Evolution of Spreading Processes on Complex Networks

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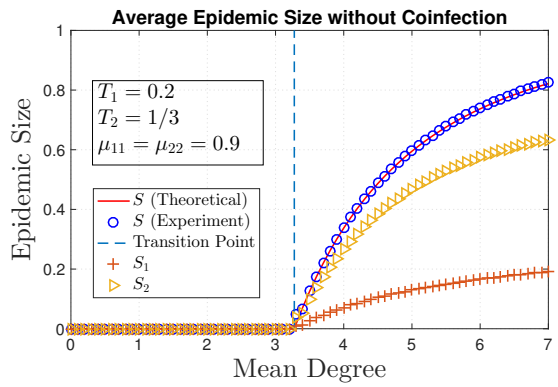
Abstract

Most existing works on spreading processes assume that the propagating object, i.e., a virus or a piece of information, is transferred across the nodes without going through any modification [1]–[9]. However, in real-life spreading processes, pathogens often evolve in response to changing environments and medical interventions [10], [11], and information is often modified by individuals before being forwarded. In this paper [12], we investigate the *evolution* of spreading processes with the aim of i) revealing the role of evolution on the threshold, probability, and final size of epidemics; and ii) understanding the interplay between the structural properties of the network and the evolution process.

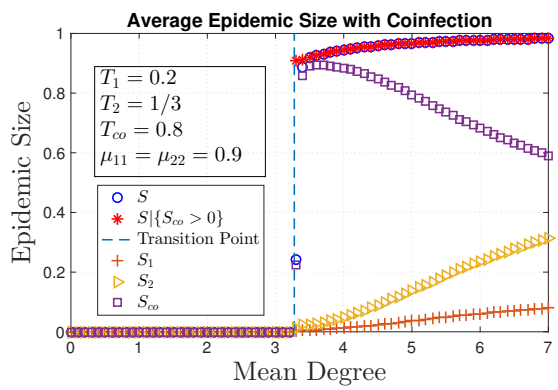
We consider a SIR spreading process that starts with a single individual infected with strain-1 (that has *transmissibility* T_1). Once a susceptible individual receives the infection from the seed, the pathogen may evolve within that host with some probability. In particular, the pathogen may remain as strain-1 with probability μ_{11} or mutate to strain-2 (that has transmissibility T_2) with probability $\mu_{12} = 1 - \mu_{11}$. If the pathogen remains as strain-1 (respectively, mutates to strain-2), then the host infects each of her susceptible neighbors in the subsequent stages independently with probability T_1 (respectively, T_2). Similarly, if any susceptible individual receives strain-2 at some point, the pathogen may remain as strain-2 with probability μ_{22} or mutate to strain-1 with probability $\mu_{21} = 1 - \mu_{22}$.

We start by considering the case where coinfection is not possible, i.e., each infected host either carries strain-1 or strain-2, but not both. Existing research [11] only explores the probability of epidemics, but lacks any insights on the expected epidemic size (denoted by S) or, more precisely, the expected fraction of individuals infected by each strain (denoted by S_1 and S_2, respectively). We present a *theoretical framework that computes the expected epidemic size and the expected fraction of individuals infected by each strain* for any choice of T_1, T_2, μ_{11}, μ_{22}, and any given degree distribution for the underlying contact network. [1].

[1]We remark that it is straightforward to extend our theoretical results to any arbitrary number of strains.



(a)



(b)

Fig. 1: a) Average epidemic size undergoes a second-order phase transition without coinfection. The expected epidemic size predicted by our analysis matches the simulation. (b) Average epidemic size may undergo a first-order phase transition depending on coinfection dynamics. S_{co} denotes the expected fraction of coinfected individuals. Coinfection dominates the dynamics once it appears at the transition point leading to a first-order phase transition, in contrast to the case where coinfection is not possible.

We then explore the case where the emergence of coinfection is possible. In particular, a susceptible individual who gets infected with strain-1 and strain-2 *simultaneously* becomes coinfected, and starts to transmit the coinfection with transmissibility T_{co}. We show that coinfection gives rise to a rich set of dynamics: it can amplify or inhibit the spreading dynamics,

and more remarkably *lead the order of phase transition to change from second-order to first-order*. We investigate the delicate interplay between the network structure, mutation schemes, and coinfection dynamics and reveal the cases where such interplay induces first-order phase transitions for the expected epidemic size.

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