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Epidemic Threshold in Continuous-Time Evolving Networks

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Current understanding of the critical outbreak condition on temporal networks relies on approximations (time scale separation, discretization) that may bias the results. We propose a theoretical framework to compute the epidemic threshold in continuous time through the infection propagator approach. We introduce the *weak commutation* condition allowing the interpretation of annealed networks, activity-driven networks, and time scale separation into one formalism. Our work provides a coherent connection between discrete and continuous time representations applicable to realistic scenarios.

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Contagion processes, such as the spread of diseases, information, or innovations [1–5], share a common theoretical framework coupling the underlying population contact structure with contagion features to provide an understanding of the resulting spectrum of emerging collective behaviors [6]. A common keystone property is the presence of a threshold behavior defining the transition between a macroscopic-level spreading regime and one characterized by a null or negligibly small contagion of individuals. Known as the *epidemic threshold* in the realm of infectious disease dynamics [1], the concept is analogous to the phase transition in nonequilibrium physical systems [7,8], and is also central in social contagion processes [5,9–13].

A vast array of theoretical results characterize the epidemic threshold [14], mainly under the limiting assumptions of quenched and annealed networks [4,15–18], i.e., when the time scale of the network evolution is much slower or much faster, respectively, than the dynamical process. The recent availability of data on time-resolved contacts of epidemic relevance [19] has, however, challenged the time scale separation, showing it may introduce important biases in the description of the epidemic spread [19–33] and in the characterization of the transition behavior [31,34–37]. Departing from traditional approximations, few novel approaches are now available that derive the epidemic threshold constrained to specific contexts of generative models of temporal networks [22,32,35,38–41] or considering generic discrete-time evolving contact patterns [42–44]. In particular, the recently introduced infection propagator approach [43,44] is based on a matrix encoding the probabilities of transmission of the infective agent along time-respecting paths in the network. Its spectrum allows the computation of the epidemic threshold at any given time scale and for an arbitrary discrete-time temporal network. Leveraging an original mapping of the temporal network and epidemic spread in terms of a multilayer structure, the approach is valid in the discrete representation only, similarly to previous methods [17,18,35].

Meanwhile, a large interest in the study of continuously evolving temporal networks has developed, introducing novel representations [19,20,27,45] and proposing optimal discretization schemes [44,46,47] that may, however, be inaccurate close to the critical conditions [48]. Most importantly, the two representations—continuous and discrete—of a temporal network remain disjointed in current network epidemiology. A discrete-time evolving network is indeed a multilayer object interpretable as a tensor in a linear algebraic representation [49]. This is clearly no longer applicable when time is continuous, as it cannot be expressed in the form of successive layers. Hence, a coherent theoretical framework to bridge the gap between the two representations is still missing.

In this Letter, we address this issue by analytically deriving the infection propagator in continuous time. Formally, we show that the dichotomy discrete time–continuous time translates into the separation between a linear algebraic approach and a differential one, and that the latter can be derived as the structural limit of the former. Our approach yields a solution for the threshold of epidemics spreading on generic continuously evolving networks, and a closed form under a specific condition that is then validated through numerical simulations. In addition, the proposed novel perspective allows us to cast an important set of network classes into one single rigorous and comprehensive mathematical definition, including annealed [4,50,51] and activity-driven [35,52] networks, widely used in both methodological and applied research.

Let us consider a susceptible-infected-susceptible (SIS) epidemic model unfolding on a continuously evolving temporal network of N nodes. The SIS model constitutes a basic paradigm for the description of epidemics with

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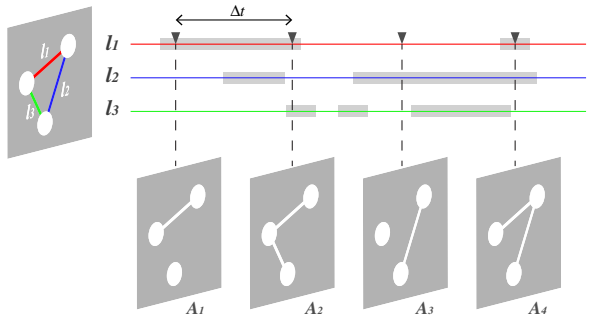

