

Adaptive human behavior in epidemiological models

Eli P. Fenichel^{a,1}, Carlos Castillo-Chavez^b, M. G. Ceddia^c, Gerardo Chowell^{b,d}, Paula A. Gonzalez Parra^e, Graham J. Hickling^f, Garth Holloway^c, Richard Horan^g, Benjamin Morin^b, Charles Perrings^a, Michael Springborn^h, Leticia Velazquez^e, and Cristina Villalobosⁱ

^aSchool of Life Sciences and ecoSERVICES Group, Arizona State University, Tempe, AZ 85287-4501; ^bSchool of Human Evolution and Social Change, Arizona State University, Tempe, AZ 85287; ^cDepartment of Food Economics and Marketing, School of Agriculture Policy and Development, University of Reading, RG6 6AR Reading, United Kingdom; ^dDivision of Epidemiology and Population Studies, Fogarty International Center, National Institutes of Health, Bethesda, MD 20892-2220; ^eProgram in Computational Science, University of Texas at El Paso, El Paso, TX 79968-0514; ^fCenter for Wildlife Health, Department of Forestry, Wildlife, and Fisheries, and National Institute for Mathematical and Biological Synthesis, University of Tennessee, Knoxville, TN 37996-4563; ^gDepartment of Agricultural, Food, and Resource Economics, Michigan State University, East Lansing, MI 48824; ^hDepartment of Environmental Science and Policy, University of California, Davis, CA 95616; and ⁱDepartment of Mathematics, University of Texas–Pan American, Edinburg, TX 78539

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The science and management of infectious disease are entering a new stage. Increasingly public policy to manage epidemics focuses on motivating people, through social distancing policies, to alter their behavior to reduce contacts and reduce public disease risk. Person-to-person contacts drive human disease dynamics. People value such contacts and are willing to accept some disease risk to gain contact-related benefits. The cost-benefit trade-offs that shape contact behavior, and hence the course of epidemics, are often only implicitly incorporated in epidemiological models. This approach creates difficulty in parsing out the effects of adaptive behavior. We use an epidemiological-economic model of disease dynamics to explicitly model the trade-offs that drive person-to-person contact decisions. Results indicate that including adaptive human behavior significantly changes the predicted course of epidemics and that this inclusion has implications for parameter estimation and interpretation and for the development of social distancing policies. Acknowledging adaptive behavior requires a shift in thinking about epidemiological processes and parameters.

susceptible–infected–recovered model | R_0 | reproductive number | bioeconomics

The science and management of infectious disease is entering a new stage. The increasing focus on incentive structures to motivate people to engage in social distancing—reducing interpersonal contacts and hence public disease risk (1)—changes what health authorities need from epidemiological models. Social distancing is not new—for centuries humans quarantined infected individuals and shunned the obviously ill, but new approaches are being used to deal with modern social interactions. Scientific development of social distancing public policies requires that epidemiological models explicitly address behavioral responses to disease risk and other incentives affecting contact behavior. This paper models the role of adaptive behavior in an epidemiological system. Recognizing adaptive behavior means *explicitly* incorporating behavioral responses to disease risk and other incentives into epidemiological models (2, 3). The workhorse of modern epidemiology, the compartmental epidemiological model (4, 5), does not explicitly include behavioral responses to disease risk. The transmission factors in these models combine and confound human behavior and biological processes. We develop a simple compartmental model that explicitly incorporates adaptive behavior and show that this modification alters understanding of standard epidemiological metrics. For example, the basic reproductive number, R_0 , is a function of biological processes and human behavior, but R_0 lacks a behavioral interpretation in the existing literature. Biological and behavioral feedbacks muddle R_0 's biological interpretation and confound its estimation.

