The spread of awareness and its impact on epidemic outbreaks

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When a disease breaks out in a human population, changes in behavior in response to the outbreak can alter the progression of the infectious agent. In particular, people aware of a disease in their proximity can take measures to reduce their susceptibility. Even if no centralized information is provided about the presence of a disease, such awareness can arise through first-hand observation and word of mouth. To understand the effects this can have on the spread of a disease, we formulate and analyze a mathematical model for the spread of awareness in a host population, and then link this to an epidemiological model by having more informed hosts reduce their susceptibility. We find that, in a well-mixed population, this can result in a lower size of the outbreak, but does not affect the epidemic threshold. If, however, the behavioral response is treated as a local effect arising in the proximity of an outbreak, it can completely stop a disease from spreading, although only if the infection rate is below a threshold. We show that the impact of locally spreading awareness is amplified if the social network of potential infection events and the network over which individuals communicate overlap, especially so if the networks have a high level of clustering. These findings suggest that care needs to be taken both in the interpretation of disease parameters, as well as in the prediction of the fate of future outbreaks.

mathematical model | rumor spread | behavioral response | social networks

Human reactions to the presence of disease abound, yet they have rarely been systematically investigated (1). Such reactions can range from avoiding social contact with infected individuals (*social distancing*) to wearing protective masks, vaccination, or more creative precautions. It has been shown, for instance, that local measles outbreaks are correlated with the demand for measles, mumps, and rubella vaccines (2). Similarly, the demand for condoms rises in areas where AIDS is prevalent (3), and condom use has been linked to the knowledge of someone who has died of AIDS (4).

Behavior that is responsive to the presence of a disease can potentially reduce the size of an epidemic outbreak. On closer inspection, it is not so much the presence of the disease itself that will prompt humans to change their behavior, as *awareness* of the presence of the disease. A change in behavior can be prompted without witnessing the disease first hand, but by being informed about it through others. This information in itself will spread through the population and have its own dynamic. For example, according to the Chinese Southern Weekend newspaper, the text message "There is a fatal flu in Guangzhou" was sent 126 million times in Guangzhou alone during the 2003 severe acute respiratory syndrome (SARS) outbreak (5), causing people to stay home or wear face masks when going outside. This figure stands in stark contrast to the comparatively low number of 5,327 cases recorded in the whole of China (6). It is not clear how much the individual behavioral responses contributed to containing the disease.

The spread of rumors has been described as "infection of the mind" (7) or "thought contagion" (8), and their spread is analogous to the spread of an infectious disease: information is passed on from carrier to carrier through a network of contacts. Therefore, when humans respond to the presence of a disease, we have a situation where an infectious agent and the information about the presence of this agent spread simultaneously, and will interact in their spread by a change in human behavior.

Here, we present a network model for the spread of awareness about a contagious disease. Awareness arises at the location of the disease and spreads among the population similarly to the way a disease would, an analogy that was suggested as early as 1964 (9). To capture the ephemeral nature of information, we implement an idea presented in ref. 10: as the information is passed from person to person, it loses its *quality*; in other words, first-hand information about a disease case will lead to a much more determined reaction than information that has passed through many people before arriving at a given individual.

Efforts to assess the potential for prevention of future outbreaks of contagious diseases have motivated previous studies on the effects of social distancing (11, 12) which, however, focused on behavioral changes imposed by a central organization on the population level. Attempts at extending this to incorporate individual behavioral reactions have focused on vaccination decisions and consequences thereof (13–16), dynamic rewiring of transmissive contacts (17), or incidence-dependent reductions in contact rate (18).

In this study, we will investigate how the spread of awareness, prompted by first-hand contact with the disease, affects the spread of a disease. In this context, we understand awareness as the possession of information about the outbreak one is willing to act on as opposed to just generally knowing about the disease through media coverage or government programs without taking action. To study this, we have overlaid our model of information spread with a model for the spread of a contagious disease on two, not necessarily identical networks, with more informed individuals acting to reduce their susceptibility.

In the following, we will introduce the model and, in a first approximation, cast it into a system of ordinary differential equations under the assumption of random mixing of individuals within the population. This will allow us to show how awareness can reduce the number of individuals infected during an epidemic, while the threshold for disease invasion, and thus the potential for outbreaks, remains unchanged. Subsequently, we will consider a full spatial version of the same model. We will see that if the assumption of random mixing is lifted and the local nature of the interaction taken into account, locally spreading awareness can prevent a disease from breaking out, and how social network structure and overlap between the networks have an effect on this interaction.

