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Review

Coupled disease–behavior dynamics on complex networks: A review

Zhen Wang [a,b,*], Michael A. Andrews [c], Zhi-Xi Wu [d], Lin Wang [e], Chris T. Bauch [f,**]

[a] *School of Automation, Northwestern Polytechnical University, Xi'an 710072, China*
[b] *Interdisciplinary Graduate School of Engineering Sciences, Kyushu University, Fukuoka, 816-8580, Japan*
[c] *Department of Mathematics and Statistics, University of Guelph, Guelph, ON, N1G 2W1, Canada*
[d] *Institute of Computational Physics and Complex Systems, Lanzhou University, Lanzhou, Gansu 730000, China*
[e] *School of Computer and Communication Engineering, Tianjin University of Technology, Tianjin 300384, China*
[f] *Department of Applied Mathematics, University of Waterloo, Waterloo, ON N2L 3G1, Canada*

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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

* Corresponding author at: School of Automation, Northwestern Polytechnical University, Xi'an 710072, China.
** Corresponding author.
E-mail addresses: zhenwang0@gmail.com (Z. Wang), mandre04@uoguelph.ca (M.A. Andrews), wuzhx@lzu.edu.cn (Z.-X. Wu), wanglinieee@outlook.com (L. Wang), cbauch@uwaterloo.ca (C.T. Bauch).

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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

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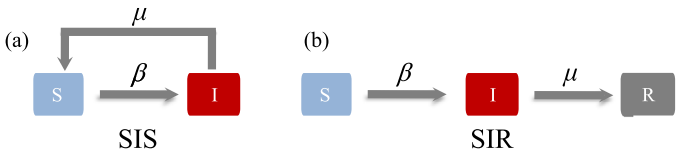

Fig. 1. Schematic illustration of disease natural histories within the compartmental models: (a) SIS natural history and (b) SIR natural history.

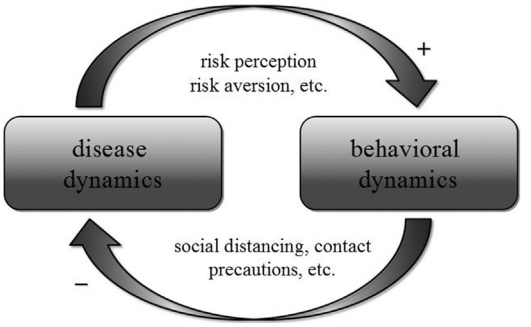

Fig. 2. Schematic illustration of disease–behavior interactions as a negative feedback loop. In this example, the loop from disease dynamics to behavioral dynamics is positive (+) since an increase in disease prevalence will cause an increase in perceived risk and thus an increase in protective behaviors. The loop from behavioral dynamics back to disease dynamics is negative (−) since an increase in protective behaviors such as contact precautions and social distancing will generally suppress disease prevalence.

transmission subsystem are coupled to one another (see Fig. 2). Moreover, because the human and natural subsystems are themselves typically nonlinear, the coupled system is therefore also typically nonlinear. This means that phenomena can emerge that cannot be predicted by considering each subsystem in isolation. For example, protective behavior on the part of humans may ebb and flow according to disease incidence and according to a characteristic timescale (as opposed to being constant over time, as would occur in the uncoupled subsystems).

1.1.2. Game theory

To explore strategic interactions between individual behaviors, game theory has become a key tool across many disciplines. It provides a unified framework for decision-making, where the participating players in a conflict must make strategy choices that potentially affect the interest of other players. Game theory and its corresponding equilibrium concepts, such as the Nash Equilibrium, emerged in seminal works from the 1940s and 1950s [5,6]. A Nash Equilibrium is a set of strategies such that no player has an incentive to unilaterally deviate from the present strategy. That is, the Nash Equilibrium makes strategies form best responses to one other, since every player, who has a consistent goal to maximize his own benefit or utility, is perfectly rational. Game theory has been applied to fields such as economics, biology, mathematics, public health, ecology, traffic engineering, and computer science [7–12]. For example, in voluntary vaccination programs, the formal theory of games can be employed as a framework to analyze the vaccination equilibrium level in populations [9,13,14]. In the context of vaccination, the feedback between individual decisions of vaccination (or other prevention behaviors) and disease spreading is captured, hence these systems exemplify coupled disease–behavior systems.

In spite of the great progress of game theory, the classical paradigm still shows its limitations in many scenarios. It thus becomes instructive to relax some key assumptions, such as the introduction of bounded rationality. Game theory has been extended into evolutionary biology, which has generated great insight into the evolution of strategies [15–19] under both biological and cultural evolution. For instance, the replicator equation, which consists of sets of differential equations describing how the strategies of a population evolve over time under selective pressures, has also been used to study learning in various scenarios [20]. Except for temporal concepts, spatial interaction topology has also proved to be crucial in determining system equilibria (also see Refs. [16,17] for a comprehensive overview). Evolutionary game theory has been extensively applied to behavioral epidemiology, whose details will be surveyed in the following sections.

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1.2. Related concepts in statistical physics

1.2.1. Physics of lattices and networks

Several methods from statistical physics have become useful in the study of disease–behavior interactions on complex networks. Most populations are spatially structured in the sense that individuals preferentially interact with those who share close geographic proximity. Perhaps, the most simple population structure is a regular lattice: all the agents are assigned specific locations on it, normally a two-dimensional square lattice, just like atoms in crystal lattice sites, which interact with only nearest neighbors. In a regular lattice population, each individual meets the same people they interact with regularly, rather than being randomly reshuffled into a homogeneous mixture, as in well-mixed population models. In addition, another type of homogeneous network attracting great research interest is the Erdös–Rényi (ER) graph [21], which is a graph where nodes are linked up randomly and which is often used in the rigorous analysis of graphs and networks.

However, in reality, there is ubiquitous heterogeneity in the number of contacts per individual, and recent studies have shown that the distribution of contact numbers of some social networks is not homogeneous but appears to follow a power-law [22]. Moreover, social contact networks also display small-world properties (i.e., short average path length between any two individuals and strong local clustering tendency), which cannot be well described by regular lattices or random graphs [23]. With both motivations, two significant milestones were born in the late 90s: the theoretical models of small-world (SW) networks and scale-free (SF) networks [24,25]. Subsequently, more properties of social networks have been extensively investigated, such as community structure (a kind of assortative structure where individuals are divided into groups such that the members within each group are mostly connected with each other) [26], clusters [27], and the recent proposal of multilayer as well as time-varying frameworks [28–32]. Due to the broad applicability of complex networks, network models have been widely employed in epidemiology to study the spread of infectious diseases [27]. In networks, a vertex represents an individual and an edge between two vertices represents a contact over which disease transmission may occur. An epidemic spreads through the network from infected to susceptible vertices.

1.2.2. Epidemic spreading on networks

With the advent of various network algorithms, it becomes instructive incorporating disease dynamics into such infrastructures to explore the impact of spatial contact patterns [33–38]. Replacing the homogeneous mixing hypothesis that any individual can come into contact with any other agents, networked epidemic research assumes that each individual has comparable number of contacts, denoted by its degree k. Under this treatment, the most significant physics finding is that network topology will directly determine the threshold of epidemic outbreak and phase transition. For example, compared with the finite epidemic threshold of random network, Romualdo et al. found that disease with SIS dynamics and even a very small transmission rate can spread and persist in the SF networks (i.e., there is absence of a disease threshold) [39]. This point helps to explain why it is extremely difficult to eradicate viruses on Internet and World Wide Web, and why those viruses have an unusual long lifetime. But the absence of epidemic threshold is only suitable for SF networks with a power-law degree distribution $P(k) \sim k^{-\gamma}$ with $\gamma \in (2, 3]$. If γ is extended to the range $(3, 4)$, an anomalous critical behavior takes place [39,40]. To show the condition of disease spread, it is meaningful to define the relative spreading rate $\lambda \equiv \beta/\mu$. The larger is λ, the more likely the disease will spread. Generally, for an SF network with arbitrary degree distribution, the epidemic threshold is

$$\lambda_c = \frac{\langle k \rangle}{\langle k^2 \rangle}. \tag{3}$$

In particular, for an SF network $\langle k^2 \rangle$ diverges in the $N \to \infty$ limit, and so the epidemic threshold is expected to die out. Similarly, it is easy to derive the threshold of SIR model

$$\lambda_c = \frac{\langle k \rangle}{\langle k^2 \rangle - \langle k \rangle}, \tag{4}$$

which is related with average degree $\langle k \rangle$ and the second moment $\langle k^2 \rangle$ of networks as well. Along these findings, more endeavors are devoted to the epidemic threshold of spatial networks with various properties, such as degree correlation [41,42], SW topology [23], community structure [43], and k-core [44]. On the other hand, more analysis and prediction methods (such as mean-field method, generation function) are also proposed to explain the transition of disease on realistic networks [27,45] and immunization strategies of spatial networks are largely identified [46].

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1.3. A simple example of a disease–behavior system on a complex network

To illustrate the meaning of studying disease–behavior dynamics on complex networks, it is instructive to firstly describe a simple example of such a system.

Consider a population of individuals who are aware of a spreading epidemic. The information each individual receives regarding the disease status of others is derived from the underlying social network of the population. These networks have been shown to display heterogeneous contact patterns, where the node degree distribution often follows a power-law fashion [47,48]. It is possible to use these complex network patterns to model a realistic population that exhibits adaptive self-protective behavior in the presence of a disease.

A common way to incorporate this self-protective behavior is to allow individuals to lower their susceptibility according to the proportion of their contacts that are infectious, as demonstrated by Bagnoli et al. [49]. In this model, the authors reduce the susceptibility of an individual to a disease which has a simple SIS natural history by multiplying the transmission rate by a negative exponential function of the proportion of their neighbors who are infectious. Specifically, this is given by $\beta I(\psi, k)$ where β is the per contact transmission probability and

$$I(\psi, k) = exp\left\{-J\left(\frac{\psi}{k}\right)^{\tau}\right\} \tag{5}$$

models the effect an individual's risk perception has on its susceptibility, where J and τ are constants that govern the level of precaution individuals take, ψ is the number of infectious contacts an individual has, and k is the total number of contacts an individual has.

