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The impact of threshold decision mechanisms of collective behavior on disease spread

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Humans are a hyper-social species, which greatly impacts the spread of infectious diseases. How do social dynamics impact epidemiology and what are the implications for public health policy? Here, we develop a model of disease transmission that incorporates social dynamics and a behavior that reduces the spread of disease, a voluntary nonpharmaceutical intervention (NPI). We use a "tipping-point" dynamic, previously used in the sociological literature, where individuals adopt a behavior given a sufficient prevalence of the behavior in the population. The thresholds at which individuals adopt the NPI behavior are modulated by the perceived risk of infection, i.e., the disease prevalence and transmission rate, costs to adopt the NPI behavior, and the behavior of others. Social conformity creates a type of "stickiness" whereby individuals are resistant to changing their behavior due to the population's inertia. In this model, we observe a nonmonotonicity in the attack rate as a function of various biological and social parameters such as the transmission rate, efficacy of the NPI, costs of the NPI, weight of social consequences of shirking the social norm, and the degree of heterogeneity in the population. We also observe that the attack rate can be highly sensitive to these parameters due to abrupt shifts in the collective behavior of the population. These results highlight the complex interplay between the dynamics of epidemics and norm-driven collective behaviors.

epidemiology | nonpharmaceutical interventions | public health | social behavior | tipping-point

Significance

Behaviors such as mask-wearing that can hinder the spread of infectious diseases are often driven by social norms, so their adoption depends on how common they are around us. Through a mathematical model, we show that norm-driven behavioral change can drive waves of infection in the absence of external drivers. We show that if norm-driven behaviors are the only factor affecting the course of the epidemic (i.e., no vaccines or drugs), we can get counter-intuitive outcomes: for example, decreasing the effectiveness of nonpharmaceutical interventions or increasing the intrinsic R_0 of the disease can reduce the fraction of the population infected throughout the epidemic. These results illustrate the complex interplay between norm-driven social behaviors and infectious disease dynamics.

The spread of pathogens in human populations crucially depends on social, political, psychological, and economic factors (1). Informal social rules such as norms and cultural practices can impact the efficacy of treatment and public policy through their effects on human behavior. Such behavioral factors may either promote or inhibit the spread of disease. The COVID-19 pandemic has demonstrated with unusual force how critical the interactions between social and epidemiological dynamics are to controlling diseases, and how much we still have to learn about them.

There is a rich literature of disease models incorporating social behavior (2–11). Some models consider behavioral change in response to the disease as a parallel epidemic where behavior is spread through contact (12–14), whereas others model behavioral change with a game theoretic analysis. Game theory models tend to focus on if and when individually optimal behavior leads to optimality of aggregate outcomes for social welfare. A common intuition from game theory (and deeply ingrained in many cultures) is that selfish motives by the players can lead to suboptimal outcomes for a society. Indeed, this is the focus of much literature on cooperation faced with a social dilemma. In the context of infectious disease, self-interested individual decision-making, where individuals weigh their personal perceived risks and rewards to determine their optimal strategies, can be in conflict with broader social goals (15–19). Examples include whether or not to be vaccinated and whether or not to wear a mask.

In addition to economic and risk incentives, human behavior with respect to disease spread is strongly shaped by socially transmitted behaviors and social norms, the unwritten rules of social interactions (20). Social norms help determine the expectations individuals have on both what others will do and what they should do and thus guide personal decision-making. In the context of disease spread, socially transmitted practices among doctors and expectations amongst patients contributed to the emergence of antibiotic resistance due to antibiotic overuse (21–23). Likewise, West African burial traditions contributed to the spread of Ebola (24, 25), and antivaccination movements contributed to the spread of measles (26). On the other hand, social norms can be helpful in preventing the spread of disease, such as in East Asia where social acceptance of mask wearing is high (27). Mask wearing can be driven by not only belief in its effectiveness but also in the prevalence of mask wearing (28). Further, adherence to norms of mask wearing can vary

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within a population due to group identity, which can frustrate behavioral interventions (29–31). Though norms can lead to coordination of human behavior (32, 33) and the emergence of cooperative communities (34, 35) thereby overcoming social dilemmas, they also can lead to harmful outcomes and be difficult to dislodge. It is important to incorporate these social phenomena into epidemiological theory and the design of public health control efforts.

Here, we aim to understand how social dynamics drive the use of nonpharmaceutical interventions (NPI), such as social distancing and mask wearing, in blunting the spread of disease. Previous studies have looked at how a rational actor would behave in epidemics (36–38). However, social dynamics driven by imitation and social norms are equally, if not more, important than decision-making of rational actors, and there is a growing literature incorporating them (10, 11). We develop an epidemiological model that incorporates social dynamics of NPI usage where individuals weigh the risks of infection, the cost of using the NPI, and the social cost of diverging from a social norm for the NPI usage. We do this by combining material and relational utility and applying a threshold model of collective decision-making. The model induces a "tipping-point" dynamic, introduced in social behavior models (39–44), which has been found in other models of disease spread (11). We also observe nonmonotonicity in the attack rate as a function of the transmission rate but show that it can be more complex than a single shift down. Additionally, we observe nonmonotonicity when changing the efficacy of the NPI and the costs individuals face in their decision-making. From these observations and assuming no other interventions (such as vaccines or therapeutics) or external drivers, a surprising result emerges that an intermediate level for a variety of key parameters result in the lowest attack rate, but the attack rate can be highly sensitive to changes in parameters due to changes in population behavior. We discuss the implications of this work for real-world epidemics in the *Discussion*.