The Model

We associate with each individual X in the population of size N a level of awareness indicated by an index i which denotes the

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Table 1. Transitions of the model

	Transition	Rate
Infection	$S_i + I_j \rightarrow I_i + I_j$	$(1-\rho^i)\hat{\beta}$
Recovery	$I_i \rightarrow R_i$	γ
Information transmission	$X_i + X_{j>(i+1)} \rightarrow X_i + X_{i+1}$	$\hat{\alpha}$
Information fading	$X_i \rightarrow X_{i+1}$	λ
Information generation	$I_i \rightarrow I_0$	ω

number of passages the information has undergone before arriving at the given individual, i.e., X_0 will stand for an individual with first-hand information and X_i for one with information that has passed through i other individuals before arriving at the given individual. The two transitions governing the information dynamics are *information transmission* ($X_i + X_{j>(i+1)} \rightarrow X_i + X_{i+1}$) and *fading of awareness* ($X_i \rightarrow X_{i+1}$). As the quality of information decreases at each transmission event while it is also gradually lost within each individual, information eventually disappears from the population if it is not refreshed.

We link this model to an epidemiological susceptible-infected-recovered (SIR) model (19), assigning each individual a disease-related state of susceptible (denoted S_i, the subscript i again representing the level of information), infected (I_i) or recovered (R_i) with the usual transitions of *infection* and *recovery*.

To capture the impact of individual actions, we make transmission of the disease dependent on the quality of the information available to a given susceptibility. The susceptibility of individuals in states S_i increases with i as $(1-\rho^i)$, $0 < \rho < 1$. The decay constant ρ therefore governs how much the tendency to act is reduced with decreasing quality of information. The total amount of awareness in the susceptible part of the population $g(\rho, \{S_i(t)\})$ at any given time t can then be calculated as $g(\rho, \{S_i(t)\}) = \sum(S_i(t)/S(t))\rho^i$, $i = 0, 1, 2, \ldots$, the probability generating function of awareness within susceptibles.

We assume that information can be generated de novo if the disease is present, so we link generation of new information to a transition through which awareness about the disease is generated in infected individuals at rate ω. As the parameter ω thus reflects the likelihood per unit time of an infected individual to find out about their infection, it distinguishes between diseases with obvious and readily interpreted symptoms and cases where, for instance, the infection is contagious but asymptomatic, or where infection does not necessarily entail awareness about its nature (e.g., SARS, which may be mistaken for common flu). All the transitions and their respective rates are summarized in Table 1. There, and in the following, we denote with a hat per contact as opposed to population-level rates such that $\hat{\alpha}$ is the rate of information transmission per contact, whereas α is the total rate of information in the population, and analogously for β and $\hat{\beta}$. This is relevant only for the contact processes governed by these two parameters, because the processes not depending on contact happen with the same rate at the individual level as at the population level.

Mean-Field Analysis

In the mean-field approximation, individual variables are replaced by population aggregates. By assuming random mixing and therefore ignoring any spatial structure within the population, we can describe the model system fully considering only the number of individuals in each possible state.

In the mean-field version of our model of information spread, the population is compartmentalized according to level of awareness, and the information dynamics for the part of the population at awareness level i is governed by

$$\frac{dN_i}{dt} = -\alpha\frac{N_i}{N}N_{<i} + \alpha\frac{N_{i-1}}{N}(N - N_{<i}) - \lambda(N_i - N_{i-1}),$$

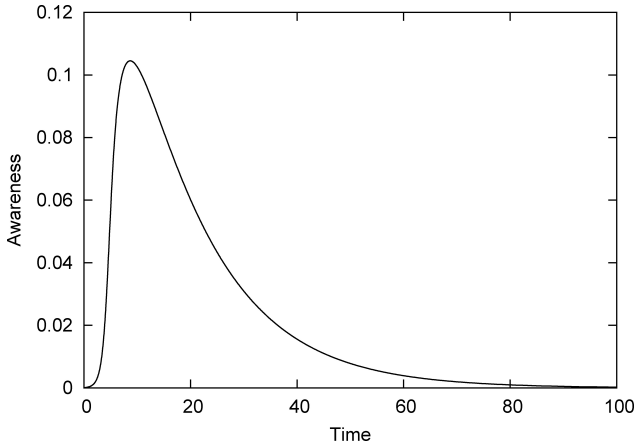

Fig. 1. Awareness $g(\rho, t)$ in the well-mixed population as a function of time for a given $\rho < 1$ if information is not replenished by the presence of the disease.

where $N_{<i} = \sum_0^{i-1} N_j$ is the number of individuals having better than ith hand information.

