}(t) \gg \beta\Delta t a_{k\to l}(t) I_{k\to l}(t)$, we can linearize Eq. (10b) and thus arrive at:

$$S_{k\to l}(t+\Delta t) = z_k \prod_{j\in\mathcal{N}_k\setminus\{l\}} \theta_{j\to k}(t) \cdot \left[1 - \sum_{j'\in\mathcal{N}_k\setminus\{l\}} \frac{\beta\Delta t a_{k\to l}(t) I_{j'\to k}(t)}{\theta_{j'\to k}(t)}\right] \tag{11a}$$

$$S_{k\to l}(t+\Delta t) = S_{k\to l}(t)\left[1 - \beta\Delta t \sum_{j'\in\mathcal{N}_k\setminus\{l\}} a_{k\to l}(t)\frac{I_{j'\to k}(t)}{\theta_{j'\to k}(t)}\right] \tag{11b}$$

In Eq. (11b) we inserted the definition of $S_{k\to l}(t)$ from Eq. (6) and this leads directly to our second continuous-time dynamic equation

$$\frac{\mathrm{d}}{\mathrm{d}t}S_{k\to l}(t) = -\beta S_{k\to l}(t) \sum_{j\in\mathcal{N}_k\setminus\{l\}} a_{k\to l}(t)\frac{I_{j\to k}(t)}{\theta_{j\to k}(t)}. \tag{12}$$

The quotient $I_{j\to k}(t)/\theta_{j\to k}(t)$ can be interpreted as the conditional probability that j is infected *given* that cavity node k has not yet contracted the disease from j. It is worth noting that Eq. (12) is well defined because we start from the initial condition $\theta_{j\to k}(t) = 1$ for all edges $k \to j$ and Eq. (9) asserts positivity for $\theta_{j\to k}(t)$ for all finite observation times t. The remaining discrete-time Eq. (5) can be immediately written down in terms of a difference quotient $\Delta X(t) = [X(t+\Delta t) - X(t)]/\Delta t$:

$$\frac{\Delta I_{k\to l}(t)}{\Delta t} = \left[-\mu - \beta a_{k\to l}(t) + \mu\beta\Delta t a_{k\to l}(t)\right] I_{k\to l}(t) - \frac{\Delta S_{k\to l}(t)}{\Delta t}. \tag{13}$$

In the continuous-time limit, the higher order term $\mu\beta\Delta t a_{k\to l}(t)$ vanishes, leading to

$$\frac{\mathrm{d}}{\mathrm{d}t}I_{k\to l}(t) = [-\mu - \beta a_{k\to l}(t)]I_{k\to l}(t) - \frac{\mathrm{d}}{\mathrm{d}t}S_{k\to l}(t). \tag{14}$$

At last it is also instructive to formulate the dynamic equation for $R_{k\to l}(t)$, i.e., the probability that node k has recovered at time t without transmitting the disease to cavity node l. The value of $R_{k\to l}(t)$ can only increase over time and the corresponding in-flux at time t is given by (i) the probability $I_{k\to l}(t)$ that node k is in state I and has not infected its neighbor l together with (ii) the probability $\mu\Delta t$ to recover within the time step Δt. With this, we obtain:

$$R_{k\to l}(t+\Delta t) = R_{k\to l}(t) + \mu\Delta t I_{k\to l}(t). \tag{15}$$

The corresponding continuous-time equation thus reads

$$\frac{\mathrm{d}}{\mathrm{d}t}R_{k\to l}(t) = \mu I_{k\to l}(t). \tag{16}$$

Unlike the discrete-time model, it is now obvious that the dynamic Eqs. (9), (12), (14), and (16) satisfy the conservation condition

$$\theta_{k\to l}(t) = S_{k\to l}(t) + I_{k\to l}(t) + R_{k\to l}(t) \tag{17}$$

at every time t. Moreover, we can rescale time according to $\mu t \mapsto t$ and rewrite the continuous-time contact-based model in terms of the dimensionless epidemic parameter $\gamma = \beta/\mu$:

$$\frac{\mathrm{d}}{\mathrm{d}t}\theta_{k\to l}(t) = -\gamma a_{k\to l}(t) I_{k\to l}(t) \tag{18a}$$

$$\frac{\mathrm{d}}{\mathrm{d}t}S_{k\to l}(t) = -\gamma S_{k\to l}(t) \sum_{j\in\mathcal{N}_k\setminus\{l\}} a_{j\to k}(t)\frac{I_{j\to k}(t)}{\theta_{j\to k}(t)} \tag{18b}$$