Methods

We consider an SEIR model with the addition of a behavioral dynamic. Our equations are

$$\begin{aligned} &\dot{S} = -\beta(p)SI, \quad \dot{E} = \beta(p)SI - \delta E, \quad \dot{I} = \delta E - \gamma I, \\ &\dot{R} = \gamma I, \qquad\qquad \dot{p} = \epsilon F(I,p) \end{aligned} \tag{1}$$

with S, E, I, and R being the frequencies of susceptible, exposed, infectious, and recovered individuals, respectively, and $p \in [0, 1]$ being the degree to which the susceptible population adopts the NPI. The quantities $1/\delta$ and $1/\gamma$ are the mean latent and recovery periods, respectively. We assume that p affects the transmission rate $\beta(p)$. Specifically, $\beta(p) = \beta_0(1 - \eta p)$ is a decreasing function where $\beta(0) = \beta_0$, the intrinsic transmission rate (i.e., the transmission rate without NPI usage), and $\eta \in [0, 1]$ is the efficacy of the NPI in reducing transmission. There is some evidence from mask mandates of risk compensation wherein individuals engage in more risky behavior due to confidence in the protection provided by masks or other NPIs (45). However, we assume that any risk compensation due to an increased adoption of the NPI or increased perception of its efficacy is insufficient to increase the transmission rate (though it may blunt its effect). For numerical simulations, we assume the parameter values displayed in Table 1. The transmission rate and average recovery and latency periods are chosen to roughly match the original variant of the SARS-CoV-2 virus, giving an average 5 d for infection maturation, 10 d for recovery from the infected state, and an R_0 comparable to estimates at the beginning of the COVID-19 pandemic. We assume that behavior changes roughly daily and hence $\epsilon = 1$. The best response scaling κ was chosen such that individuals have a smoothly changing response to infection and NPI adoption. This accounts for the fact that observations and behavioral decisions can involve some noise. The material and relational utility parameters c and θ are defined relative to the (perceived) cost of infection risk, so they have no absolute scale.

Table 1. Summary definitions of parameters with default values

Parameter	Definition	Default value
β_0	Intrinsic transmission rate	0.4/d
$1/\gamma$	Average recovery period	10 d
$1/\delta$	Average latency period	5 d
ϵ	Behavioral change rate	1/d
η	Efficacy of the NPI	0.8
κ	Payoff differential sensitivity	1000
c	Material cost	0.02
θ	Relational cost	0.01

The NPI impacts the transmission rate. However, the adoption of the NPI can change during the course of an epidemic. It is driven by individuals balancing their perceived benefits and costs of adopting the NPI. The rate at which the NPI use changes, dp/dt, is governed by $F(I, p)$, which is a function of the current frequency of infectious individuals and the level of NPI usage in the population: both of which we assume individuals know, which is a reasonable assumption in the case of conspicuous NPI's or rapid gossip. The parameter ϵ governs the magnitude of the rate of change of p. We consider a Granovetter-Schelling updating process for the adoption of the NPI (40, 42), which is a well-known dynamic in the social science literature for threshold action. Under such a dynamic, there can be a critical point at which there are enough individuals that hold an opinion or engage in a behavior such that mass adoption of it occurs. Below this threshold, there is mass rejection of the behavior/opinion. The continuous time equation for this dynamic is

$$F(I,p) = \mathrm{BR}(I,p) - p. \tag{2}$$

$\mathrm{BR}(I, p)$ is a smoothed "best response" function to the current perceived state of the epidemic:

$$\mathrm{BR}(I,p) = \frac{1}{1 + \exp(\kappa(u(I,p,0) - u(I,p,1)))}. \tag{3}$$

$u(I, p, q)$ is an individual's utility from adopting the NPI with probability/frequency/degree q when the frequency of infections is I and the overall fraction of susceptible individuals that adopt the NPI is p. The variable q is thus an individual's strategy while p is the social prevalence of the behavior. In the case of mask wearing, for example, q can be interpreted as the probability an individual wears a mask, the frequency that they wear a mask over some time period, or the degree (how well) they wear a mask. The function $u(I, p, 1)$ is the utility to an individual fully compliant with the NPI, and $u(I, p, 0)$ is the utility to an individual who is fully noncompliant. Individuals compare these two payoffs to determine their behavior. The constant $\kappa > 0$ is the sensitivity to the payoff differential of these two behaviors. For example, if κ is very large, then the best response for an individual is to adopt the NPI if $u(I, p, 0) < u(I, p, 1)$ and not to adopt it if

$u(I,p,0) > u(I,p,1)$. By Eq. **2**, p increases in the former case and decreases in the latter.

The utility function is a sum of material (economic) utility $u^m(I,q)$ and relational (social) utility $u^r(p,q)$. Material utility is a function of the risk of infection (related to the frequency of infectious individuals and the degree of social distancing) and the economic cost of social distancing. We set $u^m(I,q) = -\beta(q)I - cq$. Since $\beta(q)$ is an increasing function, $u^m(I,q)$ is a decreasing function with respect to I: the more infectious individuals, the higher the risk to being infected. It is increasing or decreasing with respect to q depending on the state of the epidemic. When infections are low, the NPI is primarily costly due to the cost c to adopt it and thus $u^m(I,q)$ is decreasing with respect to q. When infections are high, the reduced chance of infection from the NPI outweighs this cost, and thus, material utility increases with respect to q. On the other hand, relational utility is solely governed by what individuals do and what others do. Guilt and pressure to socially conform can be relational utilities and can overwhelm economic incentives. Under a promoting norm (46), relational utility favors a specific behavior—such as NPI usage—and is relative to the average behavior in the population. In such a case, one earns positive relational utility (a "warm glow") if $q > p$, and one earns negative utility (guilt, social pressure) (47) if $q < p$. Such a norm can lead to bistability, however, if the initial adoption of the norm is low (46). The norm that we focus on here is a norm of conformity in which deviations from the population average impose negative relational utility (46, 48). Under this norm, $u^r(p,q) = -\theta(p-q)^2$. The relational utility is related to the average behavior in the population and is mediated by the parameter θ, which is a weighting of the cost to deviating from the norm. Utility is thus given by

$$u(I,p,q) = u^m(I,q) + u^r(p,q) = -\beta(q)I - cq - \theta(p-q)^2. \quad [4]$$

The utility of strategy q increases with increased disease prevalence I. Its material component increases or decreases with increased q, and its relational component increases as the strategy gets closer to the population average. As the epidemic spreads, this utility has an impact on the threshold at which the NPI is adopted.