At any moment, awareness is then somehow distributed in the population, and this distribution changes over time according to the model dynamics. If new, and thus high-quality information is introduced once in a population in which no or only low-quality information is available, this will initially spread to increase the total amount of information in the population, given by $\sum_{i=0}^{\infty} \rho^i N_i$, only if $\alpha/\lambda > (1-\rho)/\rho$, which ensures that sufficiently many get informed to counteract the contemporaneous loss of quality (see supporting information (SI) *Appendix*). As the quality of the information diminishes while it is passed through the population, and at the same time the population gradually forgets, the only equilibrium here is one in which information is completely absent, i.e., information always disappears eventually after an initial rise (Fig. 1). Only if first-hand information is continually refreshed by the presence of a disease, the distribution of the information reaches a nonzero equilibrium.

By linking the model of information spread with the SIR model of the spreading disease, we obtain the full set of differential equations describing the interaction between the two processes (see *SI Appendix*). Now, a mutual feedback between information and disease emerges: higher prevalence of the disease entails more highly informed individuals, which in turn disseminate more information into the susceptible population, thereby impeding the further spread of the disease.

We can obtain a clearer picture of this interaction by summing the equations over the information states. In that case, the mean-field equations reduce to a form similar to the SIR equations,

$$\frac{dS}{dt} = -\beta'\frac{S}{N}I,$$
$$\frac{dI}{dt} = \beta'\frac{S}{N}I - \gamma I,$$
$$\frac{dR}{dt} = \gamma I,$$

where $\beta'(\rho, \{S_i(t)\}) = \beta \cdot [1 - g(\rho, \{S_i(t)\})]$ reflects the current level of awareness within the susceptible population and can be interpreted as the *effective* rate of infection as part of the population is shielded by its awareness and the corresponding behavioral response. Since $\beta'(\rho, \{S_i(t)\})$ depends on the distribution of the $S_i(t)$, this system is not closed, but it is still useful for understanding the behavior at the start of an outbreak. If at any time all susceptibles were maximally aware ($S = S_0$), β' would be 0 and

the disease would not spread at all, a situation that will never arise in the model because susceptibles can at best obtain S_1 status if they are informed by infecteds with first-hand information (I_0). If, however, at any instant nobody is aware ($S \to S_\infty$), β' becomes equal to β, and the model reduces to the conventional SIR model (see, e.g., refs. 19 and 20) with infection rate β and recovery rate γ. In the conventional SIR model, the epidemic threshold is at $R_0 = \beta/\gamma = 1$, meaning that an initially low number of infecteds will increase if $\beta > \gamma$ to cause an epidemic, whereas the disease will die out if $\beta < \gamma$.

Intriguingly, in this version of our model, the epidemic threshold does not change compared with the conventional SIR model if we start with a fully uninformed and susceptible population. In that case, awareness arises only through the process of *information generation*, coupled to the parameter ω and the number of infected I. This becomes relevant only once sufficiently many carry the disease, and only then is β' reduced with respect to β. During the initial stages of the outbreak, however, $\beta' \approx \beta$, and the number of infected will always increase initially if $\beta > \gamma$. Only if a certain level of awareness were already present at the time t_0 of the beginning of the outbreak, the threshold would be reduced to $R_0 = \beta'(\rho, \{S_i(t_0)\})/\gamma$.

Even with an unchanged epidemic threshold, the outbreak ceases to grow once $S(t) = N\gamma/\beta'(\rho, \{S_i(t)\})$, which can be at a significantly lower level than the usual peak at $S = N\gamma/\beta$, and, similarly, the final size of the epidemic can be much lower than without the effect of spreading awareness (see *SI Appendix*).

Individual-Based Analysis

The analysis presented in the previous section regarded the system at the population level under the assumption of random mixing, such that both the pathogen and the different levels of awareness were each distributed homogeneously within the population. In individual-based network models, however, each member of the population is embedded into a network of contacts and can infect others only over the connections of that network. In real social networks, mixing is far from random, and the number of connections each individual forms is limited and can vary significantly (21).