Prior approaches that incorporate behavior into epidemiological models generally fall into three categories: specification of nonlinear contact rate functions, expanded epidemiological com-

partments or agent-based models, and epidemiological–economic (epi-economic) models. Classical epidemiological models assume contact rates are constant (frequency dependent) or proportional to density (density dependent), although many extensions exist (6). A common extension is to specify a contact rate that is nonlinear in the state variables—generally in the density of infected individuals (e.g., refs. 6–8). Such extensions are a reduced-form approach to modeling behavioral responses to disease risks. This approach is limited in that it does not model the underlying decision process and does not readily help decision makers design incentives for socially desirable behaviors during an epidemic. A second approach is to include behaviorally related compartments in addition to health status compartments. This approach involves developing behavioral rules for types of individuals in different compartments, such as hospitalization and fear compartments (9, 10) or spatial compartments joined as a network (11). Individuals in these compartments experience different disease incidence. Extending this approach, so that all individuals have unique behavioral rules, yields an agent-based model (e.g., ref. 12). This approach often requires the analyst to specify *ex ante* how changing incentives alters behavior and thus is restricted in its ability to aid in designing social distancing incentives.

Epi-economic models merge economics and epidemiology by explicitly analyzing individual behavioral choices in response to disease risk (13–18). People are assumed to make decisions to maximize utility, an index of well-being. People weigh the expected utility associated with decisions that include the possibility of future infection when choosing between behaviors such as vaccination choices (17) or different levels of interpersonal contact (12–15). Disease risks simultaneously affect and are affected by agents' decisions, creating a risk feedback—infection levels drive behaviors and contact rate decisions shape disease spread. The epi-economics literature is largely built on top of classical epidemiology, so that the impact of economic behaviors on epidemiological processes and metrics generally is not explored. In this paper, we explore how economic feedbacks alter the underlying epidemiology and can fundamentally shift interpretation of epidemiological processes and metrics.

The approach to modeling behavior has implications for public health policy design. Nonlinear contact rate models and models involving increasing compartmentalization generally focus on estimating the basic reproductive number of the disease,

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¹To whom correspondence should be addressed. E-mail: Eli.Fenichel@asu.edu.

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R_0 , defined as the number of secondary infections in a naive population that result from the initial introduction of a pathogen. Most of the literature recommends adopting public health policies to reduce R_0 . Roberts and Heesterbeek (ref. 19, p. 1359) state that R_0 is “the most pervasive and useful concept in mathematical epidemiology” due to its perceived role in guiding disease management. However, R_0 implicitly includes disease-free behavior and should be thought of as a reduced-form function. Estimates of R_0 confound biological aspects of the pathogen with social aspects of adaptive human responses to disease risk. Moreover, R_0 represents past events that may not be informative of future events when behavior is adaptive.

The epi-economic literature on health policy focuses on the trade-offs associated with adopting management goals not based explicitly on R_0 (20–23), as R_0 may not reliably guide postoutbreak disease management (23, 24). Most epi-economic literature assumes managers completely control behavior, e.g., require vaccination (20). But contact behavior, which is particularly important for contagious emerging diseases that have limited treatment options (e.g., novel influenza viruses and Severe Acute Respiratory Syndrome), is not easily controlled. Individuals commonly respond to disease risks by limiting contacts (25, 26), but may face insufficient private incentives to alter contacts to achieve broader public health goals. Thus, understanding contact decisions and the linkages between social distancing policies and the incentives for contacts is crucial for policy.

Methods and Models

We explore the impact of contact behavior on epidemiological processes and metrics by combining a classical compartmental epidemiological model and an economic behavioral model based on a forward-looking, representative agent. Both components are abstracted from, but have key features found in, realistic settings. Even in this simple setting behavior impacts understanding of epidemiological processes and metrics, suggesting that behavior plays an important role in complex realistic settings.