1. Results

According to our best-response function and assuming $\kappa \gg 1$, individuals should favor engaging in the NPI behavior if $u(I,p,1) > u(I,p,0)$ which occurs when

$$I > \frac{c + \theta(1-2p)}{\beta_0 \eta}. \quad [5]$$

This threshold is a function of both the material and social utilities. On the material side, the threshold decreases as the efficacy of the norm η increases and increases as its cost c increases. On the relational side, the relational cost θ has divergent effects depending on population behavior: if the majority of the population adopts the NPI (i.e., $p > 1/2$), then the threshold decreases as θ increases. However, if only a minority adopt the NPI (i.e., $p < 1/2$), then increasing the relational cost increases the threshold. Social conformity can thus retard initial adoption of the NPI relative to when it would be materially rational to adopt it. On the other hand, it also causes individuals to use the NPI longer than they should with respect to material well being. This norm "stickiness" is illustrated in Fig. 1*B*. As the number of infectious individuals increases, the best response function shifts, which can shift equilibrium behavior from a state where only a few individuals adopt the NPI to a bistable system where most people will either adopt the NPI or not, depending on what they believe others do, and then to a state where almost all engage in the behavior. Such phenomena have been previously explored in the formation and dynamics of social groups (35). Fig. 1*A* depicts such shifts.

The switching dynamic can produce behavioral waves of NPI usage, which in turn can generate epidemic waves: Fig. 2 *C*–*F* serve as an example. As the frequency of infectious individuals dips above and below the threshold of Eq. **5**, NPIs are rapidly employed and then rapidly abandoned. The number of these waves is attenuated by the parameters. As shown in Fig. 2 *C*–*F*, a higher efficacy of the NPI can produce further waves. The

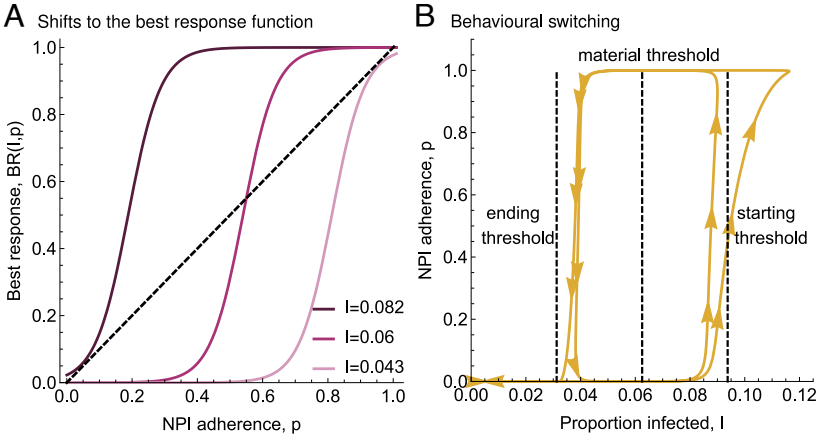

Fig. 1. Coupled dynamics of behavioral change and the epidemic caused by behavioral switches induced by norms. Panel *A* depicts how the best response curve shifts for different frequencies of infectious individuals, *I*. For $I \approx 0.082$ and $I \approx 0.043$, the best response function is tangent to the diagonal. Increasing *I* for the former results in everyone adopting the NPI, while decreasing *I* from the latter results in everyone abandoning the NPI. For *I* between these values, the system has two stable fixed points in *p* (high and low) and an intermediate, unstable one. Panel *B* depicts a sample trajectory of an epidemic depicted in the space of infectious individuals *I* and fraction of NPI adopters *p*. The arrows depict the direction of the trajectory, which is counter-clockwise. The dashed vertical lines give different thresholds for switching behavior as determined by Eq. **5** (i.e., a nonsmoothed best response). When there is no relational utility (no social norms or $\theta = 0$), the thresholds for adoption and abandonment are the same (depicted by the "material threshold" in the middle). With relational utility ($\theta > 0$), the adoption of the behavior happens at higher infection levels ("starting threshold") but abandonment happens at lower infection levels compared to the material threshold. The parameter values are taken from Table 1.