FIG. 1. Discrete sampling of a continuous-time temporal network. Links (l_1, l_2, l_3) activate in time as marked by the colored segments (top). This time evolution is sampled at intervals Δt, building a sequence of snapshots (bottom), corresponding to adjacency matrices $\{A_1, A_2, \ldots\}$.

reinfection [1]. Infectious individuals (I) can propagate the contagion to susceptible neighbors (S) with rate λ, and recover to the S state with rate μ. The temporal network is described by the adjacency matrix $A(t)$, with $t \in [0, T]$. We consider a discretized version of the system by sampling $A(t)$ at discrete time steps of length Δt (Fig. 1). This yields a finite sequence of adjacency matrices $\{A_1, A_2, \ldots, A_{T_{\text{step}}}\}$, where $T_{\text{step}} = \lfloor T/\Delta t \rfloor$, and $A_h = A(h\Delta t)$. The sequence approximates the original continuous-time network with increasing accuracy as Δt decreases. We describe the SIS dynamics on this discrete sequence of static networks as a discrete-time Markov chain [17,18]:

$$p_{h+1,i} = (1 - p_{h,i})\left[1 - \prod_j (1 - \lambda \Delta t A_{h,ji} p_{h,j})\right] + p_{h,i}(1 - \mu \Delta t), \tag{1}$$

where $p_{h,i}$ is the probability that a node i is in the infectious state at time step h, and $\mu\Delta t$ ($\lambda\Delta t$) is the probability that a node recovers (transmits the infection) during a time step Δt, for sufficiently small Δt.

By mapping the system into a multilayer structure encoding both network evolution and diffusion dynamics, the infection propagator approach derives the epidemic threshold as the solution of the equation $\rho[P(T_{\text{step}})] = 1$ [43,44], where ρ is the spectral radius of the following matrix:

$$P(T_{\text{step}}) = \prod_{k=1}^{T_{\text{step}}} [1 - \mu \Delta t + \lambda \Delta t A_k]. \tag{2}$$

The generic element $P_{ij}(T_{\text{step}})$ represents the probability that the infection can propagate from node i at time step 1 to node j at time step T_{step}, when λ is close to λ_c and within the quenched mean-field approximation (locally treelike network [53]). For this reason, P is denoted as the infection propagator.

To compute the continuous-time limit of the infection propagator, we observe that P obeys the recursive relation $P(h+1) = P(h)[1 - \mu\Delta t + \lambda\Delta t A_{h+1}]$. Expressed in continuous time and dividing both sides by Δt, the relation becomes

$$\frac{P(t + \Delta t) - P(t)}{\Delta t} = P(t)[-\mu + \lambda A(t + \Delta t)], \tag{3}$$

that in the limit $\Delta t \to 0$ yields

$$\dot{P}(t) = P(t)[-\mu + \lambda A(t)], \tag{4}$$

a system of N^2 coupled differential equations whose components are

$$\dot{P}_{ij}(t) = \sum_k [\lambda A_{kj}(t) - \mu \delta_{kj}] P_{ik}(t). \tag{5}$$

The lhs of Eq. (4) is the derivative of P that is well behaved if all entries are continuous functions of time. $A_{ij}(t)$ are, however, often binary, so that their evolution is a sequence of discontinuous steps. To overcome this, it is possible to approximate these steps with one-parameter families of continuous functions, compute the threshold, and then perform the limit of the parameter that recovers the discontinuity. More formally, this is equivalent to interpreting derivatives in the sense of tempered distributions [54].