Consider a communicable disease that causes significant utility loss, but not mortality, to infected individuals within a population, N , in a given area. We divide N into three compartments: susceptible, S ; infected and infectious (which we use interchangeably), I ; and recovered with immunity, Z . The epidemiological model is

$$\dot{S} = -C(\cdot)\beta SI/N \quad [1]$$

$$\dot{I} = (C(\cdot)\beta SI/N) - \nu I \quad [2]$$

$$\dot{Z} = \nu I. \quad [3]$$

$C(\cdot)$ is the rate that susceptibles contact others, and $C(\cdot)I/N$ is the rate that susceptibles contact infectious individuals. Parameter β represents the likelihood that contact with an infectious individual yields infection, i.e., the conditional “infectiveness” of a pathogen. The rate of recovery and acquired immunity is ν , and there is no loss of immunity. The model is constructed so that N is fixed and that any outbreak is temporary. Therefore, we focus on dynamics as opposed to steady states, which exist in this model only when $I = 0$. Health status is the only source of heterogeneity within N ; individuals within a particular compartment are homogeneous with respect to behavior. Additional compartments are required to model heterogeneous behaviors (and hence heterogeneous infection risks) within a health class. The model can be extended to incorporate population turnover (*SI Text S2: Sensitivity Analysis*), changes in N , additional sources of agent heterogeneity, loss of resistance, or additional health

management choices (e.g., vaccination and treatment). Abstracting from these features facilitates clear illustration of how adaptive contact behavior affects epidemiological dynamics and metrics.

System Eqs. 1–3 imply $R_0 = \beta C(\cdot)N/\nu|_{\lim_{S \rightarrow N, \lim_{I \rightarrow 0, \lim_{Z \rightarrow 0}}}$ (27); ergo this metric depends on contact behavior, as do system dynamics. In classical epidemiological models, $C(\cdot)$ is assumed the same for all individuals regardless of health status, and mixing is assumed to be homogeneous. It is common to assume either contacts are proportional to N , i.e., $C(\cdot) = cN$, so that $\beta C(\cdot)I/N = c\beta I$, or contacts are constant, i.e., $C(\cdot) = c$, so $\beta C(\cdot)I/N = c\beta I/N$ (6). In each case, c is a fixed parameter, implying fixed (nonadaptive) contact behavior and relegating behavior to a term that is indistinguishable from β . These common assumptions hide behavior crucial to spreading infection. The critical feature that is lost through this simplification is that $C(\cdot)$ is determined by individuals’ aggregate behavioral choices.

The assumption that individuals of each health class are equally likely to come in contact (homogeneous mixing) is violated if individuals in different compartments behave differently. Heterogeneity in preferences (e.g., contacting family over strangers), exogenous conditions (e.g., age), and income lead to heterogeneous behavior. Even if the population is homogeneous in social and economic attributes, which we assume for simplicity, then individuals of different health types still face different behavioral incentives and adapt to the epidemic differently. The reason is that the expected benefits and costs of contact vary by health status. For instance, susceptible individuals have incentives to consider infected individuals’ contact behaviors, as this consideration affects the likelihood of contacting an infected individual and becoming infected.

To relax the assumption of homogeneous behavior, first index individuals by health type, denoting $Y = \{s, i, z\}$ to be the set of possible health types (corresponding to S , I , and Z). Next, define contacts between m -type and n -type individuals, with $m, n \in Y$, as

$$C^{mn}(\cdot) = C^m C^n N / (SC^s + IC^i + ZC^z). \quad [4]$$

C^{mn} is the expected number of contacts made by a type- m individual. When $m = s$ and $n = i$, $C^{mn}(\cdot) = C^{si}(\cdot)$ corresponds to $C(\cdot)$ in Eqs. 1 and 2. We emphasize that C^m is a choice made by a type- m individual. C^m may be chosen directly or by engaging in certain activities (e.g., taking public transportation). We assume individuals know their own type, but not the health status of others (additional compartments could be developed for individuals who are unaware of being infected or for cases where others have signaled their status). Accordingly, Eq. 4 implies conditional proportional mixing. Mixing is proportional, but also conditional on the behaviors and the distribution of individuals of different health types. In what follows, we simplify notation by scaling N to unity so that S , I , and Z are proportions.