The authors show that the introduction of adaptive behavior has the potential to not only reduce the probabilities of new infections occurring in highly disease-concentrated areas, but can also cause epidemics to go extinct. Specifically, when $\tau = 1$, there is a value of J for which an epidemic can be stopped in regular lattices and SW networks [25]. However, for certain SF networks, there is no value of J that is able to stop the disease from spreading. In order to achieve disease extinction in these networks, hub nodes must adopt additional self-protective measure, which is accomplished by decreasing τ for these individuals. The conclusions derived from this model highlight the significant impact different types of complex networks can have on health outcomes in a population, and how behavioral changes can dictate the course of an epidemic.

1.4. Organization of this review

The remainder of this review is organized as follows. In Section 2, we will focus on the disease–behavior dynamics of homogeneously mixed populations, and discuss when the homogeneous mixing approximation is or is not valid. This provides a comprehensive prologue to the overview of the coupled systems on networks in Section 3. Within the latter, we separately review dynamics in different types of networked populations, which are frequently viewed through the lens of physical phenomena (such as phase transitions and pattern formation) and analyzed with physics-based methods (like Monte Carlo simulation, mean-field prediction). Based on all these achievements, we can capture how coupled disease–behavior dynamics affects disease transmission and spatial contact patterns. Section 4 will be devoted to empirical concerns, such as types of data that can be used for these study systems, and how questionnaires and digital equipment can be used to collect data on relevant social and contact networks. In addition, it is meaningful to examine whether some social behaviors predicted by models really exist in vaccination experiments and surveys. Finally, we will conclude with a summary and an outlook in Section 5, describing the implications of statistical physics of spatial disease–behavior dynamics and outlining viable directions for future research. Throughout, we will generally focus on preventive measures other than vaccination (such as social distancing and hand washing), although we will also touch upon vaccination in a few places.

2. Disease–behavior dynamics in well-mixed populations

A large body of literature addresses disease–behavior dynamics in populations that are assumed to be mixing homogeneously, and thus spatial structure can be neglected. Incorporating adaptive behavior into a model of disease spread can provide important insight into population health outcomes, as the activation of social distancing and other non-pharmaceutical interventions (NPIs) have been observed to have the ability to alter the course of an epidemic [50–52].

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Table 1
Disease–behavior models applied to well-mixed populations, classified by infection type and whether economic-based or rule-based.

Disease types	Economic epidemiology	Rule-based models
HIV/AIDS	[53–55]	[61–63]
SARS		[64,65]
Influenza		[66,63]
Generic SIR/SIRS	[57,58,60,59]	[68,77,69,71,73,75,76]
Generic SI/SIS	[56]	[67,70,72,74]

When making decisions regarding self-protection from an infection, individuals must gather information relevant to the disease status of others in the population. Prophylactic behavior can be driven by disease prevalence, imitation of others around them, or personal beliefs of probable health outcomes. In this section, we will survey the features and results of mathematical models that incorporate prophylactic decision making behavior in homogeneously mixed populations. The approaches we consider can be classified into two separate categories: economic-based and rule-based. Economic based models (such as game theoretical models) assume individuals seek a maximization of their social utility, whereas rule-based models prescribe prevalence-based rules (not explicitly based on utility) according to which individuals and populations behave. Both of these methods can also be used to study the dynamics of similar diseases (see Table 1), and are discussed in detail below.

2.1. Economic epidemiology models

The discovery of human immunodeficiency virus (HIV)/acquired immune deficiency syndrome (AIDS) and its large economic impacts stimulated research into behaviorally based mathematical models of sexually transmitted diseases (STDs). In disease–behavior models, a population often initiates a behavior change in response to an increasing prevalence of a disease. In the context of STDs, this change in behavior may include safer sex practices, or a reduction in the number of partnerships individuals seek out. Following this prevalence-based decision making principle, researchers have used the concept of utility maximization to study the behavior dynamics of a population [53–57]. In these models, individuals seek to maximize their utility by solving dynamic optimization problems. Utility is derived by members of the population when engaging in increased levels of social contact. However, this increased contact or partner change rate also increases the chance of becoming infected. One consequence of this dynamic is that higher levels of prevalence can result in increased prophylactic behavior, which in turn decreases the prevalence over time. As this occurs, self-protective measures used by the population will also fall, which may cause disease cycles [53,56]. Nonetheless, in the case of STDs which share similar transmission pathways, a population protecting themselves from one disease by reducing contact rates can also indirectly protect themselves from another disease simultaneously [53]. In general, the lowering of contact rates in response to an epidemic can reduce its size, and also delay new infections [57]. However, this observed reduction of contact rates may not be uniform across the whole population. For example, an increase in prevalence may cause the activity rates of those with already low social interaction to fall even further, but this effect may not hold true for those with high activity rates [54]. In fact, the high-risk members of the population will gain a larger fraction of high-risk partners in this scenario, resulting from the low-risk members reducing their social interaction rates. This dynamic serves to increase the risk of infection of high activity individuals even further. These utility-based economic models show us that when considering health outcomes, one must be acutely aware of the welfare costs associated with self-protective behavior or implementing disease mitigation policies [56]. A health policy, such as encouraging infectious individuals to self-quarantine, may actually cause a rise in disease prevalence due to susceptible individuals feeling less threatened by infection and subsequently abandoning their own self-protective behavior [56]. Also, a population who is given a pessimistic outlook of an epidemic may in fact cause the disease to spread more rapidly [55].

Recently, approaches using game theory have been applied to self-protective behavior and social distancing [58–60]. When an individual's risk of becoming infected only depends on their personal investment into social distancing, prophylactic behavior is not initiated until after an epidemic begins, and ceases before an epidemic ends. Also, the basic reproductive number of a disease must exceed a certain threshold for individuals to feel self-protective behavior is worth the effort [58]. In scenarios where the contact rate of the population increases with the number of

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people out in public, a Nash Equilibrium exists, but the level of self-protective behavior in it is not socially optimal [59]. Nonetheless, these models also show that the activation of social distancing can weaken an epidemic.

2.2. Rule-based models

Some models of disease–behavior dynamics, rather than assuming humans are attempting to optimize a utility function, represent human behavior by specifying rules that humans follow under certain conditions. These could include both phenomenological rules describing phenomenological responses to changes in prevalence, or more complex psychological mechanisms. Rule-based compartmental models using systems of differential equations have also used to study heterogeneous behavior and the use of NPIs by a population during an epidemic. A wide range of diseases are modeled using this approach, such as HIV [61–63], severe acute respiratory syndrome (SARS) [64,65], or influenza [66,63]. These models often utilize additional compartments, which are populated according to specific rules. Examples of such rules are to construct the compartments to hold a constant amount of individuals associated with certain contact rates [61,62,67], or to add and remove individuals at a constant rate [64,65,63,68], a rate depending on prevalence [69–74], or according to a framework where behavior that is more successful is imitated by others [75,66,76]. Extra compartments signify behavioral heterogeneities amongst members of a population, and the disease transmission rates associated with them also vary. Reduction in transmission due to adaptive behavior is either modeled as a quarantine of cases [64,65,63], or prophylactic behavior of susceptible individuals due to increased awareness of the disease [75,66,69–74,77]. These models agree that early activation of isolation measures and self-protective behavior can weaken an epidemic. However, due to an early decrease in new infections, populations may see a subsequent decrease in NPI use causing multiple waves of infection [69,75,76,71]. Contrasting opinions on the impact behavioral changes have on the epidemic threshold also result from these models. For example, Perra et al. [71] show that although infection size is reduced, prophylactic behavior does not alter the epidemic threshold. However, the models studied by Poletti et al. [75] and Sahneh et al. [70] show that the epidemic threshold can be altered by behavioral changes in a population.

2.3. A brief summary and potential limitation of well-mixed populations

The classes of models presented in this section use homogeneous mixing patterns (i.e., well-mixed populations) to study the effects of adaptive behavior in response to epidemics and disease spread (see Table 1 for a summary). Often, populations will be modeled to alter their behavior based on reactions to changes in disease prevalence, or by optimizing their choices with respect to personal health outcomes. If possible, early activation of prophylactic behavior and NPIs by a population will be the most effective course of action to curb an epidemic.

Homogeneous mixing can be an appropriate approximation for the spread of an epidemic when the disease to be modeled is easily transmitted, such as measles and other infection that can be spread by fine aerosol particles that remain suspended for a long period. However, this mixing assumption does not always reflect real disease dynamics. For example, human sexual contact patterns are believed to be heterogeneous [48] and can be represented as networks (or graphs), while other infections, such as SARS, can only be spread by large droplets, making the homogeneous mixing assumption less valid. The literature surrounding epidemic models that address this limitation by incorporating heterogeneous contact patterns through networks is very rich, and is discussed in the following section.

3. Disease–behavior dynamics in networked populations

In Section 2, we reviewed disease–behavior dynamics in well-mixed populations. However, in real populations, various types of complex networks are ubiquitous and their dynamics have been well studied. The transmission of many infectious diseases requires direct or close contact between individuals, suggesting that complex networks play a vital role in diffusion of disease. It thus becomes of particular significance to review the development of behavioral epidemiology in networked populations. Many of the dynamics exhibited by such systems have direct analogues to processes in statistical physics, such as how disease or behavior percolate through the network, or how a population can undergo a phase transition from one social state to another social state.

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3.1. Dynamics on lattices and static networks

Perhaps the easiest way to begin studying disease–behavior dynamics in spatially distributed populations is by using lattices and static networks, which are relatively easy to analyze and which have attracted much attention in theoretical and empirical research. We organize research by several themes under which they have been conducted, such as the role of spreading awareness, social distancing as protection, and the role of imitation, although we emphasize that the distinctions are not always "hard and fast".

The role of individual awareness. The awareness of disease outbreaks may stimulate humans to change their behavior, such as washing hands and wearing masks. Such behavioral responses can reduce susceptibility to infection, which itself in turn can influence the epidemic course. In the seminal work, Funk and coworkers [78] formulated and analyzed a mathematical model for the spread of awareness in well-mixed and spatially structured populations to understand how the awareness of disease and also its propagation impact the spatial spread of a disease. In their model, both disease and the information about the disease spread spontaneously by, respectively, contact and word of mouth in the population. The classical epidemiological SIR model is used for epidemic spreading, and the information dynamics is governed by both information transmission and information fading. The immediate outcome of the awareness of the disease information is the decrease in the possibility of acquiring the infectious disease when a susceptible individual (who was aware of the epidemic) contacts with an infected one. In a well-mixed population, the authors found that, the coupling spreading dynamics of both the epidemic and the awareness of it can result in a lower size of the outbreak, yet it does not affect the epidemic threshold. However, in a population located on the triangular lattice, the behavioral response can completely stop a disease from spreading, provided the infection rate is below a threshold. Specifically, the authors showed that the impact of locally spreading awareness is amplified if the social network of potential infection events and the communication network over which individuals communicate overlap, especially so if the networks have a high level of clustering.

