Evolutionary Adaptations on Clustered Contact Networks

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The emergence of the novel COVID-19 virus sheds the light on the critical role of evolutionary adaptations in enabling pathogen establishment in new host species. At a high level, a chain of genetic mutations could allow a pathogen to cross the species barrier and start infecting humans at a large scale. A common theme, however, with existing mathematical and simulation models for epidemiology is the assumption that the propagating pathogen is transferred across the nodes without going through any modification or *evolution*; an assumption that renders these models incapable of accurately predicting or tracking the emergence of novel strains that are fit for human hosts. Additionally, most of the research studies on *evolutionary adaptations* of infectious diseases either assume a homogeneous-mixing host population, or focus entirely on the ecological or environmental factors of pathogen evolution, ignoring the key role of the host contact network on the spread and evolution of the pathogen.

In this article, we aim to tackle the above limitations by presenting a mathematical theory that characterizes the role of evolutionary adaptations on the spread of pathogens on *clustered* contacts networks, hence pushing the frontiers of network epidemics one step closer to reality. Clustering is characterized by the phenomenon that two individuals who have mutual friends are likely to be friends; more generally, it is characterized by the existence of groups of individuals with dense contact patterns internally and sparse contact patterns externally. Indeed, the prevalence of clustering in real-world contact networks signals its importance and significant impact on the behavior of various spreading processes.

We focus on the case where co-infection with multiple pathogen strains is not possible, and derive a mathematical theory that predicts the epidemic threshold and the probability of emergence as functions of the characteristics of the spreading object, the evolutionary pathways of the pathogen, and the structure of the underlying network as given by the degree distribution and clustering coefficient. Our contributions include i) revealing the role of evolutionary adaptations on the epidemic threshold and probability of emergence when the network exhibits a non-vanishing clustering coefficient; and ii) identifying the interplay between the structural properties of the network and the evolutionary adaptations of the pathogen.



Figure 1: We consider a fitness landscape consisting of two pathogen strains with different transmissibilities and mutation rates. a) The probability of emergence on contact networks with doubly Poisson distribution. *S* denotes the expected epidemic size. Experimental results are in agreement with our theoretical results. b) With *c* denoting the level of clustering, our experimental results show that high clustering increases the threshold of epidemics and reduces the probability of emergence around the transition point.