Localization, bistability and optimal seeding of contagions on higher-order networks

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Abstract

A standard problem in complex systems science has been to understand how infectious diseases, information, or any other contagion can spread within a system. Simple models of contagions tend to assume random mixing of elements, but real interactions are not random pairwise encounters: they occur within clearly defined higher-order structures. These higher-level structures could represent communities in social systems, cells in organisms or modules in neural networks. For a broader understanding of contagion dynamics in complex networks, we need to embrace higher-order structure, which can itself take many forms such as simplicial complexes or hypergraphs. To accurately describe spreading processes on these higher-order networks and correctly account for the heterogeneity of the underlying structure, we use a set of approximate master equations. This general framework allows us to unveil and characterize important properties of these systems. Here we focus on three of them: the localization of contagions within certain substructures, the bistability of the stationary state and a crossover of the optimal seeding strategies to maximize early spread.

To describe higher-order networks, we consider infinite-size random hypergraphs, allowing mathematical treatments akin to mean-field theory. Each node has an intrinsic *membership* value m that indicates to how many structures (called groups) they belong. Groups can be of various size n, and both the membership and group size are distributed according to a distribution g_m and p_n. We then assign nodes to groups completely at random, formally creating a bipartite structure. This structure can then be used to represent various types of higher-order networks: we can project all groups to fully connected cliques [3], or associate a group to a simplex with the perspective of describing a contagion on the resulting simplicial complex [2].

With this structure representation, we can describe contagion processes on this random ensemble using *approximate master equations* (see Refs. [1, 4] for the approach), providing a highly detailed description of the inner dynamics within groups. This framework is amenable to analytical treatments, allowing us to distinguish different dynamical regimes. For instance, in Fig. 1(a)-(b), we show the phase transition associated with the Susceptible-Infectious-Susceptible (SIS) model for two different types of structures. As expected, in both cases we see that there exists two phases : for a transmission rate $\beta \leq \beta_c$, the contagion does not persist in the population, while for $\beta > \beta_c$, it invades the system. Less expected is the fact that in one case, all groups are affected beyond β_c [Fig. 1(a)], while only the largest structures are in the other [Fig. 1(b)]. This is what we call a phenomenon of *mesoscopic localization*, affected by the structural properties of the higher-order networks (see Ref. [4] for more details). It is worth mentioning that mesoscopic localization may have important repercussions on the efficiency of an intervention to suppress epidemics [5].

Complex contagion models, where the infection rate of a node increases non-linearly with the number of contagious neighbors, give rise to even richer dynamical features. In Fig. 2(a), we show the emergence of a bistable regime when the non-linearity is large enough. This regime also clearly depends on the underlying higher-order structure. Interestingly, our approximate master-equation framework shows that the first three moments of g_m determine whether or not it is possible to observe bistability. This is illustrated in figure 2(b), where we show the phase diagram for two different values of $\langle m^3 \rangle$, while using the same first two moments $\langle m \rangle$ and $\langle m^2 \rangle$.

Higher-order networks also challenge our preconception of what should be the optimal subset of nodes to infect in order to invade a system as fast as possible. For simple contagion models and considering only random pairwise interactions, nodes with the most contacts should always be favored. In our setting, this implies choosing nodes with maximal membership m. This *influential spreaders* strategy is, however, not always optimal for complex contagions on higher-order networks. Indeed, for increasing non-linearity of the contagion, we show in Fig. 2(c) that targeting and choosing wisely which nodes to infect within groups, while ignoring the membership of the nodes, becomes more profitable. We call this strategy *influential structures*.

Altogether, our results highlight the importance of considering higher-order interactions in the modeling of complex dynamical systems.