The finding that spatial structure can prevent an epidemic is echoed in an earlier model where the effects of awareness are limited to the immediate neighbors of infected nodes on a network [79]. In the model, individuals choose whether to accept ring vaccination depending on perceived disease risk due to infected neighbors. By exploring a range of network structures from the limit of homogeneous mixing to the limit of a static, random network with small neighborhood size, the authors show that it is easier to eradicate infections in spatially structured populations than in homogeneously mixing populations [79]. Hence, free-riding on vaccine-generated herd immunity may be less of a problem for infectious diseases spreading spatially structured populations, such as would more closely describe the situation for close contact infections.

Along similar lines of research, Wu et al. explored the impact of three forms of awareness on the epidemic spreading in a finite SF networked population [80]: contact awareness that increases with individual contact number; local awareness that increases with the fraction of infected contacts; and global awareness that increases with the overall disease prevalence. They found that the global awareness cannot decrease the likelihood of an epidemic outbreak while both the local awareness and the contact awareness do it. Generally, individual awareness of an epidemic contributes toward the inhibition of its transmission.

The universality of such conclusions (i.e., individual behavioral responses suppress epidemic spreading) is also supported by a recent model [81], in which the authors focused on an epidemic response model where the individuals respond to the epidemic according to, rather than the density of infected nodes, the number of infected neighbors in the local neighborhood. Mathematically, the local behavioral response is cast into the reduction factor $(1-\theta)^{\psi}$ in the contact rate of a susceptible node, where ψ is the number of infected neighbors and $\theta < 1$ is a parameter characterizing the response strength of the individuals to the epidemic. By studying both SIS and SIR epidemiological models with the behavioral response rule in SF networks, they found that individual behavioral response can in general suppress epidemic spreading, due to crucial role played by the hub nodes who are more likely to adopt protective response to block the disease spreading path.

In a somewhat different framework, how the diffusion of individual's crisis awareness affects the epidemic spreading is investigated in Ref. [82]. In this work, the epidemiological SIR model is linked with an information transmission process, whose diffusion dynamics is characterized by two parameters, say, the information creation rate ζ and the information sensitivity η. In particular, at each time step, ζN packets will be generated and transferred in the network according to the shortest-path routing algorithm (N hither denotes the size of networks). When a packet is routed by an

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infected individual, its state is marked by infection. Each individual determines whether or not to accept vaccine based on how many infected packets are received from immediate neighbors, and on how sensitive the individual response is to the information as well, weighed by the parameter η. The authors considered their "SIR with information-driven vaccination" model on homogeneous ER networks and heterogeneous SF networks, and found that the epidemic spreading can be significantly suppressed in both the homogeneous and heterogeneous networks provided that both ζ and η are relatively large.

Social distancing as a protection mechanism. Infectious disease outbreaks may trigger various behavioral responses of individuals to take preventive measures, one of which is social distancing. Valdez and coworkers have investigated the efficiency of social distancing in altering the epidemic dynamics and affecting the disease transmission process on ER network, SF networks, as well as realistic social networks [83]. In their model, rather than the normally used link-rewiring process, an intermittent social distancing strategy is adopted to disturb the epidemic spreading process. Particularly, based on local information, a susceptible individual is allowed to interrupt the contact with an infected individual with a probability σ and restore it after a fixed time t_b, such that the underlying interaction network of the individuals remains unchanged. Using the framework of percolation theory, the authors found that there exists a cutoff threshold σ_c, whose value depends on the network topology (i.e., the extent of heterogeneity of the degree distribution), beyond which the epidemic phase disappears. The efficiency of the intermittent social distancing strategy in stopping the spread of diseases is owing to the emergent "susceptible herd behavior" among the population that protects a large fraction of susceptible individuals.

Impact of behavior imitation on vaccination coverage. Vaccination is widely employed as an infection control measure. To explore the role of individual imitation behavior and population structure in vaccination, recent seminal work integrated an epidemiological process into a simple agent-based model of adaptive learning, where individuals use anecdotal evidence to estimate costs and benefits of vaccination [85]. Under such a model, the disease–behavior dynamics is modeled as a two-stage process. The first stage is a public vaccination campaign, which occurs before any epidemic spreading. At this stage, each individual decides whether or not to vaccinate, and taking vaccine incurs a cost C_V to the vaccinated individuals. The vaccine is risk-free and offers perfect protection against infection. The second stage is the disease transmission process, where the classic SIR compartmental model is adopted. During the whole epidemic spreading process, those susceptible individuals who caught the disease incur an infection cost C_I, which is usually assumed to be larger than the cost C_V for vaccination. Those unvaccinated individuals who remain healthy are free-riding off the vaccination efforts of others (i.e., no any cost), and they are indirectly protected by herd immunity. For simplicity, the authors rescale these costs by defining the relative cost of vaccination $c = C_V/C_I$ $(0 < c < 1)$ and $C_I = 1$. As such, after each epidemic season, all the individuals will get some payoffs (equal to the negative value of corresponding costs) dependent on their vaccination strategies and also on whether they are infected or not, then they are allowed to change or keep their old strategies for the next season, depending on their current payoffs. The rule of thumb is that the strategy of a role model with higher payoff is more likely to be imitated. By doing so, each individual i randomly chooses another individual j from the neighborhood as role model, and imitates the behavior of j with the probability

$$\Pi_{i\to j} = \frac{1}{1+\exp[-\beta(P_j - P_i)]}, \tag{6}$$

where P_i and P_j are, respectively, the payoffs of two involved individuals, and β $(0 < \beta < \infty)$ denotes the strength of selection. This form of decision alternative is also known as the Fermi law [16,86] in physics. A finite value of β accounts for the fact that better performing individuals are readily imitated, although it is not impossible to imitate one agent performing worse, for example due to imperfect information or errors in decision making.

The authors studied their coupled "disease–behavior" model in well-mixed populations, in square lattice populations, in random network populations, and in SF network populations, and found that population structure acts as a "double-edged sword" for public health: it can promote high levels of voluntary vaccination and herd immunity given that the cost for vaccination is not too large, but small increases in the cost beyond a certain threshold would cause vaccination to plummet, and infections to rise, more dramatically than in well-mixed populations. This research provides an example of how spatial structure does not always improve the chances of infection control, in disease–behavior systems.

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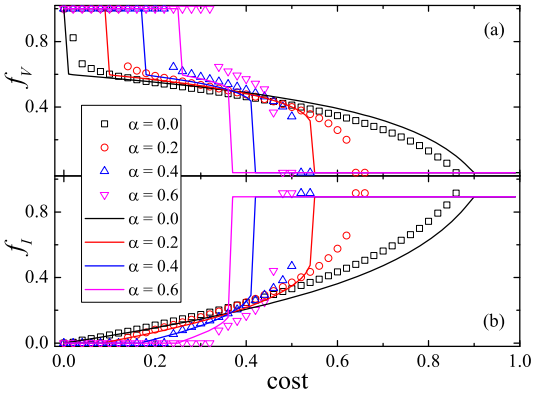

Fig. 3. The faction of vaccinated individuals f_V (a) and infected individuals f_I (b) as a function of vaccine cost in the ER random graph. The symbols and lines correspond, respectively, to the simulation results and mean-field predictions (whose analytical framework is shown in Appendix A). The parameter α determines just how seriously the peer pressure is considered in the decision making process of the individuals to taking vaccine. The figure is reproduced from [84].

In the similar vein, peer pressure among the populations is considered to clarify its impact on the decision-making process of vaccination, and then on the disease spreading [84]. In reality, whether or not to change behavior depends not only on the personal success of each individual, but also on the success and/or behavior of others. Using this as motivation, the authors incorporated the impact of peer pressure into a susceptible-vaccinated-infected-recovered (SVIR) epidemiological model, where the propensity to adopt a particular vaccination strategy depends both on individual success as well as on the strategy-configuration of their neighbors. To be specific, the behavior imitation probability of individual i towards its immediate neighbor j (namely, Eq. (6)) becomes

$$\Pi_{i\to j} = (N_i/k_i)^{\alpha}\frac{1}{1+\exp[-\beta(P_j - P_i)]}, \tag{7}$$

where N_i is the number of neighbors that have a different vaccination strategy than the individual i, and k_i is the interaction degree of i, and the parameter α determines just how seriously the peer pressure is considered. Under such a scenario, Fig. 3 displays how vaccination and infection vary as a function of vaccine cost in ER random graph. It is clear that plugging into the peer pressure also works as a "double-edged sword", which, on the one hand, strongly promotes vaccine uptake in the population when its cost is below a critical value, but, on the other hand, it may also strongly impede it if the critical value is exceeded. The reason is due to the fact that the presence of peer-pressure can facilitate cluster formation among the individuals, whose behaviors are inclined to conform to the majority of their neighbors, similar to the early report of cooperation behavior [88]. Such behavioral conformity is found to expedite the spread of disease when the relative cost for vaccination is high enough, while promote the vaccine coverage in the opposite case.

Self-motivated strategies related with vaccination. Generally, it is not so much the actual risk of being infected, as the perceived risk of infection, that will prompt humans to change their vaccination behavior. Previous game-theoretic studies of vaccination behavior typically have often assumed that individuals react to the disease incidence with same responsive dynamics, i.e., the same formulas of calculating the perceived probability of infection. But that may not actually be the case. Liu et al. proposed that a few will be "committed" to vaccination, perhaps because they have a low threshold for feeling at risk (or strongly held convictions), and they will want to be immunized as soon as they hear that someone is infected [87]. They studied how the presence of committed vaccinators, a small fraction of individuals who consistently hold the vaccinating strategy and are immune to influence, impacts the vaccination dynamics in well-mixed and spatially structured populations. The researchers showed that even a relatively small proportion of these agents (such as 5%) can significantly reduce the scale of an epidemic, as shown in Fig. 4. The effect is much stronger when all the individuals are uniformly distributed on a square lattice, as compared to the case of well-mixed population. Their results suggested that those committed individuals can have a remarkable effect, acting as "steadfast role models" in the population to seed vaccine uptake in others while also disrupting the appearance of clusters of free-riders, which might otherwise seed the emergence of a global epidemic.