If all types choose the same number of contacts and choices are constant over time ($C^h = c \forall h \in Y, \forall t$), then $C^{mn}(\cdot) = c$. Accordingly, transmission takes the classic form $\beta c SI$ and R_0 takes the classic form $R_0^c = \beta c / \nu$. More generally, individuals of different health types choose different contact levels on the basis of infection risk, so that transmission is based on Eq. 4 and R_0 is defined as $R_0^i = \beta C^i / \nu|_{\lim_{S \rightarrow N, \lim_{I \rightarrow 0, \lim_{Z \rightarrow 0}}}$. The calculation of R_0 depends on how infected individuals alter behavior in response to disease.

Consider why people make contacts. Individuals derive utility from making contacts, but incur costs from infection (i.e., reduced utility, which persists during the illness). Contact choices are made to maximize the expected net present value of utility, with the choice influencing current utility and the probability of infection and therefore expected utility in future periods. We assume individuals consider the future, have some understanding of how choices impact disease risks, and take these risks into

account when making decisions. To model this dynamic maximization problem, we define utility within a period and define the probability of transitioning across health classes. We switch to a discrete-time formulation, with time incremented in days and transition probabilities reformulated below on the basis of Eqs. 1–3.

We model a representative agent whose current-period utility depends on his current health state, $h \in Y$, and current-period contacts with others. Specifically, a type- h individual's utility at time t is $u_t(h_t, C_t^h)$. Utility is concave and single peaked in contacts, and infection reduces utility. In our simulations, we adopt $u_t^h = (b^h C_t^h - C_t^{h2})^\gamma - a^h$, where γ, b^h , and a^h are fixed parameters with $b^s = b^z \geq b^i \geq 0$, $a^s = a^z = 0$, $\gamma > 0$, and $a^i > 0$. These parameter assumptions imply that infection reduces the marginal utility of contacts and impose a lump sum cost during the infection period.

If an individual does not expect that his contact choices influence his probability of transitioning to another health state, then his optimization problem is static and the optimality condition is $\partial u_t^h / \partial C_t^h = 0$. The utility-maximizing number of contacts in our example is $C_t^{h*} = 0.5b^h$. This is the optimal choice for i - and z -type individuals, although $C_t^i < C_t^{z*}$ when $b^i < b^z$, as neither type is at risk for becoming infected. The probability of recovery, denoted $P^z = 1 - e^{-\nu}$, is independent of contacts. The probability takes this form even if infected individuals optimally choose a treatment regime, so long as the optimal treatment regime is independent of the state of the population. Without loss of generality, we scale a^i so that that utility-maximizing infected individuals receive zero utility.

Only susceptible individuals face a risk of transitioning to an alternative health state, thereby influencing their expected future utility. According to model Eqs. 1–3, the probability that an s -type individual becomes infected at time t is

$$P_t^i(C_t^s, C_t^{s*}, C_t^{i*}, C_t^{z*}, S_t, I_t, Z_t) = 1 - e^{-\beta I_t C_t^i C_t^{i*} / (S_t C_t^s + I_t C_t^{i*} + Z_t C_t^{z*})}.$$

[5]

In Eq. 5, others' behaviors are denoted by C_t^s, C_t^{i*} , and C_t^{z*} , where the superscript $*$ indicates contact choices are set at their optimized values. We distinguish between C_t^s , which is the choice of the susceptible individual under consideration, and C_t^{i*} , which is the optimal choice of other susceptible individuals that the present susceptible individual may contact. An optimizing individual takes the choices of all other susceptible individuals as given when making his contact decision. Mathematically, optimality conditions (shown below) are derived holding C_t^{i*} constant, as this value is outside the control of the individual. After deriving the optimality conditions, C_t^s is set equal to C_t^{i*} as all susceptible individuals behave identically.

The key feature of Eq. 5 is that the probability of becoming infected depends on the s -type individual's behavior, C_t^s , the distribution of types within N (i.e., the current values of S_t, I_t , and Z_t), and the behavior of others (which, in the case of other susceptible individuals, implicitly depends on the distribution of types). We assume an s -type individual has statistically complete information about the risk he currently faces, with his information set including S_t, I_t , and Z_t and knowledge of how others make decisions so that he can accurately predict others' choices. The result is that the individual's expectation of becoming infected equals Eq. 5. Assuming individuals know the true infection probability simplifies exposition, but is not required for our qualitative results.