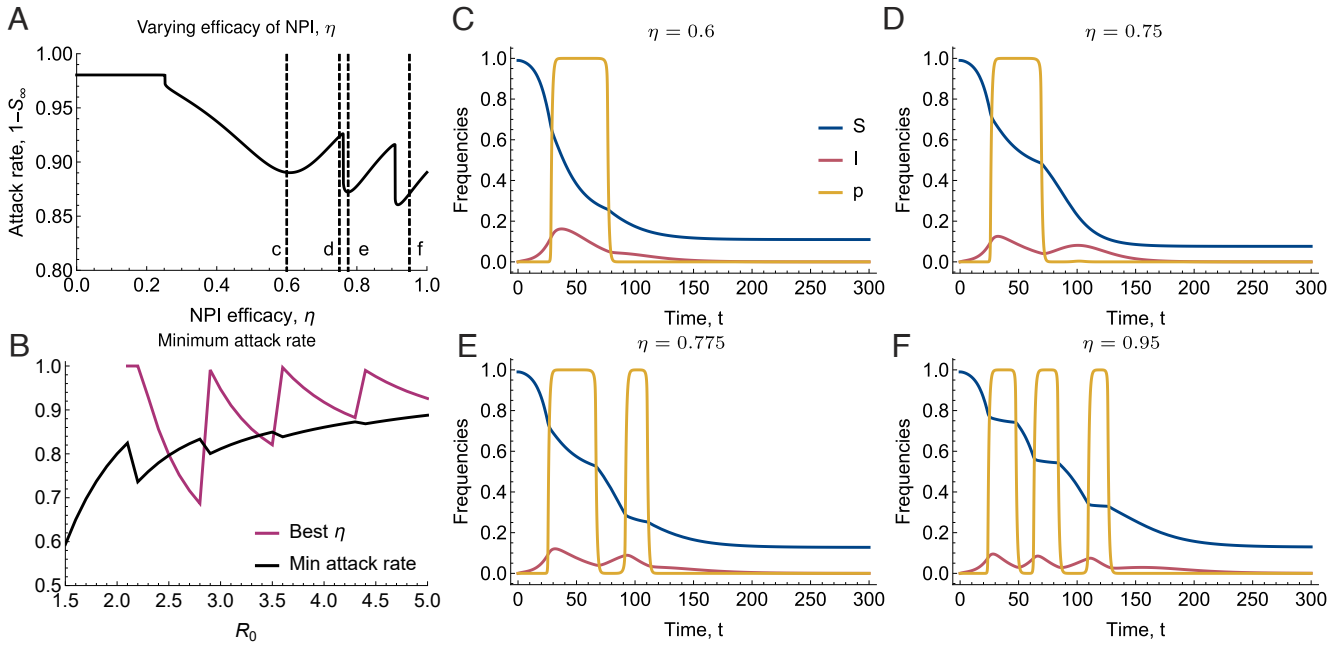

Fig. 2. The attack rate changes nonmonotonically with changing η, the efficacy of the NPI behavior. Panel *A* shows that there is a saw-tooth pattern in η and increasing number of behavior waves as η increases. Panel *B* depicts the minimum attack rate (black) and the NPI efficacy η needed to obtain it as a function of R_0 (varied by changing β_0). Panels *C*–*F* depict the time trajectories of the epidemic and behavioral change for different values of η, as marked by the vertical dashed lines on Panel *A*. Unless otherwise stated, the parameter values are taken from Table 1.

lower the threshold of infectious individuals, the more waves can be induced. Regardless of the number of waves in which behavior change is induced, there is always an "exit wave" where infections never again rise to the level required to trigger the behavioral change. The size of this exit wave is a function of the number of susceptibles left after the last wave when behavioral change abates.

The number of waves and the impact of parameters can have counter-intuitive effects on the attack rate of the epidemic. We find a nonmonotonicity in the attack rate when varying key parameters. Fig. 2*A* depicts the attack rate when we vary the efficacy of the NPI. We can explain this result by noting that there is a trade-off when NPI efficacy is increased. The higher the NPI efficacy, the earlier the population reacts to the epidemic, since the threshold to adopt the behavior is lower (as seen in Eq. **5**). Although the threshold to end the behavior is also lower, the time until this threshold is reached can be longer or shorter. Thus, the duration of NPI usage does not depend monotonically upon the NPI efficacy. Likewise, increasing NPI efficacy also decreases the transmission rate during the period of NPI use. Both of these effects lead to a larger pool of susceptibles immediately prior to the exit wave, and thereby a larger exit wave. We can develop some mathematical intuition of this by considering an SIR dynamic (where $p = 0$ and does not change) and examining the final size equation (49):

$$I_\infty = I_x + S_x - S_\infty + \frac{\gamma}{\beta_0}\int_x^\infty \frac{1}{S'} dS', \qquad [6]$$

where S_∞ and $I_\infty = 0$ are the final sizes of susceptibles and infectious, and S_x and I_x are the initial sizes at the beginning of the exit wave. Here, we're assuming that the NPI was successful in suppressing the infection to low levels, and hence, $I_x \approx 0$. Rearranging we have

$$S_\infty - S_x - \frac{1}{R_0}\ln\left(\frac{S_\infty}{S_x}\right) \approx 0. \qquad [7]$$

Differentiating with respect to S_x provides

$$S'_\infty\left(1 - \frac{1}{R_0 S_\infty}\right) = 1 - \frac{1}{R_0 S_x}. \qquad [8]$$

Assuming there is an exit wave, we have $R_0 S_x > 1$ and $R_0 S_\infty < 1$. Therefore the attack rate $1 - S_\infty$ is increasing with respect to S_x. Lowering the threshold increases the duration of NPI use and increases the number of susceptibles left at the end of the wave. Therefore, increasing efficacy may increase the size of the exit wave and thus the attack rate due to a larger overshoot. However, increasing effectiveness further can lower the threshold infection numbers such that what previously was an exit wave triggers another wave of behavioral change. This would cause a drop in S_x such that the final epidemic size drops again. This effect is what causes the saw-tooth pattern in Fig. 2*A* in the final epidemic size. Thus, increasing the NPI efficacy can sometimes come at a trade-off of leaving a higher number of susceptibles, such that for a given value of R_0, the most effective NPI might not be the one that minimizes the final attack rate (*SI Appendix*, section 1 for further mathematical details of the impact of η on the attack rate). Fig. 2*B* depicts the NPI efficacy that minimizes the attack rate for different R_0. Below a threshold $R_0 \approx 2$, behavioral waves are never initiated. For higher R_0, however, a saw-tooth pattern emerges where the NPI that minimizes attack rate can change sharply at some R_0 values. This can make policy making difficult in the fact of uncertainty over disease transmission rates. It is worth emphasizing that this discussion of the NPI efficacy for minimizing the attack rate applies in the absence of any other interventions. In reality (e.g., as it was in the COVID-19 pandemic), other goals such as pushing as many infections as possible into the future might be more important, for example, to allow development and deployment of vaccines or therapeutics. These objectives would tend to favor more effective NPIs. For example, more effective NPIs will always lower the