In order to check that our limit process correctly connects the discrete-time framework to the continuous time one, let us now consider the standard Markov chain formulation of the continuous dynamics:

$$\dot{p}_i(t) = \lambda[1 - p_i(t)] \sum_j A_{ij}(t) p_j(t) - \mu p_i(t). \tag{6}$$

Performing a linear stability analysis of the disease-free state [i.e., around $p_i(t) = 0$] in the quenched mean-field approximation [17,18], we obtain

$$\dot{p}_i(t) = \sum_j [\lambda A_{ij}(t) - \mu \delta_{ij}] p_j(t). \tag{7}$$

We note that this expression is formally equivalent to Eq. (5). In particular, each row of P_{ij} of Eq. (5) satisfies Eq. (7). Furthermore, the initial condition $P_{ij}(0) = \delta_{ij}$ guarantees that in varying the row i, we consider all vectors of the space basis as initial condition. Every solution $p(T)$ of Eq. (7) can therefore be expressed as a linear combination of the rows of $P(T)$. Any fundamental matrix solution of Eq. (7) obeys Eq. (5) within the framework of the Floquet theory of nonautonomous linear systems [55].

The equivalence of the two equations shows that our limit of the discrete-time propagator encodes the dynamics

of the continuous process. It is important to note that the limit process leading to Eq. (4) entails a fundamental change of paradigm on the representation of the network structure and contagion process, where the linear algebraic representation suitable in discrete time turns into a differential geometrical description of the continuous-time flow. While network and spreading dynamics in discrete time are encoded in a multilayer adjacency tensor, the continuous time description proposed in Eq. (5) rests on a representation of the dynamical process in terms of a manifold whose points are adjacency matrices (or a rank-2 tensor in the sense of Ref. [49]) corresponding to possible network and contagion states. The dynamics of Eq. (5) is then a curve on such a manifold, indicating which adjacency matrices to visit and in which order. In practice, we recover that the contagion process on a discrete temporal network corresponding to an ordered subset of the full multilayer structure of Ref. [49] becomes in the limit $\Delta t \to 0$ a spreading on a continuous temporal network represented through a one-dimensional ordered subset of a tensor field (formally the pullback on the evolution curve). The two frameworks, so far considered independently and mutually exclusive, thus merge coherently through a smooth transition in this novel perspective.

We now turn to solving Eq. (4) to derive an analytic expression of the infection propagator. By defining the rescaled transmissibility $\gamma = \lambda/\mu$, we can solve Eq. (4) in terms of a series in μ [56],

$$P(t) = 1 + \sum_{j>0} \mu^j P^{(j)}(t), \tag{8}$$

with $P^{(0)} = 1$ and under the assumption that γ remains finite around the epidemic threshold for varying recovery rates. The recursion relation from which we derived Eq. (4) provides the full propagator for $t = T$. Equation (8) computed in T therefore yields the infection propagator for the continuous-time adjacency matrix $A(t)$, and is defined by the sum of the following terms:

$$P^{(j)}(T) = \int_0^T dx_1 \int_0^{x_1} dx_2 \cdots \int_0^{x_{j-1}} dx_j [\gamma A(x_j) - 1] \cdot [\gamma A(x_{j-1}) - 1] \cdots [\gamma A(x_1) - 1]. \tag{9}$$

Equations (8) and (9) can be put in a compact form by using Dyson's time-ordering operator $\mathcal{T}$ [57]. It is defined as $\mathcal{T} A(t_1)A(t_2) = A(t_1)A(t_2)\theta(t_1 - t_2) + A(t_2)A(t_1)\theta(t_2 - t_1)$, with θ being Heaviside's step function. The expression of the propagator is thus

$$P(t) = \mathcal{T} \exp\left(\int_0^t dx [-\mu + \lambda A(x)] \right). \tag{10}$$

Equation (10) represents an explicit general solution for Eq. (4) that can be computed numerically to arbitrary precision [56]. The epidemic threshold in the continuous-time limit is then given by $\rho[P(T)] = 1$.

We now discuss a special case where we can recover a closed-form solution of Eq. (10), and thus of the epidemic threshold. We consider continuously evolving temporal networks satisfying the following condition (*weak commutation*):

$$\left[A(t), \int_0^t dx A(x) \right] = 0, \quad \forall\ t \in [0, T]; \tag{11}$$

i.e., the adjacency matrix at a certain time $A(t)$ commutes with the aggregated matrix up to that time. In the introduced tensor field formalism, the weak commutation condition represents a constraint on the temporal trajectory, or equivalently, an equation of motion for $A(t)$.