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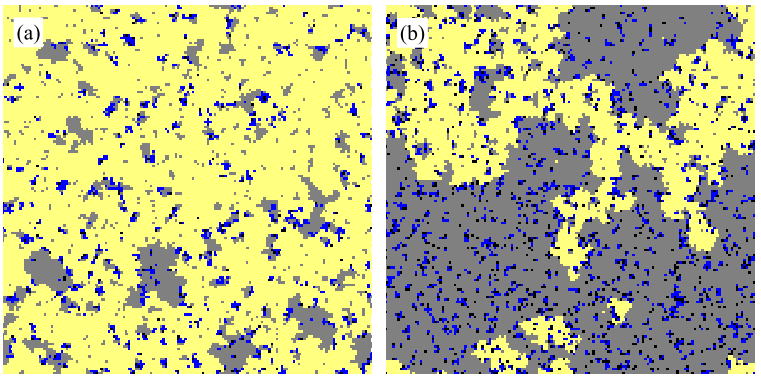

Fig. 4. Typical snapshots of the state configuration of the square lattice population after an epidemic season. The fraction of committed individuals (denoted by black), who always take vaccine, is 1% in (a) and 5% in (b). Other parameters are the same, and blue (dark gray) is for vaccinated individuals, gray successful free-riders, and yellow (light gray) infected individuals, respectively.
The figure is reproduced from [87].

One important message taken away from Ref. [87] is that we might never guess what would happen by just looking at the decision-making rules alone, in particular when our choices will influence, and be influenced by, the choice of other people. Another good example can be found in a recent work [89], in which Zhang et al. proposed an evolutionary epidemic game where individuals can choose their strategies as vaccination, self-protection or laissez faire, towards infectious diseases and adjust their strategies according to their neighbors' strategies and payoffs. The "disease–behavior" coupling dynamical process is similar to the one implemented by Ref. [85], where the SIR epidemic spreading process and the strategy updating process succeed alternatively. By both stochastic simulations and theoretical analysis, the authors found a counter-intuitive phenomenon that a better condition (i.e., larger successful rate of self-protection) may unfortunately result in less system payoff. The trick is that, when the successful rate of self-protection increases, people become more speculative and less interested in vaccination. Since a vaccinated individual brings the "externality" effect to the system: the individual's decision to vaccinate diminishes not only its own risk of infection, but also the risk for those people with whom the individual interacts, the reduction of vaccination can remarkably enhance the risk of infection. The observed counter-intuitive phenomenon is reminiscent of the well-known Braess's Paradox in traffic, where more roads may lead to more severe traffic congestion [90]. This work provides another interesting example analogous to Braess's Paradox, namely, a higher successful rate of self-protection may eventually enlarge the epidemic size and thus diminish positive health outcomes.

This work raises a challenge to public health agencies regarding how to protect the population during an epidemic. The government should carefully consider how to distribute their resources and money between messages supporting vaccination, hospitalization, self-protection, and so on, since the outcome of policy largely depends on the complex interplay among the type of incentive, individual behavioral responses, and the intrinsic epidemic dynamics. In their further work [91], the authors investigated the effects of two types of incentives strategies, partial-subsidy policy in which certain fraction of the cost of vaccination is offset, and free-subsidy policy in which donees are randomly selected and vaccinated at no cost on the epidemic control. Through mean-field analysis and computations, they found that, under the partial-subsidy policy, the vaccination coverage depends monotonically on the sensitivity of individuals to payoff difference, but the dependence is non-monotonous for the free-subsidy policy. Due to the role models of the donees for relatively irrational individuals and the unchanged strategies of the donees for rational individuals, the free-subsidy policy can in general lead to higher vaccination coverage. These findings substantiate, once again, that any disease-control policy should be exercised with extreme care: its success depends on the complex interplay among the intrinsic mathematical rules of epidemic spreading, governmental policies, and behavioral responses of individuals.

3.2. Dynamics in multilayer networks

As the above subsection shows, research on disease–behavior dynamics on networks has become one of the most fruitful realms of statistical physics and non-linear science, as well as shedding novel light on how to predict the

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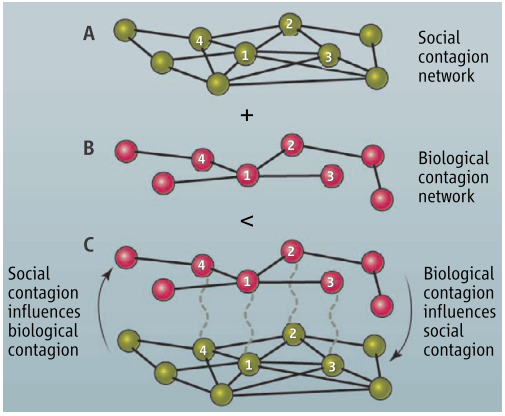

Fig. 5. Schematic illustration of multilayer architecture composed of two networks. Though the social network (namely, Network A) and infection contact network (namely, Network B) possess the same nodes marked by numbers, they support different dynamic processes, which are separately studied in most previous literature. Now, if both networks are encapsulated into a multilayer framework (namely, Network C), the interaction between them may create completely different outcomes that go beyond what isolated networks can capture.
The figure is reproduced from [101].

impact of individual behavior on disease spread and prevention [92–94,85,95–99,79]. However, in some scenarios, the simple hypothesis that individuals are connected to each other in the same infrastructure (namely, the so-called single-layer network in Section 3.1) may generate overestimation or underestimation for the diffusion and prevention of disease, since agents can simultaneously be the elements of more than one network in most, yet not all, empirical systems [29,28,100]. In this sense, it seems constructive to go beyond the traditional single-layer network theory and propose a new architecture, which can incorporate the multiple roles or connections of individuals into an integrated framework. The multilayer networks, defined as the combination class of networks interrelated in a nontrivial way (usually by sharing nodes), have recently become a fundamental tool to quantitatively describe the interaction among network layers as well as between these constituents.

An example of multilayer networks is visualized in Fig. 5 [101]. A social network layer supports the social dynamics related to individual behavior and main prevention strategies (like vaccination); while the biological layer provides a platform for the spreading of biological disease. Each individual is a node in both network layers. The coupled structure can generate more diverse outcomes than either isolated network, and could produce multiple (positive or negative) effects on the eradication of infection. Because of the connection between layers, the dynamics of control measures in turn affects the trajectory of disease on biological network, and *vice versa*.

Under such a framework, which is composed of at least 2 different topology networks, nodes not only exchange information with their counterparts in other network(s) via inter-layer connections, but also diffuse infection with their neighbors through the intra-layer connections. Subsequently, more theoretical algorithms and models, such as interdependent networks, multiplex networks and interconnected networks, have been proposed [102–104]. The broad applicability of multilayer networks and their success in providing insight into the structure and dynamics of realistic systems have thus generated considerable excitement [105–109]. Of course, the study of disease–behavior dynamics in this framework is a young and rapidly evolving research area, which will be systematically surveyed in what follows.

Interplay between awareness and disease. As Fig. 5 illustrates, different dynamical processes for the same set of nodes with different connection topologies for each process can be encapsulated in a multilayer structure (technically, these are referred to as multiplex networks [28,29]). Aiming to explore the interrelation between social awareness and disease spreading, Granell et al. recently incorporated information awareness into a disease model embedded in a multiplex network [110], where the physical contact layer supports epidemic process and the virtual contact layer supports awareness diffusion. Similar to SIS model (where the S node can be infected with a transmission probability β, and the I node recovers with a certain rate μ), the awareness dynamics, composed of aware (A) and unaware (U) states, assumes that a node of state A may lose its awareness with probability δ, and re-obtains awareness in the probability ν. Then, both processes can be coupled via the combinations of individual states: unaware-susceptible

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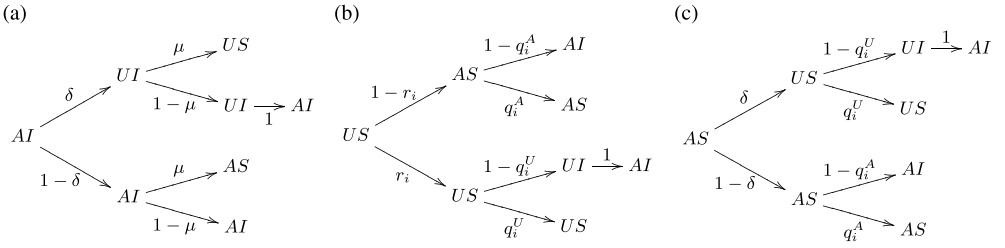

Fig. 6. Transition probability trees of the combined states for coupled awareness–disease dynamics each time step in the multilayer networks. Here aware (A) state can become unaware (U) with transition probability δ and of course re-obtains awareness with other probability. For disease, μ represents the transition probability from infected (I) to susceptible (S). There are thus four state combinations: aware-infected, (AI) aware-susceptible, (AS) unaware-infected, (UI) and unaware-susceptible (US), and the transition of these combinations is controlled by probability r_i, q_i^A and q_i^U. They respectively denote the transition probability from unaware to aware given by neighbors; transition probability from susceptible to infected, if node is aware, given by neighbors; and transition probability from susceptible to infected, if node is unaware, given by neighbors. We refer to [110], from where this figure has been adapted, for further details.

(US), aware-susceptible (AS), and aware-infected (AI), which are also revealed by the transition probability trees in Fig. 6.

Using Monte Carlo simulations, the authors showed that the coupled dynamical processes change the onset of the epidemics and allow them to further capture the evolution of the epidemic threshold (depending on the structure and the interrelation with the awareness process), which can be accurately validated by the Markov-chain approximation approach. More interestingly, they unveiled that the increase in transmission rate can lower the long-term disease incidence while raising the outbreak threshold of epidemic.

In spite of great progress, the above-mentioned findings are based on two hypotheses: infected nodes become immediately aware, and aware individuals are completely immune to the infection. To capture more realistic scenarios, the authors relaxed both assumptions and introduced mass media that disseminates information to the entire system [111]. They found that the vaccine coverage of aware individuals and the mass media affect the critical relation between two competing processes. More importantly, the existence of mass media makes the metacritical point (where the critical onset of the epidemics starts) of Ref. [110] disappear. Furthermore, the social dynamics are further extended to an awareness cascade model [112], during which agents exhibit herd-like behavior because they make decisions referring to the actions of other individuals. Interestingly, it is found that a local awareness ratio (of unaware individuals becoming aware ones) approximating 0.5 has a two-stage effect on the epidemic threshold (i.e., an abrupt transition of epidemic threshold) and can cause different epidemic sizes, irrespective of the network structure. That is to say, when the local awareness ratio is in the range of $[0, 0.5)$, the epidemic threshold is a fixed and larger value; however, in the range of $[0.5, 1]$, threshold value becomes a fixed yet smaller value. As for the final epidemic size, its increasing speed for the interval $[0, 0.5)$ is much slower than the speed when local awareness ratio lies in $[0.5, 1]$. These findings suggest a new way of understanding realistic contagions and their prevention. Except for obtaining awareness from aware neighbors, self-awareness induced by infected neighbors is another scenario that currently attracts research attention [113], where it is found that coupling such a dynamical process with disease spreading can lower the density of infection, but does not increase the epidemic threshold regardless of the information source.