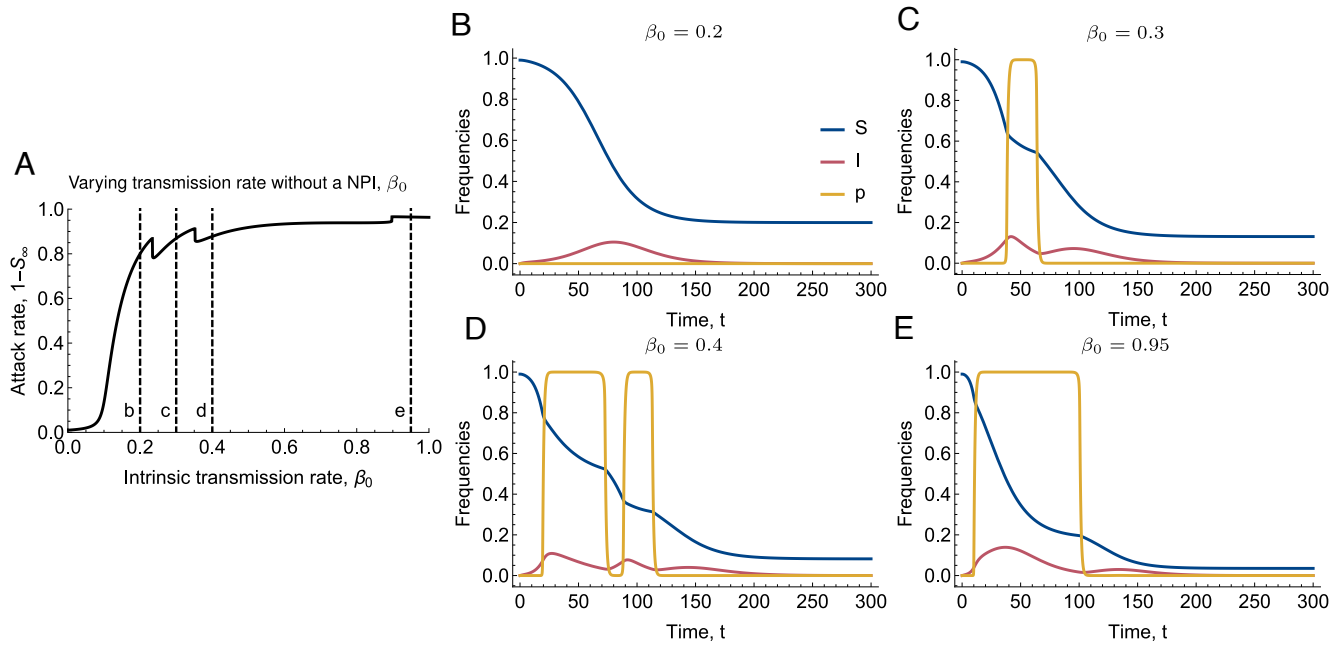

Fig. 3. The attack rate changes nonmonotonically with changing baseline transmission rate, β_0. As in Fig. 2*A*, Panel *A* depicts a saw-tooth pattern of the attack rate as β_0 varies, and Panels *B*–*E* depict time trajectories for different values of β_0, as marked by vertical dashed lines in Panel *A*. For increasing β_0, the number of behavioral waves goes from zero, to one, to two, and back to one. Unless otherwise stated, the parameter values are taken from Table 1.

highest infection peaks (*SI Appendix*, Fig. S1), and more effective NPIs also tend to keep more individuals susceptible for longer.

Varying the intrinsic transmission rate β_0—the rate when there is no NPI usage—also results in a nonmonotonic impact on the attack rate as shown in Fig. 3*A*. For sufficiently low transmission rates, no behavioral wave is induced and thus the epidemic unfolds as the standard SEIR model (Fig. 3*B*). Once transmission is sufficient to induce such a wave, we see a drop in the attack rate (Fig. 3*C*). A further increase results in another wave and thereby another drop in the attack rate (Fig. 3*D*). However,