In a conventional SIR model, the infection events originating from a given infected individual are realized independently with identical probability T, and the average number of secondary individuals infected by a randomly chosen individual that has been infected is given by (20, 22–24)

$$\hat{R}_0 = TD_k = T\left(\overline{k} - 1 + \frac{\mathrm{Var}(k)}{\overline{k}}\right),$$

where $\overline{k}$ is the average degree, or number of contacts, and $\mathrm{Var}(k)$ is the variance thereof, such that D_k represents the effective number of contacts each individual has within the network. The basic reproductive number $\hat{R}_0$ defines a threshold similar to the way R_0 does in the mean-field case, in the sense that a large outbreak is possible only if every individual infects more than one other individual on average, that is if $\hat{R}_0 > 1$. In a conventional SIRS model, $T = \hat{\beta}/(\hat{\beta} + \gamma)$ (25), and the mean-field approximation is realized by taking the limit of $\overline{k} \to \infty$ while keeping $\overline{k}\hat{\beta} = \beta$ constant, yielding $\hat{R}_0 \to R_0 = \beta/\gamma$.

Here, we will first consider the case where disease spreads locally, but information is disseminated globally, as in the case where awareness is triggered by information broadcast through the media. If the spread of information is well-described by the mean-field approximation presented above, we can assume that information quality is independent and identically distributed within susceptible contacts of infected individuals. In that case, the probability of infection at time t over a given link chosen at random is

$$T'(t) = \sum_{i=1}^{\infty} p_i(t) T_i \quad \text{with} \quad T_i = \frac{\hat{\beta}(1-\rho^i)}{\hat{\beta}(1-\rho^i) + \gamma},$$

where $p_i(t)$ is the probability of the susceptible at risk of infection to possess information having gone through i hands at time t, and T_i is the probability of infection of that neighbor. If the distribution of awareness is already present at the time t_0 of the beginning of an outbreak the basic reproductive number is reduced to

$$\hat{R}'_0 = T'(t_0) D_k = \hat{R}_0 - (T - T'(t_0)) D_k.$$

In the limit of random mixing of disease contacts, this reduces to $\hat{R}'_0 \to R'_0 = \beta'(\rho, t_0)/\gamma$ as found in our mean-field analysis.

A completely different picture emerges if awareness, just like the disease, is not just globally present but spreads locally from individual to individual during the initial stages of the outbreak. Before we look at the full picture, let us assume for the moment that information transmission is only occurring between infected individuals informing their susceptible contacts, but that the information is not passed on any further, which could be regarded as analogous to single-step contact tracing. In that case, the impact of awareness depends on the number of edges emanating from each node that are common to both networks. If we let (kc) denote the common degree, that is the number of contacts for possible disease transmission that are also information contacts, and (kd) the degree for contacts of disease transmission only, the reduced basic reproductive number is given by

$$\hat{R}'_0 = T' D_{(kc)} + T_\infty D_{(kd)} \quad T' = p_0 T_0 + p_1 T_1,$$

where $T_\infty = \hat{\beta}/(\hat{\beta}+\gamma)$ is the transmission probability to completely uninformed individuals, as the contacts of information transmission leading to individuals not at risk of disease transmission do not contribute to the reduction of $\hat{R}_0$. In this approximation, we can derive a full expression for the reduced transmission probability T' in terms of the information-related quantities ω, $\hat{\alpha}$, and ρ (see *SI Appendix*). As a consequence of the reduction in T', the basic reproductive number $\hat{R}'_0$ is lower than $\hat{R}_0$ and can drop below the threshold of $\hat{R}'_0 = 1$ even if $\hat{R}_0 > 1$, in which case the disease is prevented from growing into an epidemic, unlike it would do without the effect of spreading awareness. Given the information-related parameters, we can derive a *critical* value $\hat{R}_0^{\mathrm{crit}}(\hat{\alpha}, \omega, \rho)$ of the basic reproductive number of the disease, in the sense that information can in principle prevent it from taking hold in the population if $\hat{R}_0 < \hat{R}_0^{\mathrm{crit}}$ (see *SI Appendix*).