Coupling between disease and preventive behaviors. Thus far, many achievements have shown that considering simultaneous diffusion of disease and prevention measures on the same single-layer network is an effective method to evaluate the incidence and onset of disease [94,85,95–98,114,79]. However, if both processes are coupled on the multilayer infrastructure, how does it affect the spreading and prevention of disease? Inspired by this interesting question, Ref. [115] suggested a conceptual framework, where two fully or partially coupled networks are employed, to transmit disease (an infection layer) and to channel individual decision of prevention behaviors (a communication layer). Protection strategies considered include wearing facemasks, washing hands frequently, taking pharmaceutical drugs, and avoiding contact with sick people, which are the only means of control in situations where vaccines are not yet available. It is found that the structure of the infection network, rather than the communication network, has a dramatic influence on the transmission of disease and uptake of protective measures. In particular, during an influenza epidemic, the coupled model can lead to a lower infection rates, which indicates that single-layer models may overestimate disease transmission.

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In line with this finding, the author further extended the above setup into a triple coupled diffusion model (adding the information flow of disease on a new layer) through metropolitan social networks [116]. During an epidemic, these three diffusion dynamics interact with each other and form negative and positive feedback loop. Compared with the empirical data, it is exhibited that this proposed model reasonably replicates the realistic trends of influenza spread and information propagation. The author pointed out that this model possesses the potential of developing into a virtual platform for health decision makers to test the efficiency of disease control measures in real populations.

Much previous work shows that behavior and spatial structure can suppress epidemic spreading. In contrast, other recent research using a multiplex network consisting of a disease transmission (DT) network and information propagation (IP) network through which vaccination strategy and individual health condition information can be communicated, finds that compared with the case of traditional single-layer network (namely, symmetric interaction), the multiplex architecture suppresses vaccination coverage and leads to more infection [117]. This phenomenon is caused by the sharp decline of small-degree vaccination nodes, whose number is usually more numerous in heterogeneous networks.

Similarly, Wang et al. considered asymmetrical interplay between disease spreading and information diffusion in multilayer networks [118]. It is assumed that there exists different disease dynamics on communication layer and physical-contact layer, only where vaccination takes place. More specifically, the vaccination decision of the node in contact networks is not only related to the states of its intra-layer neighbors, but also depends on the counterpart node from communication layer. By means of numerous simulations and mean-field analysis, they found that, for uncorrelated coupling architecture, a disease outbreak in the contact layer induces an outbreak of disease in the communication layer, and information diffusion can effectively raise the epidemic threshold. However, the consideration of inter-layer correlation dramatically changes the onset of disease, but not the information threshold.

3.3. Dynamics on adaptive networks and time-varying networks

Dynamical networks play an important role in the incidence and onset of epidemics as well. Along this line of research, the most commonly used approach is adaptive networks [119–122], where nodes frequently adjust their connections according to the environment or states of neighboring nodes. Time-varying networks (also named temporal networks) provide another framework for the activity-driven changing of connection topology [31,123,32]. Here, we briefly review the progress of disease–behavior dynamics on adaptive and time-varying networks.

Contact switching as potential protection strategy. In the adaptive viewpoint, the most straightforward method of avoiding contact with infective acquaintances amounts to breaking the links between susceptible and infective agents and constructing novel connections. Along such lines, Thilo et al. first proposed an adaptive scenario: a susceptible node is able to prune the infected link and rewire with a healthy agent with a certain probability [124]. The probability of switching can be regarded as a measurement of strength of the protection strategy. It is shown that different values of this probability give rise to various degree mixing patterns and degree distributions. Based on the low-dimensional approximations, the authors also showed that their adaptive framework is able to predict novel dynamical features, such as bistability, hysteresis, and first order transitions, which are sufficiently robust against disease dynamics [125,126]. In spite of great advances, the existing analytical methods cannot generally allow for accurate predictions about the simultaneous time evolution of disease and network topology. To overcome this limitation, Vincent et al. further introduced an improved compartmental formalism, which proves that the initial conditions play a crucial role in disease spreading [127].

In the above examples, switching contact as a strategy has proven its effectiveness in controlling epidemic outbreak. However, in some realistic cases, the population information may be asymmetric, especially during the process of rewiring links. To relax this constraint, a new adaptive algorithm was recently suggested: an infection link can be pruned by either individual, who reconnects to a randomly selected member rather than susceptible agent (namely, the individual has no previous information on state of every other agent) [128,129]. For example, Ref. [129] showed that such a reconnection behavior can completely suppress the spreading of disease via continuous and discontinuous transitions, which is universally effective in more complex situations.

Besides the phenomena of oscillation and bistability, another dynamical feature, epidemic reemergence, also attracts great interest in a current study [122], where susceptible individuals adaptively break connections with infected neighbors yet avoid being isolated in a growing network. Under such operations, the authors observed that the number

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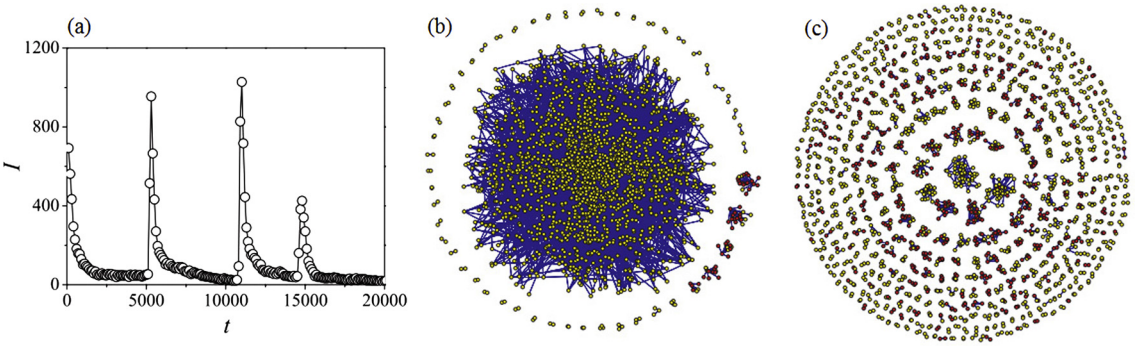

Fig. 7. Panel (a) denotes the time course for the number of infected nodes when the network growth, the link-removal process, and isolation avoidance are simultaneously involved into the adaptive framework. It is clear that this mechanism creates the reemergence of epidemic, which dies out after several such repetitions. While for this interesting phenomenon, it is closely related with the formation of giant component of susceptible nodes. Panel (b) shows the snapshot of the network topology of 5000th time step (before the next abrupt outbreak), when there is a giant component of susceptible nodes (yellow). However, the invasion of the infection individuals (red) makes the whole network split into many fragments, as shown by the snapshot of 5400th time step (after the explosion) in panel (c). We refer to [122], from where this figure has been adapted, for further details. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

of infected agents stays at a low level for a long time, and then suddenly erupts to high level before declining to a low level again. This process repeats several times until the final eradication of infection, as illustrated in Fig. 7(a). With regard to potential mechanism, it is actually related with the invasion of infected individuals to susceptible giant components. Link-removal process can suppress disease spreading, which makes susceptible (infected) agents form giant components (small yet non-isolated clusters), as shown in Fig. 7(b). But, the entrance of new nodes may bring new infection risk to such giant components, which accelerates next outbreak of infection and network crashing again (see Fig. 7(c)). Interestingly, this finding may help to explain the phenomenon of repeated epidemic explosions in real populations.

Now, if we carefully look back upon the above-cited bibliography, we will find a common feature: except for disease processes, the adaptive adjustment of individual connections finally changes the degree distribution of networks. An interesting question naturally poses itself: is there an adaptive scenario that preserves the degree distribution of networks? That is, each individual has a characteristic behavior: keeping total number of its neighbors constant. To fill up this gap, neighbor exchange model becomes a very useful tool [130], where individual number of current neighbors remains fixed while the compositions or entities of those contacts change in time. Similar to famous algorithm of Watts–Strogatz SW network [25], such a model allows an exchange mechanism in which the destination nodes of two edges are swapped with a given rate. Incorporating the diffusion of epidemic, this model constructs a bridge between static network model and mass-action model. Based on the empirical data, the authors further displayed that the application of this model is very effective to forecast and control sexually transmitted disease outbreak. Along this way, the potential influence of other topology properties (such as growing networks [131] and rewiring SF networks [132]) has recently been identified in the adaptive viewpoint, which dramatically changes the outbreak threshold of disease.

Vaccination, immunization and quarantine as avoidance behaviors. As in static networks, vaccination can also be introduced into adaptive architectures, where connection adjustment is an individual response to the presence of infection risk among neighborhoods. Motivated by realistic immunization situations, disease prevention is implemented by adding Poisson-distributed vaccination to susceptible individuals [133]. Because of the interplay between network rewiring and vaccination application, the authors showed that vaccination is far more effective in an adaptive network than a static one, irrespective of disease dynamics. Similarly, some other control measures are further encapsulated into adaptive community networks [134]. Except for various transition of community structure, both immunization and quarantine strategies show a counter-intuitive result that it is not "the earlier, the better" for prevention of disease. Moreover, it is unveiled that the prevention efficiency of both measures is greatly different, and the optimal effect is obtained when a strong community structure exists.