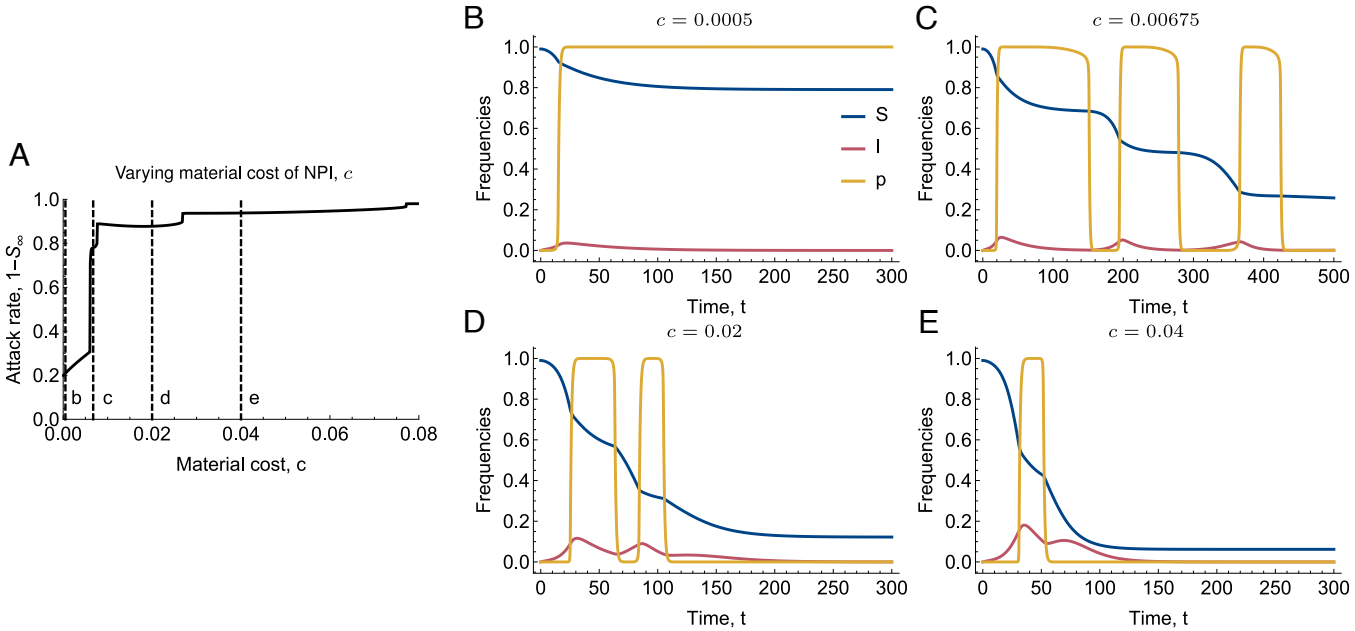

Fig. 4. Increasing material cost c reduces the number of waves, while the attack rate generally increases with c. Panel *A* again depicts attack rate as a function of the material cost, and Panels *B*–*E* sample trajectories for different material cost values. At very low material costs, a small infection level is enough to trigger the behavioral change, and once the population adopts NPIs, relational costs (i.e., conformity) ensure it is maintained even with no infections (Panel *B*). Increased material cost (specifically, $c > \theta$) means that after *I* drops to low levels, conformity cannot maintain the NPI. Abandonment of the NPI sets the stage for repeated waves of infection and behavioral change. Initially, the infection waves are small and numerous as the NPI is adopted early in the wave due to relatively low cost (starting at three waves for these parameters; Panel *C*). As the material cost increases further, the NPI is both adopted later and abandoned sooner, which makes the waves bigger and, therefore, fewer (Panels *D* and *E*). Eventually, if the cost is high enough, the NPI is never adopted, and the attack rate reaches its baseline at the far right side of Panel *A*. Unless otherwise stated, the parameter values are taken from Table 1.

further increasing the transmission rate can reduce the number of behavioral waves and thereby increase the attack rate. The higher transmission rate results in an earlier onset of the behavioral wave. However, it also increases its duration, since the number of infectious individuals remains sufficiently high—due to the higher transmission rate—to promote NPI usage. This effect causes the secondary wave of infections to not induce a secondary behavioral wave. Fig. 3*E* typifies this situation. *SI Appendix*, section 1 for further mathematical analysis.

Increasing the material cost c can reduce the number of waves, which explains the jumps we observe in the attack rate in Fig. 4*A*. Fig. 4 *B*–*E* depict time series for different values of c. Jumps in the attack rate can occur due to changes in the number of waves. However, increasing the cost will only increase the attack rate assuming that there is only one behavioral response wave (*SI Appendix*, section 1 for mathematical details). Another observation of the impact of varying material costs is that having a low c extends the pandemic, since people adopt the NPI as soon as there are flare-ups. For example, it takes longer for the pandemic to be resolved in Fig. 4*C* than it does in Fig. 4 *D* and *E*.

The parameter θ, the weighting of relational utility or relational cost, can have varying effects on the attack rate. There are essentially three qualitative regimes from low to high θ: the existence of behavioral waves, locked-in NPI usage once initiated, and no NPI usage. In the first regime, we observe a saw-tooth pattern in the attack rate for increasing relational cost. To understand this, we considered a simplified scenario in which $\epsilon \gg 1$ and thus the population rapidly adopts or abandons NPI usage. In such a scenario, if the size of the susceptible population at the end of the last NPI wave is greater than $1/R_0$, then increasing θ increases the attack rate. On the other hand, if the size of the susceptible population at the end of the last NPI wave is less than $1/R_0$, increasing θ can decrease the attack rate (though this is a necessary but not sufficient condition; *SI Appendix*, section 1). In the second regime, the attack rate precipitously drops and there is a linear increase in the attack rate for further increasing θ. In this regime, only a single sustained behavioral response is induced, as can be observed in the time series of Fig. 5*F*. The relational cost is sufficiently large that once the NPI is adopted, relational utility is sufficient to promote its use even when infections subside. However, increasing θ after this regime has begun only delays the onset of NPI adoption and therefore only increases the attack rate. In the third and final regime, θ is too high for the NPI to be adopted at all, and thus the attack rate is the same as for a nonbehavioral model. Fig. 5*B* depicts the best θ (i.e., the θ that minimizes the attack rate for a given R_0). If R_0 is too low (approximately $R_0 < 2$), behavioral waves cannot be generated. Once they can be generated, the best θ value increases as R_0 increases but saturates at $\theta \approx 0.025$. The minimum attack rate decreases even as R_0 increases until $R_0 \approx 4$, after which further increases in R_0 increase the minimum attack rate.

In summary, we observe nonmonotonicity and sharp changes in the attack rate with respect to variations in several key parameters of our model: the efficacy of the NPI, the intrinsic transmission rate, and material and relational costs. This is driven by the social norm "stickiness" whereby individuals are slow to become early adopters of a beneficial behavior, and how close the exit wave is to being large enough to spur additional waves of behavioral change. These results highlight the complexities norm-driven behaviors introduce to epidemiological dynamics.

2. Discussion

Physical and biological systems are frequently driven by human behavior, which can be driven by what other people do (50) as well as by group dynamics (51). Here, we have extended a canonical epidemiological model to incorporate human behavior driven by risk of infection, personal NPI cost, efficacy of NPI's, and social pressure of conformity. Our main finding is that the attack rate changes nonmonotonically as a function of several different parameters. First, we confirm a recent observation by Qiu et al. (11) that the attack rate displays a discontinuous