Equation (11) implies that the order of factors in Eq. (9) no longer matters. Hence, we can simply remove the time-ordering operator $\mathcal{T}$ in Eq. (10), yielding

$$P(T) = e^{T[-\mu + \lambda \langle A \rangle]}, \tag{12}$$

where $\langle A \rangle = \int_0^T dt A(t)/T$ is the adjacency matrix averaged over time. The resulting expression for the epidemic threshold for weakly commuting networks is then

$$\lambda_c = \frac{\mu}{\rho[\langle A \rangle]}. \tag{13}$$

This closed-form solution proves to be extremely useful as a wide range of network classes satisfies the weak commutation condition of Eq. (11). An important class is constituted by annealed networks [4,50,51]. In the absence of dynamical correlations, the annealed regime leads to $\langle [A(x), A(y)] \rangle = 0$, as the time ordering of contacts becomes irrelevant. Equation (11) can thus be reinterpreted as $\langle [A(t), A(x)] \rangle_x = 0$, where the average is carried out over $x \in [0, t)$. For long enough t, $\int_0^t dx A(x)/t$ approximates well the expected adjacency matrix $\langle A \rangle$ of the annealed model, leading the annealed regime to satisfy Eq. (13). This result thus provides an alternative mathematical framework for the conceptual interpretation of annealed networks in terms of weak commutation. Originally introduced to describe disorder on quenched networks [58,59], annealed networks were mathematically described in probabilistic terms, with the probability of establishing a contact depending on the degree distribution $P(k)$ and the two-node degree correlations $P(k'|k)$ [50]. Here we show that temporal networks whose adjacency matrix $A(t)$ asymptotically commutes with the expected adjacency matrix are found to be in the annealed regime.

Equation (13) can also be used to test the limits of the time scale separation approach, by considering a generic temporal network not satisfying the weak commutation condition. If μ is small, we can truncate the series of the infection propagator [Eq. (8)] at the first order, $P = 1 + \mu P^{(1)} + \mathcal{O}(\mu^2)$, where $P^{(1)}(T) = T[\gamma \langle A \rangle - 1]$, to recover indeed Eq. (13). The truncation thus provides a mathematical expression of the range of validity of the

time-separation scheme for spreading processes on temporal networks, since temporal correlations can be disregarded when the network evolves much faster than the spreading process.

Extending the result of the annealed networks, we show that the weak commutation condition also holds for networks whose expected adjacency matrix depends on time as a scalar function (instead of being constant as in the annealed case), $\langle A(t)\rangle = c(t)\langle A(0)\rangle$. Also in this case we have $\langle [A(x), A(y)]\rangle = 0$, so that the same treatment performed for annealed networks applies. Examples are provided by global trends in activation patterns, as often considered in infectious disease epidemiology to model seasonal variations of human contact patterns (e.g., due to the school calendar) [60].

When the time scale separation approach is not applicable, we find another class of weakly commuting temporal networks that are used as a paradigmatic network example for the study of contagion processes occurring on the same time scale of contacts evolution—the activity-driven model [35]. It considers heterogeneous populations where each node i activates according to an activity rate a_i, drawn from a distribution $f(a)$. When active, the node establishes m connections with randomly chosen nodes lasting a short time δ ($\delta \ll 1/a_i$). Since the dynamics lacks time correlations, the weak commutation condition holds, and the epidemic threshold can be computed from Eq. (13). In the limit of large network size, it is possible to write the average adjacency matrix as $\langle A\rangle_{ij} = (m\delta/N)(a_i + a_j) + \mathcal{O}(1/N^2)$. Through row operations we find that the matrix has $\mathrm{rank}(\langle A\rangle) = 2$, and thus only two nonzero eigenvalues, α, σ, with $\alpha > \sigma$. We compute them through the traces of $\langle A\rangle$ ($\mathrm{tr}[\langle A\rangle] = \alpha + \sigma$ and $\mathrm{tr}[\langle A\rangle^2] = \alpha^2 + \sigma^2$) to obtain the expression of $\rho[\langle A\rangle]$ for Eq. (13): $\rho[\langle A\rangle] = \alpha = m\delta(\langle a\rangle + \sqrt{\langle a^2\rangle})$. The epidemic threshold becomes

$$\lambda_c \delta = \frac{\mu}{m(\langle a\rangle + \sqrt{\langle a^2\rangle})}, \tag{14}$$

yielding the same result of Ref. [35], provided here that the transmission rate λ is multiplied by δ to make it a probability, as in Ref. [35].