Vaccination on time-varying networks. In contrast to the mutual feedback between dynamics and structure in adaptive frameworks, time-varying networks provide a novel angle for network research, where network connection and

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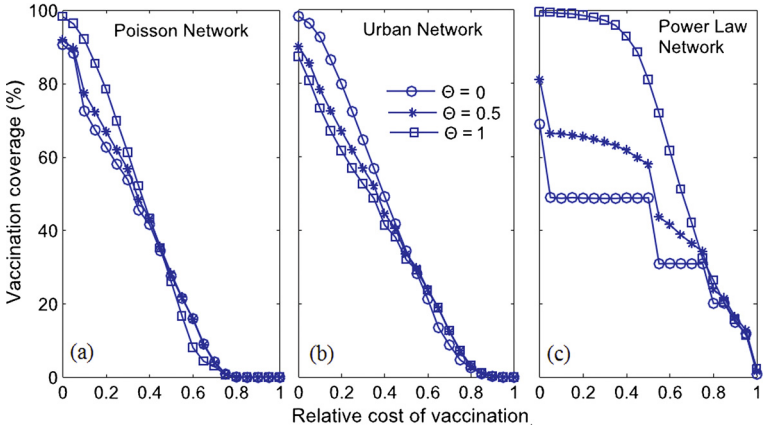

Fig. 8. Vaccination coverage as a function of the relative cost of vaccination and the fraction of imitators Θ in different networks. It is obvious that for small cost of vaccination, imitation behavior increases vaccination coverage but impedes vaccination at high cost, irrespective of potential interaction topology.
The figure is reproduced from [93].

dynamics process evolve according to their respective rules [135–137]. For example, Summin et al. recently explored how to lower the number of vaccinated people to protect the whole system on time-varying networks [138]. Based on the past information, they could accurately administer vaccination and estimate disease outbreaks in future, which proves that time-varying structure can make protection protocols more efficient. In [139], the authors displayed that limited information on the contact patterns is sufficient to design efficient immunization strategies once again. But in these two works, the vaccination strategy is somewhat independent of human behavior and decision-making process, which leaves an open issue: if realistic disease–behavior dynamics is introduced into time-varying topology (especially combining with the diffusion process of opinion cluster [140]), how does it affect the eradication of disease? We continue to discuss some of these and similar issues in Section 4 on empirically-derived networks.

3.4. Dynamics on empirically derived networks

Some research uses networks derived from empirical data in order to examine disease–behavior dynamics. We discuss these models in this subsection.

Dynamics on different topologies. Heterogeneous contact topology is ubiquitous in reality. To test its potential impact on disease spreading, Martial et al. recently integrated a behavior epidemiology model with decision-making process into three archetypical realistic networks: Poisson Network, Urban Network and Power Law Network [93]. Under these contact networks, an agent can make decision either based purely on payoff maximization or via imitating the vaccination behavior of its neighbor (as suggest by Eq. (6)), which is controlled by the fraction of imitators Θ. By means of numerous simulations, they displayed the diploid effect of imitation behavior: it enhances vaccination coverage for low vaccination cost, but impedes vaccination campaign at relatively high cost, which is depicted by Fig. 8. Surprisingly, in spite of high vaccination coverage, imitation can generate the clusters of non-vaccinating, susceptible agents, which in turn accelerate the large-scale outbreak of infectious disease (namely, imitation behavior, to some extent, impedes the eradication of infectious diseases). This point helps to explain why outbreaks of measles have recently occurred in many countries with high overall vaccination coverage [140,143,144]. With the same social networks, Ref. [141] explored the impact of heterogeneous contact patterns on disease outbreak in the compartmental model of SARS. It is interesting that, compared with the prediction of well-mixed population, the same set of basic reproductive number may lead to completely epidemiological outcomes in any two processes, which sheds light to the heterogeneity of SARS around the world.

Impact of network mixing patterns. As Ref. [93] discovered, high vaccination coverage can guarantee herd immunity, which, however, is dramatically affected and even destroyed by clusters of unvaccinated individuals. To evaluate how much influence such clusters possess, a recent work explored the distribution of vaccinated agents during seasonal influenza vaccination through a United States high school contact network [142]. The authors found that contact

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Table 2
Classification of disease–behavior research outcomes according to dynamic characteristics in networked populations reviewed by Section 3. It is clear that the same type of networks can be frequently used to different problems.

Used network types	Disease–behavior dynamics characteristics	Example Refs.
Social network ER network SF network Multilayer networks	awareness changing disease outbreak or (and) individual prevention measures	[79,78,80,82,110,81, 111–113]
Square lattice Random network SF network Urban network	learning rules deciding disease status	[85,84,93,117]
Square lattice ER network SF network Adaptive network Multilayer networks Social network	social/self-motivated protection mechanisms impacting disease status or (and) topology structure	[124,79,130,128,125,126, 129,133,83,87,89,91,115, 116,122,127,132]
Time-varying network	independent evolution of both disease and behavior processes	[138,139]
Adaptive network Multilayer networks Social network	topology properties determining disease status or (and) individual behavior	[93,117,118,131,134,141, 142]

Table 3
Observed physical phenomena and frequently used methods in the study of disease–behavior dynamics on networks.

Examples of critical phenomena	Typical analysis methods
Epidemic threshold	mean-field prediction
Phase transition	generation function
Self-organization	percolation theory
Pattern formation	stochastic processes
Bifurcation and stability analysis	Monte Carlo simulation
Vaccination/immunization threshold	Markov-chain approximation

networks are positively assortative with vaccination behavior. That is to say, large-degree unvaccinated (vaccinated) agents are more likely to contact with other large-degree unvaccinated (vaccinated) ones, which certainly results in a larger outbreak than common networks since these (positively assortative) unvaccinated agents breed larger susceptible clusters. This finding highlights the importance of heterogeneity during vaccine uptake for the prevention of infectious disease once again.

In fact, the currently growing available human generated data and computing power have driven the fast emergence of various social, technological and biological networks [145–148]. Upon these empirically networks, mass disease–behavior models can be considered to analyze the efficiency of existing or novel proposed prevention measures and provide constructive viewpoint for policy makers of public health [149–154].

3.5. Classification of disease–behavior dynamics in networked populations

Based on the above achievements, it is now clear that incorporating behavior epidemiology into networked populations has opened a new window for the study of epidemic transmission and prevention. To capture an overall image, Table 2 provides a summary for the reviewed characteristics of disease–behavior dynamics in networked populations. Here it is worth mentioning that some works (e.g., [93,117]) may appear in two categories because they simultaneously consider the influence of individual behavior and special network structure.

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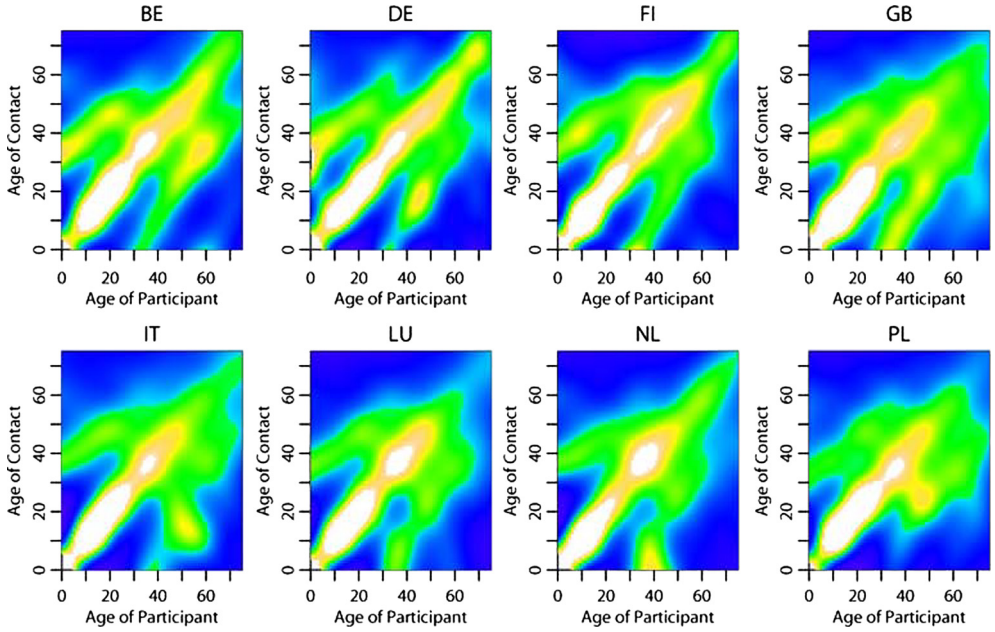

Fig. 9. Age-specific contact matrices for each of eight examined European countries. High contact rates are represented by white color, intermediate contact rates are green and low contact rates are blue. We refer to [155], from where this figure has been adapted, for further details. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

Many of these achievements are closely related with physics phenomena (see Table 3), via which we can estimate the effect of the proposed strategies and measures. On the other hand, these achievements are also inseparable from classical physics methods (Table 3). In particular, Monte Carlo simulation and mean-field prediction have attracted the greatest attention due to simplicity and high efficiency. For a comprehensive understanding, we provide a general example of mean-field theory about behavioral epidemiology in Appendix A.

4. Empirical case studies and related experiments

The first mathematical models studied the adaptive dynamics of disease–behavior responses in the homogeneously mixed population, assuming that individuals interact with each other at the same contact rate, without restrictions on selecting potential partners. Networked dynamics models shift the focus on the effects of interpersonal connectivity patterns, since everyone has their own set of contacts through which the interpersonal transmission can occur. The contacts between members of a population constitute a network, which is usually described by some well-known proxy model of synthetic networks, as shown in Section 3. This physics treatment of using evidence-based parsimonious models is valuable in illustrating fascinating ideas and revealing unexpected phenomena. However, it is not always a magic bullet for understanding, explaining, or predicting realistic cases. In recent years, the studies of social experiments become more and more popular. They contribute new insight in parameterizing and designing more appropriate dynamics models. This section briefly introduces the progress in this field.

4.1. Contact patterns of real-world networks

4.1.1. Studies using census data and questionnaires

A large-scale population-based survey of human contact patterns in eight European countries was conducted in Ref. [155], which collects the empirical data of self-reported face-to-face conversations and skin-to-skin physical contacts. The data analysis splits the population into subgroups on the basis of properties such as ages and locations, and scrutinizes the contact rate between subgroups (see Fig. 9). It reveals that across these countries, people are more probable to contact others of the same age group. Combining self-reporting contact data with serological testing data,

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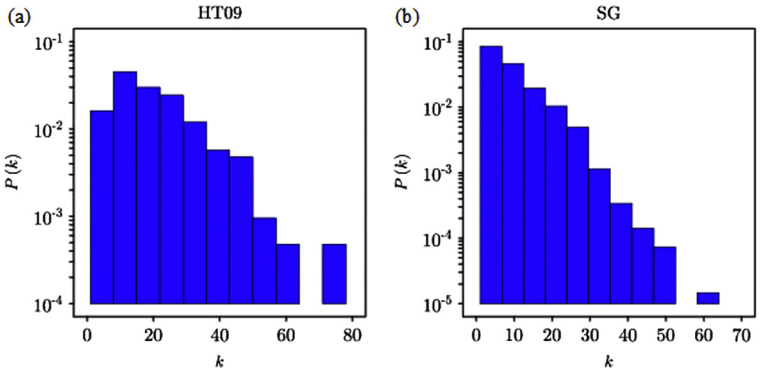

Fig. 10. Distribution of the number of distinct persons each individual encounters each day. Experiments are conducted in different conference settings (HT09 is ACM Hypertext 2009, Torino, IT; SG means Science Gallery, Dublin, IE). We refer to [164], from where this figure has been adapted, for further details.

recent case studies are able to reveal location- or age-specific patterns of exposure to pandemic pathogens [156,157], which provide rich information for complex network modeling.