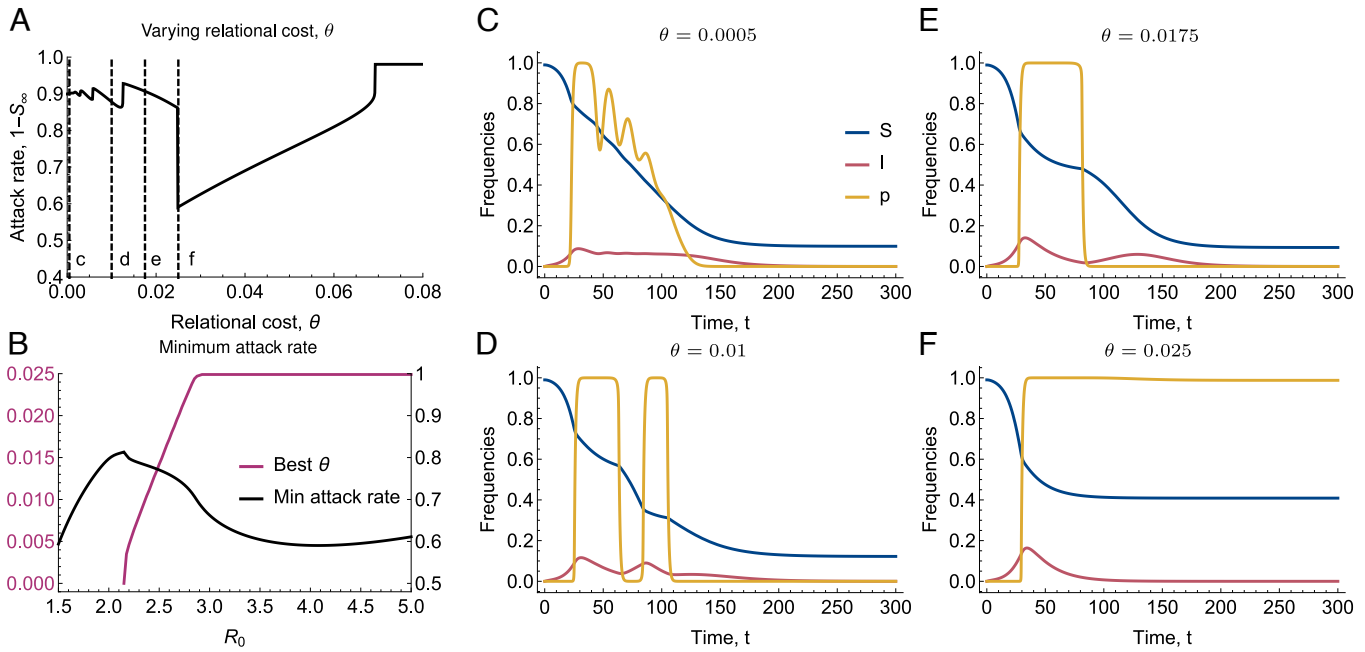

Fig. 5. The effect of relational cost θ on the attack rate. Panel *A* shows that there are three regimes of θ's impact on the attack rate: a reverse saw-tooth pattern, increasing attack rate, and a constant attack rate. For Panel *B*, we vary β_0 so as to change R_0 and find the θ (in purple, left-hand axis) that minimizes the final attack rate (in black, right-hand axis). As in the previous figures, Panels *C*–*F* depict sample time trajectories for different regimes. Unless otherwise stated, the parameter values are taken from Table 1.

nonmonotonicity when increasing the transmission rate. At an initial threshold transmission rate β_0, the attack rate suddenly drops due to the epidemic wave triggering behavioral change. After this initial threshold, the attack rate increases. In contrast to Qiu et al. (11), however, we show that there can be additional thresholds where the number of epidemic and behavioral waves change, creating multiple distinct jumps, both up and down, in the attack rate as a function of transmission rate. This difference arises because we consider a slightly different model of behavioral change: in our model, individuals combine material and relational utilities and have a single threshold for adopting mask wearing, whereas Qiu et al. (11) consider separate thresholds for fear of infection (corresponding to our material utility) and peer pressure (corresponding to our relational utility). In their model, once the entire population adopts the NPI behavior (their second tipping point), the peer pressure threshold ensures it's permanently maintained even if infection levels go to zero, while in our model individuals will gradually return to the baseline behavior, provided the relational utility cost is less than the material utility cost. These results point to the additional complexity that can arise from the coupled epidemic and social dynamics.

Previous models have also considered individuals' awareness of the epidemic (3, 10, 13, 52, 53). The main difference between these models and ours is the inclusion of the relational utility and conformism in our behavioral spread model. These models therefore don't display the stickiness of the NPI behavior we observe. Other differences also exist: Weitz et al. (10), for example, consider the death rate as driving the awareness and the resulting reduction in transmission due to behavior rather than the current number of infectious. This probably was a better description of the early stages of the COVID-19 epidemic in the US, when testing was scarce, and behavioral responses significantly lagged actual spread. They also incorporate "fatigue" where individuals get tired of the NPI, which can explain why mobility recovered in the United States in late Spring 2020 despite high death rates. Weitz et al. (10) also consider short- and long-term awareness of death rates; the latter can create some stickiness in the NPI behavior. An interesting research question going forward is how longer-term changes in risk and relational preferences interact with each other.

One of our most surprising results is that the efficacy of the NPI behavior also has a nonmonotonic effect on the attack rate. Specifically, we show that the attack rate as a function of the efficacy of the NPI can have a saw-tooth pattern. Underlying this pattern is an interesting trade-off in increasing the NPI efficacy. The higher the NPI efficacy, the earlier the population reacts to the epidemic and the later it returns to normal, thereby increasing the duration of NPI adoption. This effect on its own reduces the size of the exit wave of the pandemic. However, NPI efficacy also decreases the transmission rate during the period it is being used thus leaving a larger pool of susceptibles and thereby a larger exit wave and overshoot. This suggests that an intermediate level of NPI efficacy can be optimal. Alternatively, it suggests that policy interventions that encourage NPI adoption in the exit wave specifically to reduce overshoot can be effective in reducing the final epidemic size.