Finally, we verify that for the trivial example of static networks, with an adjacency matrix constant in time, Eq. (13) reduces immediately to the result of Refs. [17,18].

We now validate our analytical prediction against numerical simulations on two synthetic models. The first is the activity-driven model with activation rate $a_i = a$, $m = 1$, and average interactivation time $\tau = 1/a = 1$, fixed as the time unit of the simulations. The transmission parameter is the probability upon contact $\lambda\delta$ and the model is implemented in continuous time. The second model is based on a bursty interactivation time distribution $P(\Delta t) \sim (\epsilon + \Delta t)^{-\beta}$ [31], with $\beta = 2.5$ and ϵ tuned to obtain the same average interactivation time as before, $\tau = 1$. We simulate a SIS spreading process on the two networks with four different recovery rates, $\mu \in \{10^{-3}, 10^{-2}, 10^{-1}, 1\}$, i.e., ranging from a value that is 3 orders of magnitude larger than the time scale τ of the networks (slow disease), to a value equal to τ (fast disease). We compute the average simulated endemic prevalence for specific values of λ, μ using the quasistationary method [61] and compare the threshold computed with Eq. (13) with the simulated critical transition from extinction to endemic state. As expected, we find Eq. (13) to hold for the activity-driven model at all time scales of the epidemic process (Fig. 2), as the network lacks temporal correlations. The agreement with the transition observed in the bursty model, however, is recovered only for slow diseases, as at those time scales the network is found in the annealed regime. When network and disease time scales become comparable, the weakly commuting approximation of Eq. (13) no longer holds, as burstiness results in dynamical correlations in the network evolution [31].

Our theory offers a novel mathematical framework that rigorously connects discrete-time and continuous-time critical behaviors of spreading processes on temporal networks. It uncovers a coherent transition from an adjacency tensor to a tensor field resulting from a limit performed on the structural representation of the network and contagion process. We derive an analytic expression of the infection propagator in the general case that assumes a

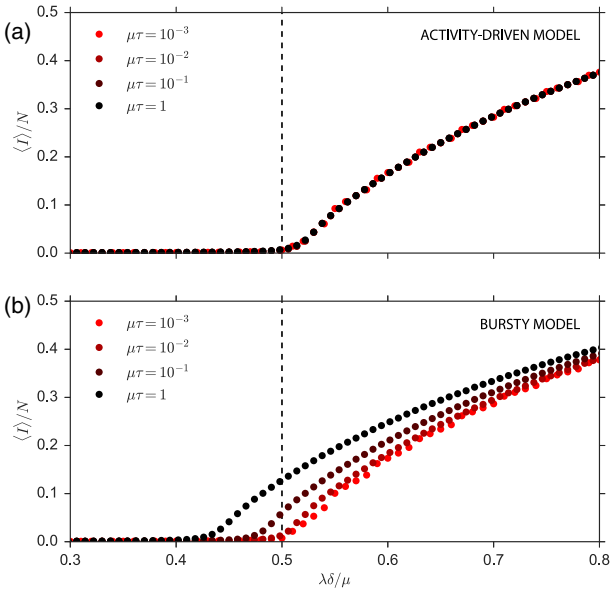

FIG. 2. Performance of the infection propagator estimate of the epidemic threshold in the continuous-time limit under the weak commutation approximation [Eq. (13)]. Panels report the average simulated endemic prevalence as a function of $\lambda\delta/\mu$ for the activity-driven model (a) and the bursty model (b). Different colors refer to explored values of the recovery rate μ. The vertical dashed line is the prediction for the critical transmissibility provided by Eq. (13).

closed-form solution in the introduced class of weakly commuting networks. This allows us to provide a rigorous mathematical interpretation of annealed networks, encompassing the different definitions historically introduced in the literature. This work also provides the basis for important theoretical extensions, assessing, for example, the impact of bursty activation patterns or of the adaptive dynamics in response to the circulating epidemic. Finally, our approach offers a tool for applicative studies on the estimation of the vulnerability of temporal networks to contagion processes in many real-world scenarios, for which the discrete-time assumption would be inadequate.