4.1.2. Studies using digital equipment

SF networks have been widely used to model the connectivity heterogeneity in human contact networks. In SF networks, each hub member can have numerous connections, including all its potential contacts relevant to the transmission. However, such common consideration might not fully agree with human physiological limitations regarding the capacity in preserving a large number of interactions. Generally, the size of individual social network is restricted to around 150 people [158,159]. To better characterize the features of human contact behavior, social experiments studying the active contacts in realistic circumstances will be valuable.

Thanks to the development of information and communication technologies, the usage of digital equipments becomes increasingly popular in collecting empirical data relevant to human contacts in realistic social circumstances. It is instructive to first review a few brief examples. Refs. [160,161] referred to the Bluetooth technique embedded in mobile phones, which collects the proxy data of person-to-person interactions of MIT Media Laboratory in the Reality Mining program; with the help of wireless sensors, the social experiment was conducted to trace the close proximity contacts among the members of an American high school [162]; Refs. [163–166] considered the active Radio Frequency Identification Devices (RFID) to establish a flexible platform recording the face-to-face proximity contacts among volunteers, which had been deployed in various social contexts such as conference, museum, hospital, and primary school; and the WiFi accessing data among all students and staffs were also analyzed as the indirect proxy records of their concurrent communications in one Chinese University [167,168]. Compared with the above-mentioned data of questionnaires, the electronic data generated by digital experiments is more accurate and objective. Moreover, some new interesting findings are also listed as follows.

The data analysis reveals an unexpected feature that the distribution of the number of distinct persons each individual encounters every day only has a small squared coefficient of variance [162,164,166–169], irrespective of the specific social contexts (see Fig. 10). This homogeneity in the so-called node-degree distribution indicates the absence of connectivity hubs, which is, to some extent, subject to our physiological limitations.

The dynamics of human interactions is not evolving at an equilibrium state, but is highly fluctuating and time-varying in realistic situations. This can be characterized by measuring the statistical distribution of the duration per contact and the time intervals between successive contacts [163]. As shown in Fig. 11, these two statistics both have a broad distribution spanning several orders of magnitude. Most contact durations and inter-contact intervals are very short, but long durations and intervals also emerge, which corresponds to a burst process without characteristic time scales [170]. The coexistence of homogeneity in degree of nodes and heterogeneity in contact durations lead to unexpected phenomena. For example, the low-degree nodes which are insignificant in conventional network models can act as hubs in time-varying networks [171].

The usage of electronic devices provides an easy and nonintrusive approach for contact tracing, which can help understand health-related behaviors in realistic settings. To measure close proximity interactions between health-care

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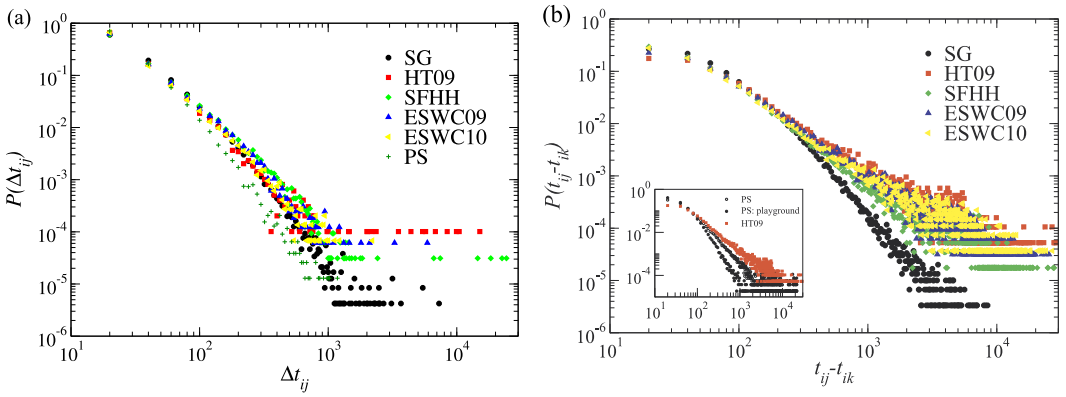

Fig. 11. (a) Distributions of contact durations measured in different social circumstances. (b) Distributions of time intervals between two successive contacts of a given individual, measured in the same social circumstances as in Fig. 11(a). Similar to Fig. 10, each symbol denotes one venue (SFHH denotes SFHH, Nice, FR; ESWC09 (ESWC10) is ESWC 2009 (2010), Crete, GR; and PS corresponds to primary school, Lyon, FR). We refer to [174], from where this figure has been adapted, for further details.

workers (HCWs) and their hand hygiene adherence, Polgreen et al. performed experiments by deploying wireless sensor networks in a medical intensive care unit of the University of Iowa hospital. They confirmed the effects of peer pressure on improving the hand hygiene participation [172], i.e., the proximity of other HCWs, can promote the hand hygiene adherence. They also analyzed the role of "superspreader", who has a high frequency in encountering others [173]. The disease severity increases with hand hygiene noncompliance of such people.

4.2. Empirical evidence related to vaccination

Except for empirical data of contact networks, social behavior experiments (or surveys) also play an important role in the vaccination campaign and disease spreading, especially combined with the decision-making process. Here we will review the recent progress within this realm.

4.2.1. Game-theoretical experiments and surveys

Role of altruistic behavior. Game theory has been extensively used in the study of behavior epidemiology, where individuals are usually assumed to decide vaccination or not based on the principle of maximizing self-interest [9,175]. However, in reality, when people make vaccination decision, do they only consider their own benefit? To test this fundamental assumption, Ref. [176] recently conducted a survey about individual vaccination decisions during the influenza season. The questionnaires, from direct campus survey and Internet-based survey, are mainly composed of two items: self-interest ones (the concern about becoming infected) and altruistic ones (the concern about infecting others), as schematically illustrated in Fig. 12. If agents are driven by self-interest, they attempt to minimize their cost associated with vaccination and infection, which gives rise to selfish equilibrium (or the so-called Nash Equilibrium). By contrary, if individual decision is guided by altruistic motivation, the vaccination probability reaches community optimum (or the so-called Utilitarian Equilibrium), at which overall cost of the community is minimal. The authors unveiled that altruism plays an important role in vaccination decision, which can be quantitatively measured by "degree of altruism". To further evaluate its impact, they incorporated the empirical data and altruistic motivation into SVIR compartmental model. Interestingly, they found that altruism can shift vaccination decisions from individual self-interest to a community optimum via greatly enhancing total vaccination coverage and reducing the total cost, morbidity and mortality of the whole community, irrespective of parameter setup.

Along this line, the role of altruistic behavior in age-structure populations was further explored [177]. According to general experience, elderly people, who are most likely to be infected in the case of influenza, should be most protected by young vaccinators, who are responsible for most disease transmission. To examine under which condition young agents vaccinate to better protect old ones, the authors organized the corresponding social behavior experiment: participants are randomly assigned to "young" and "elderly" roles (with young players contributing more to herd immunity yet elderly players facing higher costs of infection). If players were paid based on individual point totals, more elderly than young players would get vaccinated, which is consistent with the theoretical prediction of

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Survey items used in the analysis of degree of altruism.

	mean	s.d.
self-interest items		
a. Imagine that the flu shot for this year is unavailable, and you were therefore unable to get the shot. Given that you have had no shot, how worried would you be about getting the flu this Winter?[a]	2.54	1.22
b. If you were to receive the flu shot this Autumn, how worried would you be about getting the flu this Winter?[a]	1.80	0.85
c. Imagine that the flu shot for this year is unavailable, and you were therefore unable to get the shot. Given that you have not received the shot, what would you say is the likelihood that you would get the flu this Winter?[b]	52.7%	24.1%
d. Imagine that you were to receive the flu shot this Autumn. What would you say is the likelihood that you would get the flu this Winter?[b]	32.6%	17.5%
altruism items		
e. Imagine that the flu shot for this year is unavailable, and you were therefore unable to get the shot. Given that you have not received the shot, how worried would you be about infecting people at work with the flu?[a]	2.80	1.24
f. If you were to receive the flu shot this Autumn, how worried would you be about infecting people at work with the flu?[a]	1.67	0.83
g. Imagine that the flu shot for this year is unavailable, and you were therefore unable to get the shot. Given that you have not received the shot, what would you say is the likelihood that you would infect people at work with the flu?[b]	53.4%	26.3%
h. Imagine that you were to receive a flu shot this Autumn. Given that you have received the shot, what would you say is the likelihood that you would infect people at work with the flu?[b]	28.2%	18.8%

[a]Responses were on a five-point scale ranging from 1 ('not at all worried') to 5 ('very worried').
[b]Responses were on an 11-point percentage scale: 0%, 10%, 20%, …, 100%.

Fig. 12. Schematic illustration of questionnaire used in the voluntary vaccination survey. The survey items can be divided into self-interest ones (i.e., outcomes-for-self) and altruism ones (i.e., outcomes-for-others), which have corresponding scores. Based on both, it becomes possible to indirectly estimate the degree of altruism, which plays a significant role in vaccination uptake and epidemic elimination. We refer to [176], from where this figure has been adapted, for further details.

self-interested behavior (namely, Nash Equilibrium). On the contrary, players paid according to the group point totals make decisions in a manner consistent with the Utilitarian Equilibrium, which predicts community-optimal behavior: more young than elderly players get vaccinated yet less cost. In this sense, payoff structure plays a vital role in the emergence of altruistic behavior, which in turn affects the disease spreading.

From both empirically studies, we can observe that altruism significantly impacts vaccination coverage as well as consequent disease burden. It can drive system to reach community optimum, where smallest overall cost guarantees herd immunity. It is thus suggested that in realistic policies altruism should be regarded as one potential strategy to improve public health outcomes.