$$\frac{\mathrm{d}}{\mathrm{d}t}I_{k\to l}(t) = -[1+\gamma a_{k\to l}(t)]I_{k\to l}(t) - \frac{\mathrm{d}}{\mathrm{d}t}S_{k\to l}(t) \tag{18c}$$

$$\frac{\mathrm{d}}{\mathrm{d}t}R_{k\to l}(t) = I_{k\to l}(t). \tag{18d}$$

We can further reduce the set of dynamic equations using the conservation condition in Eq. (17). To this end, we first substitute $S_{k\to l}(t)$ in Eq. (17) with the definition from Eq. (6):

$$I_{k\to l}(t) = \theta_{k\to l}(t) - z_k \prod_{j\in\mathcal{N}_k\setminus\{l\}} \theta_{j\to k}(t) - R_{k\to l}(t). \tag{19}$$

With this, we replace $I_{k\to l}(t)$ in Eq. (18a) and Eq. (18d) and thus we obtain a closed set of $2E$ dynamic equations that determine the disease progression of the continuous-time CB model.

Returning to node-centric quantities, i.e., the probability that a given node l is susceptible, infected or recovered, the continuous-time equivalent formulation to Eqs. (3), (7), and (8) reads

$$P_l^S(t) = z_l \prod_{k \in \mathcal{N}_l} \theta_{k\to l}(t) \tag{20a}$$

$$P_l^I(t) = 1 - P_l^S(t) - P_l^R(t) \tag{20b}$$

$$\frac{\mathrm{d}}{\mathrm{d}t} P_l^R(t) = P_l^I(t). \tag{20c}$$

4 Spectral properties of the continuous-time model

In this section, we evaluate the low prevalence limit of Eqs. (18) in order to derive a spectral criterion that determines the epidemic threshold. To this end, we assume $\theta_{k\to l}(t) = 1 - \delta_{k\to l}(t)$, where $\delta_{k\to l}(t) \ll 1$ as well as $I_{k\to l} \ll 1$. With this, we linearize Eq. (18b) and obtain

$$\frac{\mathrm{d}}{\mathrm{d}t} S_{k\to l}(t) = -\gamma \left[1 - \sum_{j \in \mathcal{N}_k \setminus \{l\}} \delta_{j\to k}(t) \right] \cdot \sum_{j \in \mathcal{N}_k \setminus \{l\}} a_{k\to l}(t) \frac{I_{j\to k}(t)}{1 - \delta_{k\to l}(t)} \tag{21a}$$

$$= -\gamma \sum_{j \in \mathcal{N}_k \setminus \{l\}} a_{k\to l}(t) I_{j\to k}(t). \tag{21b}$$

In Eq. (21b), we keep only linear terms in $\delta_{k\to l}(t)$ and $I_{j\to k}(t)$. This allows us to decouple the set of dynamic equations and express Eq. (18c) only in terms of $I_{k\to l}(t)$:

$$\frac{\mathrm{d}}{\mathrm{d}t} I_{k\to l}(t) = [-1 - \gamma a_{k\to l}(t)] I_{k\to l}(t) + \gamma \sum_{j \in \mathcal{N}_k \setminus \{l\}} a_{k\to l}(t) I_{j\to k}(t). \tag{22}$$

Next, we vectorize Eq. (22) and to this end, we define the vectors $\boldsymbol{I}(t)$ and $\boldsymbol{a}(t)$ with elements $I_{k\to l}(t)$ and $a_{k\to l}(t)$, respectively. In order to rewrite $\sum_{j \in \mathcal{N}_k \setminus l} a_{j\to k}(t) I_{k\to l}(t)$ from Eq. (22) in terms of a matrix that acts on the state vector $\boldsymbol{I}(t)$, we introduce the time-dependent non-backtracking operator $\boldsymbol{B}(t)$ as in [37]:

$$B_{k\to l, j\to k'}(t) = \begin{cases} a_{j\to k'}(t), & \text{if } k' = k, \text{ and } j \neq l \\ 0, & \text{otherwise.} \end{cases} \tag{23}$$

Expressed in words, we find $B_{k\to l, j\to k'}(t) = 1$ if the contact (t, j, k') is incident on the edge (k, l), implying $k' = k$, and additionally $j \neq l$. The latter constraint prevents a probability flow back to the initially infected node and constitutes the non-backtracking property. In all other cases, we find $B_{k\to l, j\to k'}(t) = 0$. Unlike the static definition in [45, 33], we have to differentiate between the first and second index of the $L \times L$ dimensional matrix $\boldsymbol{B}$: The first index, i.e. $(k, l) \in \mathscr{E}$, corresponds to an

edge in the aggregated graph, thus reflecting a potential path for future infections. The second index $(t, j, k') \in \mathscr{C}$, however, is a (temporal) contact from node j to k' at time t.