While the full expression for this critical $\hat{R}_0^{\mathrm{crit}}$ is complex and does not lend itself to a simple interpretation, we can gain insight into the underlying principles by deriving upper bounds on that critical level. The protection provided by being informed is constrained by the value of the decay constant ρ. Consequently, if $\hat{R}_0$ is greater than

$$\lim_{\substack{\omega\to\infty \\ \hat{\alpha}\to\infty}} \hat{R}_0^{\mathrm{crit}} = \frac{1}{1 - \rho(1 - D_k^{-1})},$$

for a given disease, there is no chance for local information to stop the disease from growing into an epidemic, however fast it is generated and spreads within the population (Fig. 2).

Even if ρ is large, information needs to be both generated and spread at a sufficiently high rate to have an effect on the disease outbreak. Given either the rate of information generation ω, or the rate of information spread $\hat{\alpha}$, we can determine two more

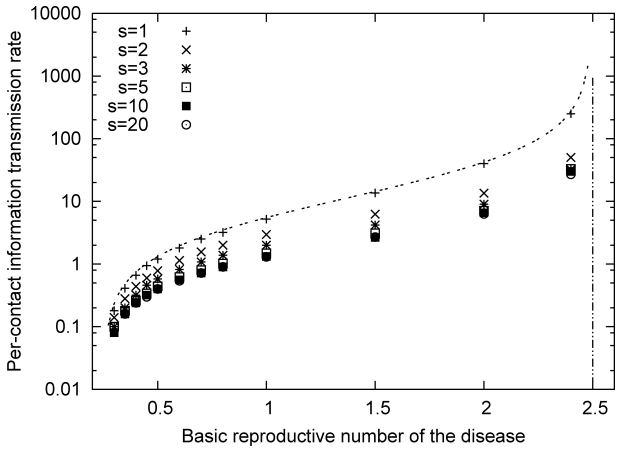

Fig. 2. Per contact information transmission rate $\hat{\alpha}$ needed to push the outbreak below the epidemic threshold for a given basic reproductive number of the disease $\hat{R}_0$. Shown is the theoretical prediction (line) and simulation results for different values of s, the number of steps information is allowed to travel from the source. The nodes were connected as a random regular graph, i.e., randomly with uniform degree $k = 6$, and the data points closely follow the predicted line. The critical $\hat{R}_0^{\text{crit}}$ for the parameters used here ($\rho = 0.9$) is indicated by a vertical line.

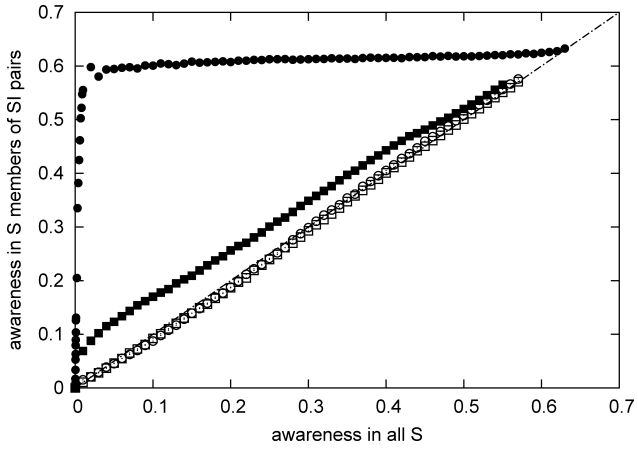

Fig. 3. Average awareness in the susceptible members of *SI* pairs in terms of the average awareness in all susceptibles, measured in stochastic simulations on the following scenarios of disease and information network structure: completely overlapping (filled squares) and completely disjointed (open squares) regular random graphs, completely overlapping lattices (filled circles) and the disease network as a lattice with the information network as a regular random graph (open circles). The line corresponds to the case $p_i^{SI} = p_i^S$.

upper bounds on the critical value of the basic reproductive number beyond which the disease cannot be stopped from reaching epidemic proportions:

$$\lim_{\substack{\omega\to\infty\\ \rho\to 1}} \hat{R}_0^{\text{crit}} = \frac{\gamma+\hat{\alpha}}{\gamma+\hat{\alpha}D_k^{-1}} \quad \text{and} \quad \lim_{\substack{\hat{\alpha}\to\infty\\ \rho\to 1}} \hat{R}_0^{\text{crit}} = \frac{\gamma+\omega}{\gamma+\omega D_k^{-1}},$$

represent situations where any information transmission or information generation, respectively, would fully protect the informed individual from getting infected. If all the parameters of information spread are given, these three limits all represent upper bounds for the critical reproductive number $\hat{R}_0^{\text{crit}}$, significantly limiting the potential impact of local information spread on the epidemic threshold. Only through a proper combination of all three involved processes (information generation, transmission, and protection) can the basic reproductive number be altered significantly.