Existence of free-riding behavior. Accompanying altruistic behavior, another type of behavior addressed within decision-making frameworks is free-riding behavior, which means that people can benefit from the action of others while avoiding any cost [79,178,179]. In a voluntary vaccination campaign, free riders are unvaccinated individuals who avoid infection because of herd immunity, as illustrated by the gray nodes in Fig. 4. To explore the impact of free-riding behavior, John et al. even organized a questionnaire containing six different hypothetical scenarios twenty years ago [180]. Under such a survey, altruism and free-riding were simultaneously considered as the potential decision motivations for vaccination. They found that, for vaccine conferring herd immunity, the free-riding frame causes less sensitivity to increase vaccination coverage than does the altruism frame, which means that free-riding lowers preference of vaccination when the proportion of others vaccinating increases. In addition to homogeneous groups of individuals, Yoko et al. recently conducted a computerized influenza experiment, where the groups of agents may face completely different conditions, such as infection risk, vaccine cost, severity of influenza and age structure [181]. They found that high vaccination rate of previous rounds certainly decreases the likelihood of individuals' vaccination acceptance in the following round, indicating the existence of free-riding behavior. Both empirical surveys thus showed that individuals' decision-making may be driven by the free-riding motive, which depresses vaccination coverage.

Besides the above examples, there exist more factors, such as individual cognition [182] and confidence [183], affecting the decision of vaccination in reality. If possible, these factors should be taken into consideration by public policy makers in order to reach the necessary level of vaccination coverage.

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4.2.2. Digital experiments measuring vaccination opinions

The growth in online social networks such as Twitter in recent years provides a new opportunity to obtain the data on health behaviors in near real-time. Using the short text messages (tweets) data collected from Twitter between August 2009 and January 2010, during which pandemic influenza A (H1N1) spread globally, Salathé et al. analyzed the spatiotemporal individuals' sentiments towards the novel influenza A (H1N1) vaccine [97]. They found that projected vaccination rates on the basis of sentiments of Twitter users can be in good accord with those estimated by the Centers for Disease Control and Prevention of United States. They also revealed a critical problem that both negative and positive opinions can be clustered to form network communities. If this can generate clusters of unvaccinated individuals, the risk of disease outbreaks will be largely increased.

5. Conclusion and outlook

We have reviewed some of the recent, rapidly expanding research literature concerning nonlinear coupling between disease dynamics and human behavioral dynamics in spatially distributed settings, especially complex networks. Generally speaking, these models show that emergent self-protective behavior can dampen an epidemic. This is also what most mean-field models predict. However, in many cases, that is where the commonality in model predictions ends. For populations distributed on a network, the structure of the disease contact network and/or social influence network can fundamentally alter the outcomes, such that different models make very different predictions depending on the assumptions about human population and diseases being studied, including findings that disease–behavior interactions can actually worsen health outcomes by increasing long-term prevalence. Also, because network models are individual-based, they can represent processes that are difficult to represent with mean-field (homogeneous mixing) models. For example, the concept of the neighbor of an individual has a natural meaning in a network model, but the meaning is less clear in mean-field models (or partial differential equation models) where populations are described in terms of densities at a point in space. We speculate that the surge of research interest in this area has been fuelled by a combination of (1) the individual-based description that characterizes network models, (2) the explosion of available data at the individual level from digital sources, and (3) the realization from recent experiences with phenomena such as vaccine scares and quarantine failures that human behavior is becoming an increasingly important determinant of disease control efforts. We also discussed how many of the salient dynamics exhibited by disease–behavior systems are directly analogous to processes in statistical physics, such as phase transitions and self-organization.

The growth in research has created both opportunities as well as pitfalls. A first potential pitfall is that coupled disease–behavior models are significantly more complicated than simple disease dynamic or behavior dynamic models on their own. For a coupled disease–behavior model, it is necessary not only to have a set of parameters describing the human behavioral dynamics and the disease dynamics separately, it is also possible to have a set of parameters to describe the impact of human behavior on disease dynamics, and another set to describe the effect of disease dynamics on human behavior. Thus, approximately speaking, these models have four times as many parameters as a disease dynamic model on its own, or a human behavioral model on its own: they are subject to the "curse of dimensionality". A second pitfall is that relevant research from other fields may not be understood or incorporated in the best possible way. For example, the concept of 'social contagion' appears repeatedly in the literature on coupled disease–behavior models. This is a seductive concept, and it appears to be a natural concept for discussing systems where a disease contagion is also present. However, the metaphor may be too facile. For example, how can the social contagion metaphor capture the subtle but important distinction between descriptive social norms (where individuals follow a morally neutral perception of what others are doing) and injunctive social norms (where individuals follow a morally-laden perception of what others are doing) [184]? Social contagion may be a useful concept, but we should remember that it ultimately is only a metaphor. A third pitfall is lack of integration between theoretical models and empirical data: this pitfall is common to all mathematical modeling exercises. The second and third pitfalls are an unsurprising consequence of combining natural and human system dynamics in the same framework. There are other potential pitfalls as well.

These pitfalls also suggest ways in which we can move the field forward. For example, the complexity of models calls for new methods of analysis. In some cases, methods of rigorous analysis (including physics-based methods such as percolation theory and pair approximations (Appendix B))–for sufficiently simple systems that permit such analysis–may provide clearer and more rigorous insights than the output of simulation models, which are often harder to fully understand. For systems that are too complicated for pen-and-paper methods, then methods for visualization

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of large and multidimensional datasets may prove useful. The second and third pitfalls, where physicists and other modelers, behavioral scientists and epidemiologists do not properly understand one another's fields can be mitigated through more opportunities for interaction between the fields through workshops, seminars and colloquia. Interactions between scholars in these fields if often stymied by institutional barriers that emphasize a 'silo' approach to academic, thus, a change in institutional modes of operation could be instrumental in improving collaborations between modelers, behavioral scientists and epidemiologists.

Scientists have already shown that these pitfalls can be overcome in the growing research in this area, and this is evidence in much of the research we have described in this review. The field of coupled disease–behavior modeling has the elements to suggest that it will continue expanding for the foreseeable future: growing availability of data needed to test empirical models, a rich set of potential dynamics created opportunities to apply various analysis methods from physics, and relevance to pressing problems facing humanity. Physicists can play an important role in developing this field due to their long experience in applying modeling methods to physical systems.

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Appendix A. Generalized mean-field theory

Whenever the classical epidemic spreading processes (SIS, SIR) are taking place in homogeneous populations, e.g., individuals are located on the vertices of regular random graph, ER random graph, completed connected graph, the qualitative properties of the dynamics can be well catched by the mean-field analysis, assuming unbiased random matching among the individuals. For simplicity yet without loss of generality, we here derive the mean-field solution for the peer-pressure effect in vaccination dynamics on the ER random graph as an example [84]. Let x be the fraction of vaccinated individuals and $w(x)$ be the probability that a susceptible individual finally gets infected in a population with vaccine coverage x. After each SIR epidemic season, individuals get payoffs: vaccinated (i, $P_i = -c$), unvaccinated and healthy (j, $P_j = 0$), and unvaccinated and infected (ς,$P_\varsigma = -1$). Individuals are allowed to modify their vaccination strategies in terms of Eq. (7). Whenever an individual from compartment i goes to compartment j or ς, the variable x drops, which can be formulated as

$$x^- = -x(1-x)^{1+\alpha}[(1-w(x))\Pi_{i\to j} + w(x)\Pi_{i\to\varsigma}], \tag{8}$$

where above we have approximated, in the spirit of mean-field treatment, the fraction of neighbors holding opposite strategy of the vaccinated individuals as $1-x$. The quantity $\Pi_{i\to j}$ is the probability that individuals from the compartment i change to the compartment j, whose value is determined by Eq. (7). Accordingly, the gain of x can be written as

$$x^+ = x(1-x)^{1+\alpha}[(1-w(x))\Pi_{i\leftarrow j} + w(x)\Pi_{i\leftarrow\varsigma}]. \tag{9}$$

Take the two cases into consideration, the derivative of x with respect to time is written as $dx/dt = x^+ + x^-$. Solving for x, we get the equilibrium vaccination level f_V. Note that the equilibrium epidemic size is expected to be $f_I = w(x)$, satisfying the self-consistent equation $f_I = (1-x)(1-\exp[-R_0 f_I])$, where R_0 is the basic reproductive number of the epidemic.

Appendix B. Pair approximation

Pair approximation is a method by which space can be implicitly captured in an ordinary differential equation framework [185,186]. To illustrate the method, consider the variable $[S]$, defined as the number of susceptible individuals in a population distributed across a network or a lattice. For an SIR natural history, the equation of motion for S is:

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$$\frac{d[S]}{dt} = -\varphi[SI] \tag{10}$$

where $[SI]$ is the number of neighboring S–I pairs on the lattice or network, and φ is the transmission rate along each edge. In a homogeneous mixing approach, one would approximate

$$[SI] \approx [S][I] \tag{11}$$

where $[I]$ is the number of infected persons on the lattice or network, yielding the classic mass-action mixing assumption. However, in pair approximation, $[SI]$ is retained as a model variable for which we must write down the equation of motion, which after intermediate steps of derivation [185,186] yields:

$$\frac{d[SI]}{dt} = \varphi[SS]Q(I \mid SS) - \varphi[SI]Q(I \mid SI) \tag{12}$$

where $[SS]$ is the number of susceptible–susceptible pairs in the population, $Q(I \mid SS)$ is the expected number of infected neighbors of a susceptible in a susceptible–susceptible pair, and similarly $Q(I \mid SI)$ is the expected number of infected neighbors of the susceptible person in a susceptible–infected pair. The first term corresponding to creation of new SI pairs from SS pairs, through infection, while the second term corresponds to destruction of existing SI pairs through infection, thereby creating II pairs. An assumption must be made in order to close the equations at the pair level, thereby preventing writing down equations of motion for triples. For instance, on a random graph, the approximation

$$Q(I \mid SS) \approx Q(I \mid S) = \frac{[SI]}{[S]} \tag{13}$$

might be applied, where $Q(I \mid S)$ is the expected number of infected persons neighboring a susceptible person in the population. Equations and pair approximations for the other pair variables $[SS]$ and $[II]$ must also be made, after which one has a closed set of differential equations that capture spatial effects implicitly, by tracking the time evolution of pair quantities.

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