Moreover, we define the diagonal matrix $\mathrm{diag}(\boldsymbol{a}(t))$ with elements $a_{k\to l}(t)$ for all edges $(k,l) \in \mathscr{E}$ on the diagonal and, additionally, we denote with $\mathbb{1}$ the identity matrix. Similar to the discrete-time derivation in [37], we thus obtain:

$$\frac{\mathrm{d}}{\mathrm{d}t}\boldsymbol{I}(t) = [-\mathbb{1} - \gamma\,\mathrm{diag}(\boldsymbol{a}(t)) + \gamma\boldsymbol{B}(t)]\,\boldsymbol{I}(t). \tag{24}$$

The only structural difference to the discrete-time result in [37] is that the higher order term $\beta a_{k\to l}\mu$ does not appear, because the simultaneous event of infection and recovery does not need to be accounted for in the continuous-time formulation.

Within the open interval $[t_n, t_{n+1})$ where the boundaries t_n and t_{n+1}, respectively, mark subsequent change points of the network topology, we integrate Eq. (24) and obtain

$$\boldsymbol{I}(t_{n+1}) = \boldsymbol{M}_n(\gamma)\boldsymbol{I}(t_n) \tag{25a}$$

$$\boldsymbol{M}_n(\gamma) = \exp\left(\int_{t_n}^{t_{n+1}} \mathrm{d}\tau[-\mathbb{1} - \gamma\,\mathrm{diag}(\boldsymbol{a}(\tau)) + \gamma\boldsymbol{B}(\tau)]\right). \tag{25b}$$

Using the initial condition $\boldsymbol{I}(0)$, we can formally state the explicit solution as follows:

$$\boldsymbol{I}(T) = \prod_{n=0}^{N_G-1} \boldsymbol{M}_n(\gamma)\boldsymbol{I}(0). \tag{26}$$

Here, N_G is the total number of discrete changing points of the network topology. Following [46] we can state the propagator $\boldsymbol{M}(\gamma) = \prod_{n=0}^{N-1}\boldsymbol{M}_n(\gamma)$ in a compact notation using Dyson's time ordering operator $\mathsf{T}\boldsymbol{B}(\tau_1)\boldsymbol{B}(\tau_2) = \boldsymbol{B}(\tau_1)\boldsymbol{B}(\tau_2)\Theta(\tau_1-\tau_2) + \boldsymbol{B}(\tau_2)\boldsymbol{B}(\tau_1)\Theta(\tau_2-\tau_1)$, where $\Theta(x)$ denotes the Heaviside function:

$$\boldsymbol{M}(\gamma) = \mathsf{T}\exp\left(\int_0^t \mathrm{d}\tau\,[-\mathbb{1} - \gamma\,\mathrm{diag}(\boldsymbol{a}(\tau)) + \gamma\boldsymbol{B}(\tau)]\right). \tag{27}$$

Any small initial perturbation will decrease exponentially if the largest eigenvalue λ_1, i.e., the spectral radius satisfies $\lambda_1[\boldsymbol{M}(\gamma)] < 1$. This result corresponds to [46] where the epidemic propagator $\boldsymbol{M}(\gamma)$ has been derived within the IB framework and reads

$$\boldsymbol{M}(\gamma) = \mathsf{T}\exp\left(\int_0^t \mathrm{d}\tau\,[-\mathbb{1} + \gamma\boldsymbol{A}(\tau)]\right). \tag{28}$$

In Eq. (28), we denote with $\boldsymbol{A}(\tau)$ the time-dependent adjacency matrix and here, $\mathbb{1}$ is the $N \times N$ dimensional identity matrix.

