Simplicial models of social contagion

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Networks have been extensively used to describe the connectivity patterns of complex systems of various nature. In particular, they have been used to represent the social structure on which dynamical processes, such as the spreading of diseases or the formation of opinions, occur in a population [1;2;3]. However, recent empirical results have shown that the dynamics of social contagion may significantly differ from the those describing the spreading of biological diseases [4;5]. In fact, the transmission of a biological virus happens through pairwise interactions between the individuals of a population, so it can be well modelled as a spreading process over the links of the underlying network. Contrarily, pairwise interactions are often not enough to characterize the mechanisms behind social contagion processes such as, for instance, the diffusion of opinions or the adoption of norms, where more complex dynamics of peer influence and social reinforcement take place [6].

Here, we introduce the Simplicial Contagion Model (SCM), a novel higher-order modelling framework of social contagion [7]. In the SCM, the structure of a social system is represented as a simplicial complex, and different channels of infections, with different transmission rates, are considered depending on the fact that a contagion can occur on a link (two-body interaction) or is due to a group interaction. Differently from standard networks, simplicial complexes are indeed able to describe in a unified manner not only pairwise but also group interactions of higher-order, by extending the network concept of links between two units to the one of simplices, encoding relations between any number of units [8]. By taking into account these high order interactions between individuals, our model extends the idea of complex contagion by considering not only multiple but also collective exposures.

In the SCM, we start from the standard SIS compartmental model in which the population of N individuals is divided into two classes of susceptible (S) and infected (I), and each individual corresponds to a vertex of the simplicial complex representing the social structure. The model of order D, with $D \in [1, N-1]$, is governed by a set of D control parameters $B = \{\beta_1, \beta_2, \dots, \beta_D\}$, whose elements represent the probability per unit time for a susceptible node i that participates to a simplex σ of dimension D to get the infection from each one of the subfaces composing σ. With this notation, β_1 corresponds to the standard probability of infection β that an infected node i passes the infection to a susceptible one j through the link (i, j), while $\beta_2 = \beta_\Delta$ corresponds to the probability that node i receives the infection from the closed triangle (2-simplex) (i, j, k) (see Figure 1a-f). Finally, the recovery dynamics is controlled by the standard recovery probability μ.

We find that the inclusion of the lowest higer-order interactions is already enough to change the nature of the spreading process from continuous to discontinuous. Through extensive numerical simulations on both empirical data and synthetic simplicial complexes we identify the regions of the parameter space where we observe bistability in the asymptotic density of infectious, with a co-existence of an healthy and an endemic state (see Fig. 1g). We further investigate this phenomena by deriving a mean field (MF) equation for the evolution of density of infected nodes, showing that the steady-state dynamics, the position, and the nature of the transition can be predicted analytically.

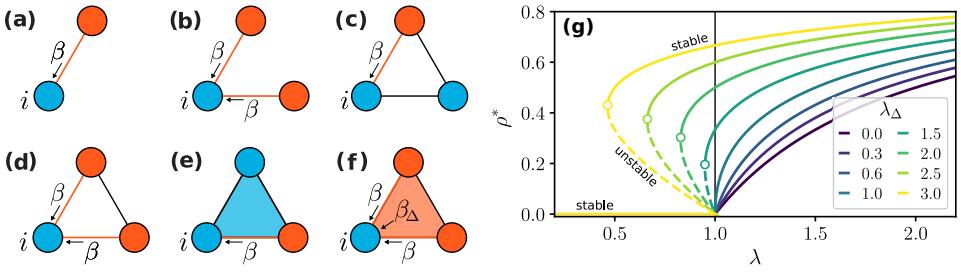

Figure 1: (a-f) Different channels of infection for a susceptible node i in the SCM of order $D = 2$ (S=blue, I=red). Node i is in contact with one (a, c) or more infected nodes (b, d) through a link (1-simplex), and for each one of these links is infected with probability β at each timestep. (e, f) Node i belongs to a 2-simplex (triangle). In (e) one of the nodes of the 2-simplex is not infected, so i can only receive the infection from the (red) link with probability β. Contrarily, in (f) node i can get the infection from each of the two 1-faces (links) of the simplex with probability β, and also from the 2-face with probability $\beta_2 = \beta_\Delta$. (g) Phase diagram of the SCM of order $D = 2$ in mean-field approximation. The stationary solutions are plotted as a function of the rescaled link infectivity $\lambda = \beta\langle k\rangle/\mu$. Different curves correspond to different values of the triangle infectivity $\lambda_\Delta = \beta_\Delta\langle k_\Delta\rangle/\mu$, with k_Δ being the number of 2-simplices incident on a node [9]. Continuous and dashed lines correspond to stable and unstable branches respectively, while the vertical line denotes the epidemic threshold of the standard SIS model. For $\lambda_\Delta \leq 1$ the high order interactions contribute to increase the density of infected in the endemic state, while leaving the threshold unchanged. Contrarily, when $\lambda_\Delta > 1$ we observe a shift of the epidemic threshold, and the transition becomes discontinuous.

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