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Review

Coupled disease–behavior dynamics on complex networks: A review

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Abstract

It is increasingly recognized that a key component of successful infection control efforts is understanding the complex, two-way interaction between disease dynamics and human behavioral and social dynamics. Human behavior such as contact precautions and social distancing clearly influence disease prevalence, but disease prevalence can in turn alter human behavior, forming a coupled, nonlinear system. Moreover, in many cases, the spatial structure of the population cannot be ignored, such that social and behavioral processes and/or transmission of infection must be represented with complex networks. Research on studying coupled disease–behavior dynamics in complex networks in particular is growing rapidly, and frequently makes use of analysis methods and concepts from statistical physics. Here, we review some of the growing literature in this area. We contrast network-based approaches to homogeneous-mixing approaches, point out how their predictions differ, and describe the rich and often surprising behavior of disease–behavior dynamics on complex networks, and compare them to processes in statistical physics. We discuss how these models can capture the dynamics that characterize many real-world scenarios, thereby suggesting ways that policy makers can better design effective prevention strategies. We also describe the growing sources of digital data that are facilitating research in this area. Finally, we suggest pitfalls which might be faced by researchers in the field, and we suggest several ways in which the field could move forward in the coming years.

Keywords: Disease–behavior dynamics; Decision-making; Vaccination; Networks; Social distancing

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1. Introduction

Infectious diseases have long caused enormous morbidity and mortality in human populations. One of the most devastating examples is the Black Death, which killed 75 to 200 million people in the medieval period [1]. Currently, the rapid spread of infectious diseases still imposes a considerable burden [2]. To elucidate transmission processes of infectious diseases, mathematical modeling has become a fruitful framework [3]. In the classical modeling framework, a homogeneously mixed population can be classified into several compartments according to disease status. In particular, the most common compartments are those that contain susceptible individuals (S), infectious (or infected) individuals (I), and recovered (and immune) individuals (R). Using these states, systems of ordinary differential equations (ODEs) can be created to capture the evolution of diseases with different natural histories. For example, a disease with no immunity where susceptible individuals who become infected return to the susceptible class after recovering (SIS natural history, see Fig. 1(a)) can be modeled as

$$\begin{aligned} \frac{d[S]}{dt} &= -\beta[S][I] + \mu[I], \\ \frac{d[I]}{dt} &= \beta[S][I] - \mu[I], \end{aligned} \tag{1}$$

where $[S]$ ($[I]$) represents the number of susceptible (infectious) individuals in the population, β is the transmission rate of the disease, and μ is the recovery rate of infected individuals. Some diseases, however, may give immunity to individuals who have recovered from infection (SIR natural history, see Fig. 1(b)). In this case, the equations become

$$\begin{aligned} \frac{d[S]}{dt} &= -\beta[S][I] \\ \frac{d[I]}{dt} &= \beta[S][I] - \mu[I], \\ \frac{d[R]}{dt} &= \mu[I], \end{aligned} \tag{2}$$

where $[R]$ is the number of recovered (and immune) individuals. In these ODE models, a general measure of disease severity is the basic reproductive number $R_0 = \beta N/\mu$, where N is the population size. In simple terms, R_0 is the mean number of secondary infections caused by a single infectious individual, during its entire infectious period, in an otherwise susceptible population [4]. If $R_0 < 1$, the disease will not survive in the population. However, if $R_0 > 1$, the disease may be able to persist. Typically, parameters like the transmission rate and recovery rate are treated as fixed.

However, new approaches to modeling have been developed in past few decades to address some of the limitations of the classic differential equation framework that stem from its simplifying assumptions. For instance, the impact of behavioral changes in response to an epidemic is usually ignored in these formulations (e.g., the transmission rate is fixed), but in reality, individuals usually change their behavior during an outbreak according to the change of perceived infection risk, and their behavioral decisions can in turn impact the transmission of infection. Another limitation of the classical compartmental models is the assumption of well-mixed populations (namely, individuals interact with all others at the same contact rate), which thus neglects heterogeneous spatial contact patterns that can arise in realistic populations. In this review we will describe how models of the past few decades have begun to address these limitations of the classic framework.

1.1. Disease–behavior systems

1.1.1. Nonlinear coupling and emergent phenomena

Traditionally, infectious disease models have treated human behavior as a fixed phenomenon that does not respond to disease dynamics or any other natural dynamics. For many research questions, this is a useful and acceptable simplification. However, in other cases, human behavior responds to disease dynamics, and in turn disease dynamics responds to human behavior. For example, the initiation of an epidemic may cause a flood of awareness in the population such that protective measures are adopted. This in turn, reduces the transmission of the disease. In such cases, it becomes possible to speak of a single, coupled "disease–behavior" system where a human subsystem and a disease