In many cases, the temporal network is sampled with equidistant time steps Δt and in this case, we can simplify the propagator Eq. (28) to

$$\boldsymbol{M}(\gamma) = \prod_{n=0}^{N_G-1} \exp\left(\Delta t\left[-\mathbb{1} - \gamma \mathrm{diag}(\boldsymbol{a}(n)) + \gamma \boldsymbol{B}(t_n)\right]\right). \tag{29}$$

The CB result in Eq. (29) is akin to the IB formulation that was first derived in [47]. In the *quenched limit*, when the disease evolves on a much faster time scale than the temporal network, we can assume a static underlying topology and thus identify $\mathrm{diag}(\boldsymbol{a}(t)) \equiv \mathbb{1}$ and $\boldsymbol{B}(t) \equiv \boldsymbol{B}(1) \equiv \boldsymbol{B}$. Then, the linearized result in Eq. (24) simplifies to

$$\frac{\mathrm{d}}{\mathrm{d}t}\boldsymbol{I}(t) = \left[-(1+\gamma)\mathbb{1} + \gamma \boldsymbol{B}\right]\boldsymbol{I}(t). \tag{30}$$

Finally, Eq. (30) is asymptotically stable if the largest eigenvalue λ_1 of the infection operator $\boldsymbol{M}(\gamma) = (1+\gamma)\mathbb{1} + \gamma\boldsymbol{B}$ is negative. Hence, we recover the continuous-time threshold as previously derived within the more general message-passing framework on static networks [13, 33]:

$$\frac{\gamma}{\gamma+1} = \frac{1}{\lambda_1(\boldsymbol{B})}. \tag{31}$$

For non-Markovian infection and recovery processes the generalized criticality condition reads $T = 1/\lambda_1(\boldsymbol{B})$ (see [13, 33]) , where the transmissibility T is given by

$$T = \int_0^\infty s(\tau)\left(\int_\tau^\infty r(\tau')\mathrm{d}\tau'\right)\mathrm{d}\tau. \tag{32}$$

Intuitively, T can be interpreted as the probability that a newly infected node transmits the disease to a given neighbor prior to recovery [13, 48]. Within this general formulation $s(\tau)\mathrm{d}\tau$ is the probability that an infected node passes the disease to a neighbor within a time interval $[\tau, \tau+\mathrm{d}\tau]$ after contracting the infection. Similarly, we define the probability $r(\tau)\mathrm{d}\tau$ that a node recovers in the interval $[\tau, \tau+\mathrm{d}\tau]$ after it has been infected. For a constant infection and recovery rate, i.e., for the Markovian dynamics that we assumed in this article, we find $s(\tau) = \beta\exp(-\beta\tau)$ and $s(\tau) = \mu\exp(-\mu\tau)$. This particularly simple and widely studied choice then leads to $T = \gamma/(\gamma+1)$ and thus to Eq. (31).

For temporal networks, we cannot separate in general the transmissibility T from the network topology in order to find a similarly elegant results like Eq. (31). The reason is that the probability to infect a given neighbor depends on the timing of contacts and as a consequence the transmissibility T would have to be both edge- and time-dependent even in the Markovian case.

5 Relation to the edge-based compartmental model

An important branch in theoretical epidemiology focuses on random graphs, i.e., an ensemble of networks derived from a generating model, instead of a single realization. In this context, the *edge-based compartmental* (EBC) model [34] is a particularly compact and accurate approach to model infections with permanent immunity.

In this section, we will explore the relation between the CB model presented in Sec. 3 and the EBC framework for static random graphs.

To this end, we will focus on random networks with unweighted and undirected edges that are derived from the *configuration model* [49, 50]. This widely used generative model allows to study the effect of the degree distribution on the spread of infections [48]. For this, we have to create an ensemble of networks with the same degree distribution that are otherwise maximally random. This can be done according to the Bender-Canfield algorithm [49], which begins with a set of N vertices. To each node we assign a number of k (undirected) *stubs*, i.e., edges with no target node that are drawn independently from the given degree distribution $p(k)$. In the next step, we connect two randomly chosen stubs which then form a proper edge between the corresponding nodes. The step is repeated until no more stubs are available and if initially the number of stubs were found to be odd then the we would replace one node repeatedly until the sum is even.