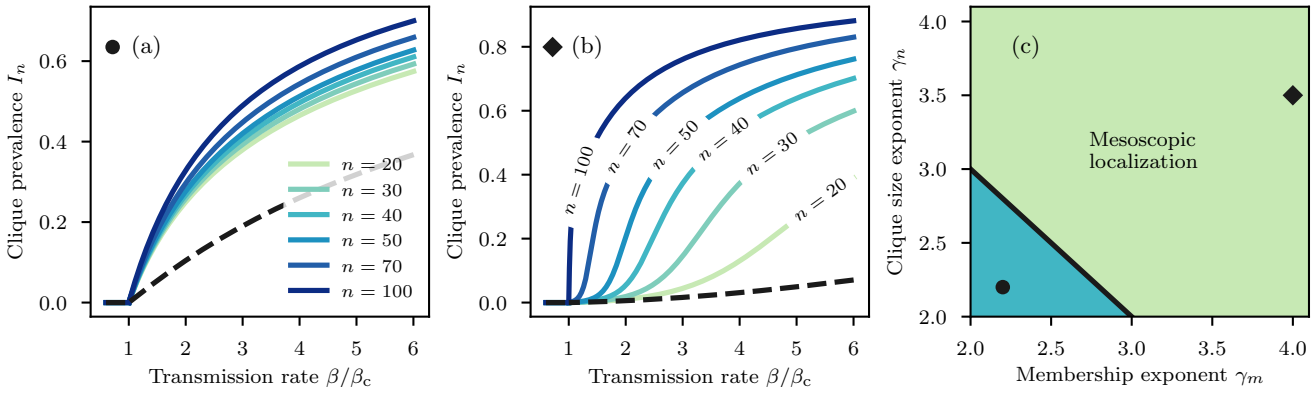

Figure 1: **Epidemic localization in networks with heterogeneous higher-order structures.** We use power-law distributions $g_m \propto m^{-\gamma_m}$ and $p_n \propto n^{-\gamma_n}$. We use the SIS model, where contagious nodes transmit the disease to susceptible nodes in their groups at rate β. (a)-(b) Solid lines represent the clique prevalence—the average fraction of contagious nodes within cliques of size n—while dashed lines represent the global prevalence. (a) If groups are highly coupled (small γ_m, γ_n), we obtain a standard collective activation of all substructures beyond the epidemic threshold. (b) With a lower coupling (larger γ_m, γ_n), we find a phenomenon of mesoscopic localization. While the global prevalence in the population can remain extremely low, larger substructures can self-sustain the epidemic. (c) Mesoscopic localization is actually the norm rather than the exception. Indeed, the phenomenon is observed for all but the most extremely coupled scenarios. The solid line separating the *delocalized regime* (blue region) and the *mesoscopic localization regime* (green region) is obtained analytically in Ref. [4]. The circle and diamond markers correspond to the networks respectively used in panels (a) $\gamma_n = \gamma_m = 2.2$ and (b) $\gamma_n = 3.5, \gamma_m = 4$.

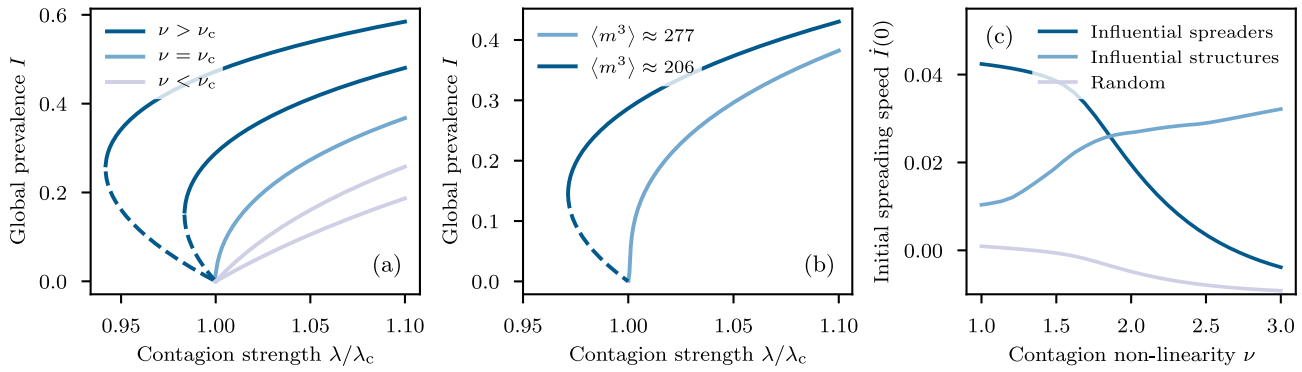

Figure 2: **Complex contagions on higher-order networks lead to bistability and crossover on optimal seeding strategies.** We use a complex contagion model akin to Ref. [2]. In our setting, a susceptible node in a group of size n with i contagious nodes becomes also contagious at rate λi^ν. For nodes belonging to multiple groups, the rates add up. (a)-(b) Global prevalence as a function of the contagion strength λ, rescaled by the invasion threshold λ_c above which the contagion always invade the system. Solid lines and dashed lines represent stable and unstable solutions respectively. (a) The structure is fixed with $g_m = \delta_{m,3}$ and $p_n = \delta_{n,4}$. Values of $\nu \in \{1.5, 1.7, \nu_c, 2.1, 2.3\}$ (bottom to top curves) were used, with bistability threshold $\nu_c \approx 1.91$. (b) The contagion non-linearity is fixed at $\nu = 2.82$ and the group size distribution is $p_n = \delta_{n,3}$. We use two distributions for g_m with different third moments but with the same first two moments $\langle m \rangle \approx 5.03$ and $\langle m^2 \rangle \approx 30.2$. (c) Initial spreading speed (time derivative of the prevalence) for different strategies as a function of the contagion non-linearity. The initial fraction of contagious nodes is $I(0) = 10^{-2}$. We used a Poisson-like distribution of group sizes with mean $\langle n \rangle \approx 5$ and a power-law distribution of memberships $g_m \sim m^{-\gamma_m}$ with $\gamma_m = 3.8$. The random strategy corresponds to simply infecting nodes uniformly at random.

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