We also observe interesting effects on epidemic dynamics and attack rates when varying the relational cost θ and material cost c. The former is due to the "stickiness" of the norm. Depending on the size of the exit wave and the basic reproductive number at that point, increasing the relational cost can be either beneficial or potentially harmful in impacting the attack rate. On one extreme, low relational costs lead to plateaus in infection rates and "flatten the curve" while having a relatively small effect on the final epidemic size. On the other hand, high enough relational costs can lock in the NPI once it is adopted (consider Japanese mask-wearing as an example of such locked-in behavior (54)). However, the higher this cost, the more adoption is delayed, which increases the initial wave. At the extreme, NPIs might never get adopted due to conformism to the status quo. This suggests that intermediate levels of relational costs might be optimal. For material costs, we find that, as might be expected, cheaper NPIs are better, as they get adopted faster and last longer. Increasing material costs introduces new waves and generally increased the attack rate. Overall, these results suggest that policies that reduce the material cost of NPIs as much as possible are beneficial, while for other parameters, the effects of increasing or decreasing them depend on the context, and there might be large jumps in the final epidemic size.

It is also worth noting again here that the above results focus on the final epidemic size in the absence of any other intervention. In any real-world epidemic, especially for a novel zoonotic disease such as COVID-19, the timing of infections as well as the peaks of the epidemic waves are also important considerations, as postponing infections until vaccines or therapeutics are available, or managing healthcare demand during infection waves becomes crucial. These objectives can make more effective NPIs preferable (even if they would otherwise result in a higher attack rate in the absence of any other measures) because they tend to lower the peaks of the infection waves and keep a larger fraction of the population uninfected for longer. On the other hand, our results suggest that the relational cost θ will still have contrasting and potentially sharply changing effects on these policy goals as well. For instance, low relational costs allow fast reaction to an epidemic wave, limiting its size, but the flexibility of NPI adoption also causes infections to remain at a high plateau for a long time (Fig. 5*C*). Therefore, whatever policy goals during an epidemic are, they are likely to have to grapple with the complexities generated by norm-driven behavior.

There are several factors that can affect the conclusions from the model presented above. We assumed individuals have homogeneous contact rates and homogeneous costs. In reality, individuals belong to different communities with different costs and benefits, as well as different contact patterns. Such heterogeneity can, for example, be reflected in mask use (55). In the *SI Appendix*, section 2, we explore a model with two types of individuals with different material or relational costs. The results from this model are largely similar to our base model. On the other hand, heterogeneity between individuals can extend farther than simply the material costs and might involve political views (56), underlying differences in social norms, beliefs, or group identity (57). Smaldino and Jones (14) explore a model with two groups of individuals which differ in both disease transmission and behavior adoption from within and outside their groups. They find that depending on the degree of homophily and outgroup aversion for adopting NPIs, one of the groups might experience a larger epidemic than otherwise, and each group might experience different epidemic waves. While Smaldino and Jones (14) model behavioral change as a simple contagion process, these results likely will carry over to our norm-based behavioral model.

Another assumption we made is that individuals know the true level of both infections and NPI adoption of others at the level of the entire population. We explore small positive or negative biases in these beliefs and found no large impact on the qualitative results (*SI Appendix*, section 3); however, larger and more persistent biases driven, for example, by media or

awareness campaigns can affect the dynamics more significantly. In a previous compartmental model of norm adoption, it was found that such inaccurate beliefs about population behavior could substantially impact the emergence of a social norm (35). In the context of NPIs, biases in beliefs about population behavior can arise through visibility or salience bias (58): for instance, people going out dining are more visible than people staying in, which can induce lower perception of NPI behaviors, and delay the onset of NPI adoption. However, some NPIs such as mask wearing will be less subject to such a bias. In addition to biases about population behavior, there might also be inaccurate beliefs about the material costs of infection and/or NPIs that can quicken or delay adoption and decay of NPIs.

We also explored the effects of local information about NPI adoption and infection levels using an individual-based model of epidemic and NPI behavior spread over complex networks (59) (*SI Appendix*, section 4 for details). To do this, we modulated whether individuals base their NPI adoption on information about infection and NPI adoption levels from only their direct contacts, their second-degree contacts, or on population-level information. We find that the results from our original deterministic model are generally robust, as long as the scale of the awareness of infection levels is not too small. If, in moderately connected networks, individuals are only aware of their immediate connections' infection status, then the threshold behavior of the NPI and the distinct infection waves disappear. This is driven by the fact that individuals typically adopt the NPI when their immediate neighborhood already has high infection prevalence, at which point there is little scope for further waves in that neighborhood. This is repeated in neighborhoods across the network and thereby causes the epidemic to occur in a single wave at the population level, whose size is a monotonic function of how effective the NPI is in reducing infections. On the other hand, if either the immediate connection neighborhood is large (of the order of a 100 individuals for our base parameters) or individuals are aware of infections in a broader neighborhood (e.g., second-degree connections), then NPI adoption picks up before most individuals in a neighborhood are infected, recovering the potential for multiple infection waves that drive our results. It seems likely that for a serious respiratory disease such as COVID-19, both the infection and NPI behavior awareness are at relatively large scales. However, other infectious diseases, such as STDs with mild symptoms, might have much smaller scales of infection awareness and therefore might not exhibit the same kind of dynamics.

Finally, we have not considered vaccination in this model. Fu et al. (60) demonstrated that vaccination behavior is not random with respect to network degree and exposure to others who are vaccinated. This creates a second contagion process that can alter the dynamics of the epidemic, where the relative rates and network structure under which vaccination and the infection spread will determine the outcome. On some level, a transmission-reducing vaccine could be seen as analogous to an NPI but once adopted, its persistence does not depend on population level of vaccination. On the other hand, as the COVID-19 pandemic showed, vaccination can also be limited by supply factors and the willingness of individuals to receive it. Modeling these factors in conjunction with NPIs will add more realism to models of coupled socio-epidemiological dynamics and likely add more wrinkles to the complex relationship between attack rate and the epidemiological parameters we present here.

Data, Materials, and Software Availability. Mathematica code data have been deposited in (61).

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