Before we proceed with the ensemble average, we restate for convenience the relevant dynamic equations of the continuous-time model, i.e., Eq. (18a) and Eq. (18d), for a network with a static topology. In this case, $a_{k\to l} \equiv 1$ for all edges $(k,l) \in \mathscr{E}$ and thus we obtain

$$\frac{\mathrm{d}}{\mathrm{d}t}\theta_{k\to l}(t) = -\gamma I_{k\to l}(t) \tag{33a}$$

$$\frac{\mathrm{d}}{\mathrm{d}t}R_{k\to l}(t) = I_{k\to l}(t) \tag{33b}$$

and close the set of equations with the conservation condition from Eq. (19):

$$I_{k\to l}(t) = \theta_{k\to l}(t) - z_k \prod_{j\in\mathscr{N}_k\setminus\{l\}} \theta_{j\to k}(t) - R_{k\to l}(t). \tag{34}$$

For static networks, we can further simplify the dynamic equations by substituting $I_{k\to l}(t)$ in Eq. (33a) with Eq. (33b) and integrating the result:

$$\frac{\mathrm{d}}{\mathrm{d}t}R_{k\to l}(t) = -\frac{1}{\gamma}\frac{\mathrm{d}}{\mathrm{d}t}\theta_{k\to l}(t) \tag{35a}$$

$$R_{k\to l}(t) = \frac{1}{\gamma}(1-\theta_{k\to l}(t)). \tag{35b}$$

From Eq. (33a), Eq. (34), and Eq. (35b), we obtain a coupled set of E dynamic equations that determine the progression of an SIR epidemic on a static graph:

$$\frac{\mathrm{d}}{\mathrm{d}t}\theta_{k\to l}(t) = 1-(1+\gamma)\theta_{k\to l}(t) + \gamma z_k \prod_{j\in\mathscr{N}_k\setminus\{l\}} \theta_{j\to k}(t). \tag{36}$$

The result in Eq. (36) constitutes a message-passing equation as derived in [13]. We will explore the connection to the more general message-passing framework for epidemics with non-Markovian dynamics in Sec. 6. Here, we continue instead with the ensemble average over random networks, thereby following closely the approach

outlined in [13]. We start with the following crucial observation: The state of a given edge $k \to l$ in a single realization of a graph displays a characteristic trajectory in state space, i.e., a time-dependent curve given by $\theta_{k\to l}(t)$ from Eq. (36). As we perform an average over the ensemble of graphs, our selected edge $k \to l$ will assume every position within a network. As a consequence the averaged state trajectory is identical to the one that we would obtain if we had started with a different edge initially and then performed the average. In other words, it is sufficient to determine the ensemble averaged probabilities for *one representative edge*:

$$\frac{\mathrm{d}}{\mathrm{d}t}\theta(t) \equiv \frac{\mathrm{d}}{\mathrm{d}t}\langle\theta_{k\to l}(t)\rangle \tag{37a}$$

$$= 1-(1+\gamma)\theta(t)+\gamma\left\langle z_k \prod_{j\in\mathscr{N}_k\setminus\{l\}} \theta_{j\to k}(t)\right\rangle. \tag{37b}$$

Next, we focus on the second term in Eq. (37b). A crucial property of large networks that are generated by the configuration model is that they are locally tree-like in the sense that the average length of the smallest cycle diverges with increasing network size. Hence, we can assume in the limit $N \to \infty$ that different branches emerging from k can be treated independently. The average over the product thus equals the product over averages. Moreover, we remember that ensemble averaged dynamic quantities are equal for all edges and in particular $\theta_{j\to k}(t) \equiv \theta(t)$ for all edges $(j,k) \in \mathscr{N}_k \setminus \{l\}$. With this the product in Eq. (37b) simplifies to $[\theta(t)]^{k_e}$, where k_e is the average number of next nearest neighbors, or equally, the *excess degree* [13]. From a given degree distribution p_n in the configuration model, we can derive the excess degree distribution q_n according to $q_n = (n+1)p_{n+1}/k$ [50], where $k = \langle n\rangle$ denotes the average degree. Finally, we make use of the corresponding generating function $G_1(x) = \sum_n q_n x^n$ and thus the second term in Eq. (37b) simplifies to

$$\left\langle z_k \prod_{j\in\mathscr{N}_k\setminus\{l\}} \theta_{j\to k}(t)\right\rangle = z\sum_{n=0}^{N} q_n[\theta(t)]^n \tag{38a}$$

$$= zG_1(\theta(t)). \tag{38b}$$

Here, $z = \langle z_k\rangle$ denotes the probability that a randomly chosen node is initially susceptible. With Eq. (37b) and Eq. (38b), we obtain the following ensemble averaged dynamic equation for θ:

$$\frac{\mathrm{d}}{\mathrm{d}t}\theta(t) = 1-(1+\gamma)\theta(t)+\gamma z G_1(\theta(t)). \tag{39}$$

This compact result captures the disease dynamic with high accuracy as demonstrated in [13] within the message-passing framework and later in [34] as a special case of the edge-based compartmental model. The authors in [34] also investigated alternative random graph models with time-varying topologies.