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[1] M. J. Keeling and P. Rohani, *Modeling Infectious Diseases in Humans and Animals* (Princeton University Press, Princeton, NJ, 2007).
[2] W. Goffman and V. A. Newill, Generalization of epidemic theory: An application to the transmission of ideas, Nature (London) **204**, 225 (1964).
[3] D. J. Daley and D. G. Kendall, Epidemics and rumours, Nature (London) **204**, 1118 (1964).
[4] R. Pastor-Satorras and A. Vespignani, Epidemic Spreading in Scale-Free Networks, Phys. Rev. Lett. **86**, 3200 (2001).
[5] D. J. Watts, A simple model of global cascades on random networks, Proc. Natl. Acad. Sci. U.S.A. **99**, 5766 (2002).
[6] A. Vespignani, Modelling dynamical processes in complex socio-technical systems, Nat. Phys. **8**, 32 (2012).
[7] T. E. Harris, Contact interactions on a lattice, Ann. Probab. **2**, 969 (1974).
[8] P. Grassberger, On the critical behavior of the general epidemic process and dynamical percolation, Math. Biosci. **63**, 157 (1983).
[9] D. Centola, V. M. Eguíluz, and M. W. Macy, Cascade dynamics of complex propagation, Physica (Amsterdam) **374A**, 449 (2007).
[10] Z. Liu, Y.-C. Lai, and N. Ye, Propagation and immunization of infection on general networks with both homogeneous and heterogeneous components, Phys. Rev. E **67**, 031911 (2003).
[11] Y. Moreno, M. Nekovee, and A. F. Pacheco, Dynamics of rumor spreading in complex networks, Phys. Rev. E **69**, 066130 (2004).
[12] Z. Ruan, G. Iñiguez, M. Karsai, and J. Kertész, Kinetics of Social Contagion, Phys. Rev. Lett. **115**, 218702 (2015).
[13] L. Böttcher, J. Nagler, and H. J. Herrmann, Critical Behaviors in Contagion Dynamics, Phys. Rev. Lett. **118**, 088301 (2017).
[14] R. Pastor-Satorras, C. Castellano, P. Van Mieghem, and A. Vespignani, Epidemic processes in complex networks, Rev. Mod. Phys. **87**, 925 (2015).
[15] R. Cohen, K. Erez, D. Ben-Avraham, and S. Havlin, Resilience of the Internet to Random Breakdowns, Phys. Rev. Lett. **85**, 4626 (2000).
[16] M. E. J. Newman, Spread of epidemic disease on networks, Phys. Rev. E **66**, 016128 (2002).
[17] Y. Wang, D. Chakrabarti, C. Wang, and C. Faloutsos, Epidemic spreading in real networks: An eigenvalue viewpoint, in *Proceedings of the 22nd International Symposium on Reliable Distributed Systems, 2003* (IEEE Computer Society, Los Alamitos, 2003), pp. 25–34.
[18] S. Gómez, A. Arenas, J. Borge-Holthoefer, S. Meloni, and Y. Moreno, Discrete time Markov chain approach to contact-based disease spreading in complex networks, Europhys. Lett. **89**, 38009 (2010).
[19] P. Holme, Modern temporal network theory: A colloquium, Eur. Phys. J. B **88**, 234 (2015).
[20] A. Vazquez, B. Rácz, A. Lukács, and A.-L. Barabási, Impact of Non-Poissonian Activity Patterns on Spreading Processes, Phys. Rev. Lett. **98**, 158702 (2007).
[21] R. R. Kao, D. M. Green, J. Johnson, and I. Z. Kiss, Disease dynamics over very different time-scales: Foot-and-mouth disease and scrapie on the network of livestock movements in the UK, J. R. Soc. Interface **4**, 907 (2007).
[22] E. Volz and L. A. Meyers, Epidemic thresholds in dynamic contact networks, J. R. Soc. Interface **6**, 233 (2009).
[23] N. H. Fefferman and K. L. Ng, How disease models in static networks can fail to approximate disease in dynamic networks, Phys. Rev. E **76**, 031919 (2007).
[24] M. C. Vernon and M. J. Keeling, Representing the UK's cattle herd as static and dynamic networks, Proc. R. Soc. B **276**, 469 (2009).
[25] J. L. Iribarren and E. Moro, Impact of Human Activity Patterns on the Dynamics of Information Diffusion, Phys. Rev. Lett. **103**, 038702 (2009).
[26] M. Karsai, M. Kivelä, R. K. Pan, K. Kaski, J. Kertész, A.-L. Barabási, and J. Saramäki, Small but slow world: How network topology and burstiness slow down spreading, Phys. Rev. E **83**, 025102 (2011).
[27] G. Miritello, E. Moro, and R. Lara, Dynamical strength of social ties in information spreading, Phys. Rev. E **83**, 045102 (2011).
[28] J. Stehlé, N. Voirin, A. Barrat, C. Cattuto, L. Isella, J.-F. Pinton, M. Quaggiotto, W. Van den Broeck, C. Régis, B. Lina, and P. Vanhems, High-resolution measurements of face-to-face contact patterns in a primary school, PLoS One **6**, e23176 (2011).
[29] P. Bajardi, A. Barrat, F. Natale, L. Savini, and V. Colizza, Dynamical patterns of cattle trade movements, PLoS One **6**, e19869 (2011).
[30] M. Kivelä, R. K. Pan, K. Kaski, J. Kertész, J. Saramäki, and M. Karsai, Multiscale analysis of spreading in a large communication network, J. Stat. Mech. (2012) P03005.
[31] L. E. C. Rocha and V. D. Blondel, Bursts of vertex activation and epidemics in evolving networks, PLoS Comput. Biol. **9**, e1002974 (2013).