The full effect of the interaction between the two spreading processes comes into play when we let the information propagate independently without limiting the number of steps it can spread. In that case, there is a chance for an infected individual to have its susceptible contacts informed through others, and T' can be further reduced. However, there remains a limit to the effect as the first upper bound on $\hat{R}_0^{\text{crit}}$ remains in place if $\rho < 1$ (Fig. 2). Only if information is perfect and individuals completely remove themselves from the epidemic system when they are informed can any disease be stopped.

A way to push that limit toward higher values of $\hat{R}_0$ even without the need for perfect protection would be for individuals to rewire their contacts dynamically (see, e.g., ref. 17), i.e. to cut a transmissive contact to the person they have just been informed by and establish a contact to another person instead. In that case, $\hat{R}_0'$ can indeed be shown to be reduced further, although it remains a mild effect as it affects only contacts that are common to the disease and information networks.

The reduction in the basic reproductive number and its limits are clearly a consequence of the contact-based view, and they did not appear in the mean-field analysis. In fact, the mean-field limit of the full expression for $\hat{R}_0'$ still yields $\hat{R}_0' \to R_0 = \beta/\gamma$. With respect to the well-mixed scenario, the existence of edges that are common to both networks introduced an element of structure that has no equivalent in the mean-field approximation.

Network Overlap

The single-step analysis presented in the previous section allowed for the two networks to be different, in that contacts of infectious individuals on the disease network that were not connected to the same individual on the information network were assumed to be completely unprotected. However, if awareness is allowed to spread for more than just one step, such missing links can partially be compensated for if there are other paths connecting an infected individual and its susceptible neighbor, i.e., if information links are clustered around the disease links.

Ultimately, the influence of spreading awareness on a disease outbreak depends on how much the individuals at the front of the growing epidemic are aware of its presence. Although the impact of heterogeneities in the degree distribution [including so-called scale-free network topologies (26)] can be captured in the factors $D_{(kc)}$ and $D_{(kd)}$, other properties of the two networks and their relation to each other can have a strong impact on the containment of the disease. Going back to a deterministic description of the system, we can get some insight into the relevant processes and their dependence on network structure and overlap by considering the dynamics of the population-level variables in terms of pairs (27). Denoting the number of pairs of a given type on the disease network with $[\ldots]^d$, the equation for the number of infected individuals contains a term

$$\dot{I} = \ldots + \hat{\beta}\sum_{i=0}^{\infty}(1-\rho^i)[S_iI]^d\ldots,$$

which can be rewritten as

$$\dot{I} = \ldots + \hat{\beta}\sum_{i=0}^{\infty}\left((1-\rho^i)p_i^{SI}\right)[SI]^d\ldots$$

Here, $p_i^{SI} = [S_iI]^d/[SI]^d$ represents the probability that the S member in a randomly chosen SI pair on the disease network to be at information level i, such that $\hat{\beta}' = \hat{\beta}\sum(1-\rho^i)p_i^{SI}$, can be regarded as the analogue to the effective infection rate β' of the mean-field equations. If no correlation exists between the locations of disease and information and they spread completely independently, the p_i^{SI} are given by

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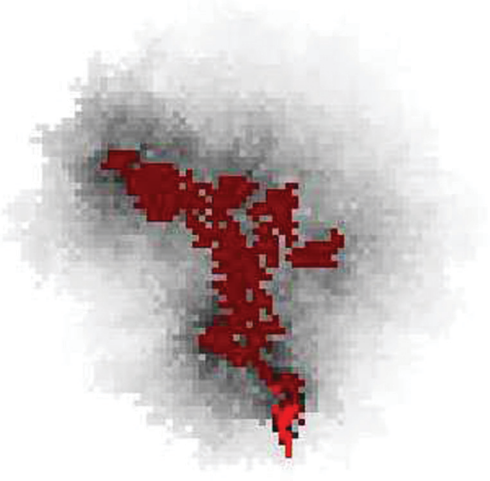

Fig. 4. Snapshot of a simulated disease outbreak on a triangular lattice. Red represents nodes that have been infected during the outbreak, with light red indicates nodes that are still spreading the disease. Darker shades of gray correspond to higher levels of awareness in susceptibles. Animated versions are available as supporting video. (See *SI Appendix*, Movies S1 and S2).

$$p_i^{SI} = p_i^S = S_i/S$$

which is nothing but the probability of a randomly chosen susceptible to be at information level i. If this equation holds, no correlation exists between information level and risk of catching the disease, and the effect of awareness is again one of a homogeneously distributed reduction in susceptibility.