We close the section with a linear stability analysis of Eq. (39). Similar to the derivation in Sec. 4, we start with a small initial perturbation: $\theta(t) = 1 - \delta(t)$ with $\delta(t) \ll 1$ and $z = 1$. We then expand the generating function $G_1(1-\delta(t))$ to the first order in $\delta(t)$:

$$G_1(1-\delta(t)) = \sum_n p_n (1-\delta(t))^n \tag{40a}$$

$$= 1 + \langle n \rangle_q (1-\delta(t)) + \mathscr{O}(\delta(t)^2). \tag{40b}$$

Here, we used two properties of the generating function, namely $G_1(1) = \sum_n q_n = 1$ and $G_1'(1) = \sum_n n q_n = \langle n \rangle_q$, where $\langle n \rangle_q = k_e$ denotes the mean excess degree. With this, the linearization of Eq. (39) around the disease-free stable fixed point reads:

$$\frac{\mathrm{d}}{\mathrm{d}t}\delta(t) = -(1 + \gamma - \gamma \langle n \rangle_q)\delta(t). \tag{41}$$

From Eq. (41) we can easily see that a transition occurs from local to global outbreaks if $1 + \gamma - \gamma \langle n \rangle_q < 0$. Commonly, $\langle n \rangle_q$ is expressed in terms of the first and second moment of the degree distribution, i.e. $\langle n \rangle = \sum_n n p_n$ and $\langle n^2 \rangle = \sum_n n^2 p_n$, respectively. For that we take the definition $\langle n \rangle_q = \sum_n n q_n$ and substitute the relation $q_n = (n+1)p_{n+1}/\langle n \rangle$ (see [50] for details). With this, we recover the well-known criticality condition from [48, 51]:

$$\frac{\gamma}{\gamma + 1} = \frac{\langle n \rangle}{\langle n^2 \rangle - \langle n \rangle}. \tag{42}$$

This result is related to the epidemic threshold in Eq. (31), where we studied a single realization of a static graph and hence expressed the right hand side of Eq. (42) through the spectral radius $\lambda_1(\boldsymbol{B})$ of the non-backtracking matrix $\boldsymbol{B}$.

6 Relation to the message-passing framework

In the seminal work of Karrer & Newman [13], the authors proposed a general model for SIR spreading processes on sparse networks with non-Markovian infection and recovery dynamics. The integro-differential formulation in [13] is a foundation of our CB model and therefore we will discuss in this section the relation to their message-passing approach. For that we first propose a generalization of the CB model to non-Markovian dynamics and then, taking the static network limit, we will arrive at the previously proposed result.

As a first step, we transform the dynamic equations in Eqs. (9), (12), (14), and (16) that define the continuous-time CB model to an integro-differential equation. To this end, we notice first that Eq. (14) is of the form

$$\frac{\mathrm{d}}{\mathrm{d}t} I_{k\to l}(t) = -\lambda_{k\to l}(t) I_{k\to l}(t) - \frac{\mathrm{d}}{\mathrm{d}t} S_{k\to l}(t). \tag{43}$$

For notational convenience, we use the short-hand notation $\lambda_{k\to l}(\tau)=\mu+\beta a_{k\to l}(\tau)$ and $\Lambda_{k\to l}(t,t_k)=\exp[-\int_{t_k}^{t}\lambda_{k\to l}(\tau)\mathrm{d}\tau]$. The former denotes the probability that node k recovers or infects the cavity node l within the time interval $[\tau,\tau+\mathrm{d}t)$ after contracting the disease and the latter corresponds to the probability that no such event took place between the time of infection and the observation time t_k and t, respectively. Here, we denote the absolute and relative time after infection with t and τ, respectively. Together with the initial condition $I_{k\to l}(0)=1-z_k$ the solution to the differential equation is given by

$$I_{k\to l}(t)=(1-z_k)\Lambda_{k\to l}(t,0)+\int_0^t\left[-\frac{\mathrm{d}}{\mathrm{d}t_k}S_{k\to l}(t_k)\right]\Lambda_{k\to l}(t,t_k)\mathrm{d}t_k. \tag{44}$$

In words, Eq. (44) states that node k has contracted the disease but not infected its neighbor l by absolute time t if (i) node k was infected initially but has neither recovered nor passed the infection or (ii) it was susceptible initially, contracted the disease at time t_k and has then neither recovered nor infected its neighbor up to the observation time t.