[32] L. Ferreri, P. Bajardi, M. Giacobini, S. Perazzo, and E. Venturino, Interplay of network dynamics and heterogeneity of ties on spreading dynamics, Phys. Rev. E **90**, 012812 (2014).
[33] N. Masuda and P. Holme, Predicting and controlling infectious disease epidemics using temporal networks, F1000Prime Rep. **5**, 6 (2013).
[34] S. Bansal, J. Read, B. Pourbohloul, and L. A. Meyers, The dynamic nature of contact networks in infectious disease epidemiology, J. Biol. Dyn. **4**, 478 (2010).
[35] N. Perra, B. Gonçalves, R. Pastor-Satorras, and A. Vespignani, Activity driven modeling of time varying networks, Sci. Rep. **2**, 469 (2012).
[36] M. Starnini and R. Pastor-Satorras, Temporal percolation in activity-driven networks, Phys. Rev. E **89**, 032807 (2014).
[37] K. Sun, A. Baronchelli, and N. Perra, Contrasting effects of strong ties on SIR and SIS processes in temporal networks, Eur. Phys. J. B **88**, 326 (2015).
[38] K. T. D. Eames and M. J. Keeling, Monogamous networks and the spread of sexually transmitted diseases, Math. Biosci. **189**, 115 (2004).
[39] T. Gross, C. J. Dommar D'Lima, and B. Blasius, Epidemic Dynamics on an Adaptive Network, Phys. Rev. Lett. **96**, 208701 (2006).
[40] Z. Zhao, J. P. Calderón, C. Xu, G. Zhao, D. Fenn, D. Sornette, R. Crane, P. M. Hui, and N. F. Johnson, Effect of social group dynamics on contagion, Phys. Rev. E **81**, 056107 (2010).
[41] M. Taylor, T. J. Taylor, and I. Z. Kiss, Epidemic threshold and control in a dynamic network, Phys. Rev. E **85**, 016103 (2012).
[42] B. Aditya Prakash, H. Tong, N. Valler, M. Faloutsos, and C. Faloutsos, Virus propagation on time-varying networks: Theory and immunization algorithms, in *Proceedings of the European Conference on Machine Learning and Knowledge Discovery in Databases (ECML PKDD 2010), Barcelona*, Part III, edited by J. L. Balcázar, F. Bonchi, A. Gionis, and M. Sebag (Springer, Berlin, 2010).
[43] E. Valdano, L. Ferreri, C. Poletto, and V. Colizza, Analytical Computation of the Epidemic Threshold on Temporal Networks, Phys. Rev. X **5**, 021005 (2015).
[44] E. Valdano, C. Poletto, and V. Colizza, Infection propagator approach to compute epidemic thresholds on temporal networks: Impact of immunity and of limited temporal resolution, Eur. Phys. J. B **88**, 341 (2015).
[45] B. Klimt and Y. Yang, in *Machine Learning: ECML 2004*, Lecture Notes in Computer Science, Vol. 3201, edited by J.-F. Boulicaut, F. Esposito, F. Giannotti, and D. Pedreschi (Springer, Berlin, 2004), pp. 217–226.