Although it is not practical to derive an analytical expression for the behavior of p_i^{SI} in terms of network structure and overlap, we can measure it on simulated networks. In Fig. 3, one sees that if the information network is connected randomly and independently of the disease network, we obtain the mean-field situation where $p_i^{SI} = p_i^S$. If both networks are connected randomly but coincide, information is distributed more effectively and we observe a mild departure from equality of p_i^{SI} and p_i^S. A much more pronounced effect, however, can be observed if the two networks are triangular lattices, which contain many clusters, or triangles of connections. In that case, information is distributed much more effectively if the two networks coincide, resulting in significant correlation between risk of infection and information level, such that that much less total information is needed to protect the part of the population most at risk. Fig. 4 illustrates this effect, showing a snapshot of a simulated disease outbreak with awareness spreading on an triangular lattice completely overlapping with the disease network. Clouds of information have already formed around infected individuals, strongly limiting the further spread of the disease.

Discussion

On a social network, spreading awareness of a contagious disease in conjunction with a reduction in susceptibility does not only lower the incidence of that disease, but in some cases can even prevent that disease from growing into an epidemic. This is the case even if the awareness is not triggered by central information, but instead based on information that is passed on from person to person. However, beyond a critical infection rate, spreading awareness can slow down the spread of a disease and lower the final incidence, but it cannot completely stop it from reaching epidemic proportions and taking over large parts of the population. Only if the disease is easily recognized and information spreads rapidly, while at the same time there is a strong tendency toward protective behavior, awareness of a disease outbreak can bring the infection rate of a disease down significantly. If all of these factors work together, rapid drops in the transmissibility of a disease, as have been observed, for example, in the 2003 outbreak of SARS in Hong Kong (28), might be rooted in processes similar to the ones here presented.

Social network structure is found to play a significant role in the way spreading awareness and a contagious disease interact. The relative clustering of the information network around infectious individuals determines how effectively spreading awareness can constrain an epidemic outbreak. This effectiveness is significantly lowered when the network of disease spread differs from the communication network. This could be of relevance in the case of sexually transmitted disease, where a strong heterogeneity in the relevant network has been observed (29) and highly sexually active individuals are of crucial importance, yet do not necessarily find themselves in the same parts of the communication network as potential infectious contacts (e.g., sex workers might not communicate frequently with their customers). However, contact tracing programs work exactly to bring the two networks to match and can be seen as a special case of overlapping networks with just one step of information transmission.

Because the presence of a disease can change human behavior, care should be taken when trying to predict disease progression from behavioral observations in populations where the disease is not present (30, 31) or from observations on a different disease (32). Our model suggests how the interaction of social network structure with the properties of the disease induces a change in behavior in individuals and our results show how this could feed back to alter the disease dynamics.

Up to now, the effects of social distancing have predominantly been studied from a viewpoint of centrally controlled action. We argue that it is of equal importance to consider the self-initiated reactions of individuals in the presence of a contagious disease. The model we analyzed here differs from the previous studies of the effect of social distancing in that we treat it as a local effect within the population which depends on the awareness of the social proximity of a disease. The importance of this is particularly relevant but not limited to cases like SARS in China where initially no information was made available by the governing bodies. Therefore, we think this can provide a valuable contribution to the ongoing discussion about the impact to be expected from social distancing in disease outbreaks to come.