Next, we integrate Eq. (9) and using the initial condition $\theta_{k\to l}(0)=1$ we get

$$1-\theta_{k\to l}(t)=\int_0^t\beta a_{k\to l}(t')I_{k\to l}(t')\mathrm{d}t' \tag{45a}$$

$$=(1-z_k)\int_0^t\mathrm{d}t' f_{k\to l}(t'|0) \tag{45b}$$

$$+\int_0^t\int_0^{t'}\mathrm{d}t'\mathrm{d}t_k\left[-\frac{\mathrm{d}}{\mathrm{d}t_k}S_{k\to l}(t_k)\right]f_{k\to l}(t'|t_k).$$

In Eq. (45b) we used $I_{k\to l}(t)$ from Eq. (44) and we also introduced the transmission probability $f_{k\to l}(t'|t_k)=\beta a_{k\to l}(t')\Lambda_{k\to l}(t',t_k)$: Given that node k contracted the infection at absolute time t_k, $f_{k\to l}(t'|t_k)$ gives the probability that the same node passes the disease to its neighbor l at absolute time t'. In the context of static networks the quantity $\int_{t_k}^{\infty}f_{k\to l}(t'|t_k)\mathrm{d}t'$ is frequently referred to as *transmissibility* and plays a crucial role in linking epidemic spreading to a percolation process [13, 48]. Note that the transmissibility can be smaller than one as node k might recover before passing on the infection and for temporal networks, unlike the static case, the value is edge- and time-dependent as we discussed already at the end of Sec 4.

The message-passing framework in [13] assumes a non-Markovian infection and recovery process. Similarly, our result in Eq. (45b) demonstrates how a general epidemic model on *temporal networks* can be formulated by redefining $f_{k\to l}(t'|t_k)$ as proposed in [13] (see also Eq. (32)).

In order to demonstrate the reduction to the message-passing formulation of [13], we reformulate Eq. (45b) for a static underlying topology. With $a_{k\to l}(t)\equiv 1$ the transmission probability $f_{k\to l}(t|t_k)\to f(\tau)$ depends only on the relative time $\tau=t-t_k$ after infection and becomes an identical function for all edges $k\to l$. Using this simplification we obtain

$$1-\theta_{k\to l}(t) = (1-z_k)\int_0^t \mathrm{d}\tau f(\tau) + \int_0^t \int_0^\tau \mathrm{d}\tau \mathrm{d}\tau_i f(\tau-\tau_i)\left[-\frac{\mathrm{d}}{\mathrm{d}\tau_i} S_{k\to l}(\tau_i)\right]. \quad (46)$$

Integrating the second term in Eq. (46) by parts, and using the fact that the double integral can be reordered as

$$\int_0^t d\tau \int_0^\tau d\tau_i = \int_0^t d\tau_i \int_{\tau_i}^t d\tau, \quad (47)$$

we arrive at the message-passing formulation equivalent to that in [13]:

$$\theta_{k\to l}(t) = 1 - \int_0^t \mathrm{d}\tau f(t)(1 - S_{k\to l}(t-\tau)). \quad (48)$$

With this we have linked the continuous-time CB model with a previously introduced message-passing framework for general non-Markovian epidemic models in the case of a static underlying graph.

7 Summary

We have presented a continuous-time description of a contact-based model. The discussed theoretical framework allows us to study the spreading of epidemics and extends the dynamic message-passing approach to networks with a time-varying topology. At the center of the contact-based model is a shift in perspective from node- to edge-centric quantities. This allows to accurately model, e.g., susceptible-infected-recovered outbreaks on time-varying trees, that is, temporal networks with a loop-free underlying topology. We have shown that on arbitrary graphs, the proposed contact-based (edge-centric) model incorporates potential structural and temporal heterogeneities of the underlying contact network and improves analytic estimations with respect to the individual-based (node-centric) approach at a low computational and conceptual cost. Within this new framework, we have derived an analytical expression for the epidemic threshold on temporal networks.

We have taken a decidedly theoretical and analytical approach to the proposed framework. This will facilitate the application to both empirical data sets and generic classes of networks.

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