Universal nonlinear infection kernel from heterogeneous exposure on higher-order networks

Networks 2021: A Joint Sunbelt and NetSci Conference

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July 21, 2021

Three properties of human dynamics and disease transmissions are often overlooked in standard epidemic models: the hypergraph structure of contacts, the burstiness of human behavior, and the complex nonlinear relationship between the exposure to infected contacts and the risk of infection.

First, the co-location of individuals in specific environments, like workplaces, restaurants, and households, is an important prerequisite for exposure to infectious diseases on a social network. This, and the mounting evidence that social interactions go beyond pairwise interactions [1], suggest the use of higher-order network representations to describe contagion dynamics. Second, human behavior is characterized by heterogeneous temporal patterns, with periods where many interactions occur, separated by long periods of low activity [2]. One way these bursty interactions can arise is through heterogeneous period of co-location of individuals in various environments. Third, evidence associated with the minimal infective dose of different infectious diseases shows that not all exposures are equal, and that some minimal dose might be required for an infection to likely occur, which motivates the use of threshold models [3].

In this work [4], we combine these three properties in a hypergraph contagion model, where hyperedges represent environments where individuals can interact, and where a minimal number of interactions with infected individuals are needed to contract the disease. The central result we obtain is that bursty exposure, modeled by a power-law distribution of participation time to environments, can induce a nonlinear relationship between the number of infected participants and the probability to become infected. We then demonstrate how conventional epidemic wisdom can break down with the emergence of discontinuous transitions, super-exponential spread, and regimes of hysteresis.

On a theoretical level, this work formally provides a connection between complex contagions based on nonlinear infection kernels and threshold models. This also allows for a deeper understanding of how higher-order interactions and burstiness affect epidemic spreading. On the epidemiological level, our results challenge a key assumption of most epidemic models and ask: Why assume a linear relationship between the number of infectious contacts and the risk of infection?

[1] Battiston, F., Cencetti, G., Iacopini, I., Latora, V., Lucas, M., Patania, A., Young, J.-G. & Petri, G. (2020). Networks beyond pairwise interactions: structure and dynamics. Phys. Rep. 874, 1–92

[2] Karsai, M., Jo, H. H., & Kaski, K. (2018). Bursty human dynamics. Springer International Publishing.

[3] Dodds, P. S., & Watts, D. J. (2004). Universal behavior in a generalized model of contagion. Phys. Rev. Lett., 92, 218701.

[4] St-Onge, G., Sun, H., Allard, A., Hébert-Dufresne, L., & Bianconi, G. Bursty exposure on higher-order networks leads to nonlinear infection kernels. arXiv:2101.07229.

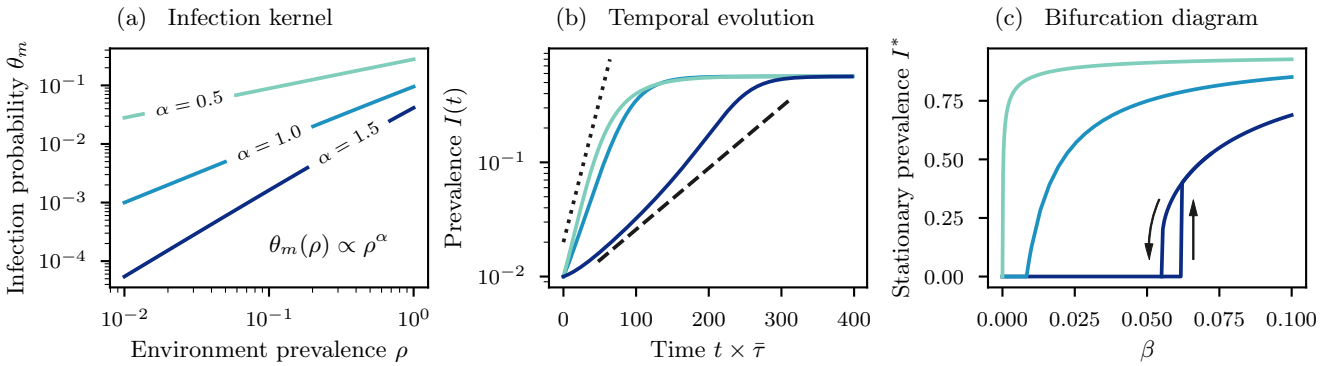

Figure 1: Bursty exposure induces contagions with nonlinear infection kernels. We assume that an individual must have ($K = 2$) interactions with infected individuals in a hyperedge (environment) to become infected. Each individual participates a time τ to a hyperedge, distributed according to $P(\tau) \propto \tau^{-\alpha-1}$ with $\tau \in [1, \infty)$. (a) Effective infection probability per participation to a hyperedge of size m. The infection probability has a power-law scaling $\theta_m(\rho) \propto \rho^\alpha$ if $K \geq \alpha$. (b)-(c) We study the consequences of nonlinear infection kernels for a SIS-type contagion dynamics on hypergraphs. We use Poisson distributions $\tilde{P}(k)$ and $\hat{P}(m)$ for the node degrees and the hyperedge sizes respectively, with $\langle k \rangle = 5$ and $\langle m \rangle = 10$. An infected individual recovers with probability μ, and the average number of interactions in an environment is proportional to β. (b) Supra-linear kernel $\alpha > 1$ leads to a super-exponential growth for the global prevalence $I(t)$. We use $\beta = 5 \times 10^{-4}$, $\beta = 0.025$ and $\beta = 0.077$ for $\alpha = 0.5$, $\alpha = 1$ and $\alpha = 1.5$ respectively. $\bar{\tau}$ is the median participation time associated with $P(\tau)$. (c) The bifurcation diagram in the stationary state ($t \to \infty$) can be continuous or discontinuous with a bistable regime. Sub-linear and linear kernels $\alpha \leq 1$ lead to a continuous phase transition, and the epidemic threshold β_c vanishes for $\alpha \to 0$. Supra-linear kernels $\alpha > 1$ can lead to a discontinuous phase transition with a bistable regime.