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1. Ferguson N (2007) Capturing human behaviour. *Nature* 446:733.
2. Philipson T (1996) Private vaccination and public health: An empirical examination for U.S. measles. *J Hum Resour* 31:611–630.
3. Ahituv A, Hotz VJ, Philipson T (1996) The responsiveness of the demand for condoms to the local prevalence of AIDS. *J Hum Resour* 31:896–897.
4. Macintyre K, Brown L, Sosler S (2001) "It's not what you know, but who you knew": Examining the relationship between behavior change and AIDS mortality in Africa. *AIDS Educ Prev* 13:160–174.
5. Tai Z, Sun T (2007) Media dependencies in a changing media environment: The case of the 2003 SARS epidemic in China. *New Media Soc* 9:987–1010.
6. World Health Organization (2003) *Consensus Document on the Epidemiology of SARS* (World Health Organization, Geneva, Switzerland).
7. Nekovee M, Moreno Y, Bianconi G, Marsili M (2007) Theory of rumour spreading in complex social networks. *Physica A* 374:457–470.
8. Lynch A (1991) Thought contagion as abstract evolution. *J Ideas* 2:3–10.
9. Goffman W, Newill VA (1964) Generalization of epidemic theory: An application to the transmission of ideas. *Nature* 204:225–228.
10. Agliari E, Burioni R, Cassi D, Neri FM (2006) Efficiency of information spreading in a population of diffusing agents. *Phys Rev E* 73:046138.
11. Hatchett RJ, Mecher CE, Lipsitch M (2007) Public health interventions and epidemic intensity during the 1918 influenza pandemic. *Proc Natl Acad Sci USA* 104: 7582–7587.
12. Bootsma MCJ, Ferguson NM (2007) The effect of public health measures on the 1918 influenza pandemic in U.S. cities. *Proc Natl Acad Sci USA* 104:7588–7593.

13. Bauch CT, Galvani AP, Earn DJD (2003) Group interest versus self-interest in smallpox vaccination policy. *Proc Natl Acad Sci USA* 100:10564–10567.
14. Perisic A, Bauch CT (2009) Social contact networks and disease eradicability under voluntary vaccination. *PLoS Comput Biol* 5:e1000280.
15. Galvani AC, Reluga TC, Chapman GB (2007) Long-standing influenza vaccination policy is in accord with individual self-interest but not with the utilitarian optimum. *Proc Natl Acad Sci USA* 104:5692–5697.
16. Salathé M, Bonhoeffer S (2008) The effect of opinion clustering on disease outbreaks. *J R Soc Interface* 5:1505–1508.
17. Gross T, D'Lima CJD, Blasius B (2006) Epidemic dynamics on an adaptive network. *Phys Rev Lett* 96:208701.
18. Del Valle S, Hethcote H, Hyman JM, Castillo-Chavez C (2005) Effects of behavioral changes in a smallpox attack model. *Math Biosci* 195:228–251.
19. Anderson RM, May RM (1991) Infectious Diseases of Humans: Dynamics and Control. (Oxford Univ Press, Oxford).
20. Diekmann O, Heesterbeek JAP (2000) *Mathematical Epidemiology of Infectious Diseases* (Wiley, Chichester, UK).
21. Eubank S, et al. (2004) Modelling disease outbreaks in realistic urban social networks. *Nature* 429:180–184.
22. Anderson RM, Medley GF, May RM, Johnson AM (1986) A preliminary study of the transmission dynamics of the human immunodeficiency virus (HIV), the causative agent of AIDS. *IMA J Math Appl Med Biol* 3:229–263.
23. Andersson H (1997) Epidemics in a population with social structures. *Math Biosci* 140:79–84.
24. Meyers LA (2007) Contact network epidemiology: Bond percolation applied to infectious disease prediction and control. *Bull Am Math Soc* 44:63–86.
25. Keeling MJ, Grenfell BT (2000) Individual-based perspectives on R_0. *J Theor Biol* 203:51–61.
26. Barabási AL, Albert R (1999) Emergence of scaling in random networks. *Science* 286:509–512.
27. Keeling MJ (1999) The effects of local spatial structure on epidemiological invasions. *Proc R Soc Lon B* 266:859–867.
28. Riley S, et al. (2003) Transmission dynamics of the etiological agent of SARS in Hong Kong: Impact of public health interventions. *Science* 300:1961–1966.
29. Liljeros F, Edling CR, Amaral LAN, Stanley HE, Åberg Y (2001) The web of human sexual contacts. *Nature* 411:907–708.
30. Mossong J, et al. (2008) Social contacts and mixing patterns relevant to the spread of infectious diseases. *PLoS Med* 5:381–391.
31. Edmunds WJ, O'Callaghan CJ, Nokes DJ (1997) Who mixes with whom? A method to determine the contact patterns of adults that may lead to the spread of airborne infections. *Proc R Soc Lond B* 264:949–957.
32. Reyes F, et al. (2008) Influenza in Canada: 2007–2008 season update. *Can Commun Dis Rep* 34:1–9.