[46] G. Krings, M. Karsai, S. Bernhardsson, V. D. Blondel, and J. Saramäki, Effects of time window size and placement on the structure of an aggregated communication network, Eur. Phys. J. Data Sci. **1**, 1 (2012).
[47] P. Holme, Epidemiologically optimal static networks from temporal network data, PLoS Comput. Biol. **9**, e1003142 (2013).
[48] P. G. Fennell, S. Melnik, and J. P. Gleeson, Limitations of discrete-time approaches to continuous-time contagion dynamics, Phys. Rev. E **94**, 052125 (2016).
[49] M. D. Domenico, A. Solé-Ribalta, E. Cozzo, M. Kivelä, Y. Moreno, M. A. Porter, S. Gómez, and A. Arenas, Mathematical Formulation of Multilayer Networks, Phys. Rev. X **3**, 041022 (2013).
[50] M. Boguñá, C. Castellano, and R. Pastor-Satorras, Langevin approach for the dynamics of the contact process on annealed scale-free networks, Phys. Rev. E **79**, 036110 (2009).
[51] C. Castellano and R. Pastor-Satorras, Thresholds for Epidemic Spreading in Networks, Phys. Rev. Lett. **105**, 218701 (2010).
[52] S. Liu, N. Perra, M. Karsai, and A. Vespignani, Controlling Contagion Processes in Activity Driven Networks, Phys. Rev. Lett. **112**, 118702 (2014).
[53] F. Radicchi and C. Castellano, Beyond the locally treelike approximation for percolation on real networks, Phys. Rev. E **93**, 030302 (2016).
[54] E. T. Whittaker, *A Course of Modern Analysis* (Cambridge University Press, Cambridge, England, 2000).
[55] J. P. Tian and J. Wang, Some results in floquet theory, with application to periodic epidemic models, Applicable Analysis **94**, 1128 (2015).
[56] S. Blanes, F. Casas, J. A. Oteo, and J. Ros, The magnus expansion and some of its applications, Phys. Rep. **470**, 151 (2009).
[57] F. J. Dyson, The radiation theories of Tomonaga, Schwinger, and Feynman, Phys. Rev. **75**, 486 (1949).
[58] S. Gil and D. H. Zanette, Optimal disorder for segregation in annealed small worlds, Eur. Phys. J. B **47**, 265 (2005).
[59] D. Stauffer and M. Sahimi, Diffusion in scale-free networks with annealed disorder, Phys. Rev. E **72**, 046128 (2005).
[60] W. P. London and J. A. Yorke, Recurrent outbreaks of measles, chickenpox and mumps: I. Seasonal variation in contact rates, American Journal of Epidemiology **98**, 453 (1973).
[61] S. C. Ferreira, C. Castellano, and R. Pastor-Satorras, Epidemic thresholds of the susceptible-infected-susceptible model on networks: A comparison of numerical and theoretical results, Phys. Rev. E **86**, 041125 (2012).