A susceptible individual decides about contacts on the basis of his single-period utility function and expected future utility, which depend on infection expectations. The susceptible individual's decision solves the dynamic programming problem via the Bellman equation

$$V_t(s) = \max_{C_t^s \in X(s)} \{u_t(s, C_t^s) + \delta[(1 - P^i)V_{t+1}(s) + P^i V_{t+1}(i)]\}. \quad [6]$$

In Eq. 6, $V_t(s)$ is the value function associated with being susceptible at time t , X is the range of possible contacts, e.g., $X = [0, 0.5b^s]$, and δ is the discount factor. Expected future utility, the term in brackets, depends on expectations about future infection levels. $V_{t+1}(s)$ is the present value of expected future utility if the individual remains susceptible. $V_{t+1}(i)$ is the present value of expected future utility if the individual becomes infected.

An individual's decision about C_t^s depends on his information set at time t (already described above) and how information enters into expectations about future values of S, I , and Z . Expectations can be modeled in multiple ways (3, 16). A reasonable assumption is that individuals learn and adapt their "forecasts" on the basis of the current information set. The simplest forecast the individual can make, and the one we adopt, is that current values of S_t, I_t , and Z_t persist. The individual could form more complex forecasts on the basis of past and current values of S, I , and Z , but this would not change the fundamental insights that behavior matters. More complex individual forecasting would only enhance the complexity of behavioral responses and hence the behavioral effects from our model.

At time $t = 0$, the individual chooses C_0^s to solve the problem formalized by Eq. 6, given the adaptive expectations described above and given a planning horizon of length τ . In period $t = 1$, the individual updates knowledge about the state of the world and uses Eq. 6 to optimize anew over the next τ planning periods. The process continues in this manner. For instance, if $\tau = 14$, then on May 1 the individual's horizon is through May 15, but on May 2 the horizon extends to May 16, and so on.

The first-order necessary condition for the problem formalized in Eq. 6 implies

$$\partial u_t / \partial C_t^s = \delta(V_{t+1}(s) - V_{t+1}(i))(\partial P^i / \partial C_t^s). \quad [7]$$

The left term in Eq. 7 represents current period benefits of increasing contacts. The right term in Eq. 7 represents the expected marginal damage costs of increasing current contacts, in terms of discounted expected reductions in future utility due to infection. Eq. 7 implies that a susceptible individual chooses C_t^s to equate marginal benefits and expected marginal costs (i.e., disease risk). The contact level decision influences the probability of becoming infected and the magnitude of future utility.

Solving Eq. 7 requires knowledge of $V_{t+1}(s)$ and $V_{t+1}(i)$. Infected individuals face a static problem. For all $t < \tau - 1$, $V_{t+1}(i)$ is defined for an infected individual by the series

$$\begin{aligned} V_{t+1}(i) &= u(z, C^{z*}) \sum_{j=1}^{\tau} \left\{ \delta^j \left(1 - (1 - P^z)^j \right) \right\} \\ &= u(z, C^{z*}) \left[\left(\frac{1 - \delta^{\tau+1}}{1 - \delta} \right) - \left(\frac{1 - (\delta(1 - P^z))^{\tau+1}}{1 - \delta(1 - P^z)} \right) \right]. \end{aligned} \quad [8]$$

For period $t = \tau - 1$, $V_{t+1}(i) = V_{\tau}(i) = u_{\tau}(i)$ because the individual becomes infected in the terminal time period. For period $t = \tau$, $V_{t+1}(i) = V_{\tau+1}(i) = 0$ because $\tau + 1$ exceeds the individual's planning horizon.

The closed-form solution for $V_{t+1}(i)$ illustrates one role of expectations for making contact choices and hence for determining disease outcomes. $\partial V_{t+1}(i) / \partial \tau > 0$ because increases in τ extend the period over which an individual expects to be immune. Expectations also influence $V_{t+1}(s)$. Intuitively, a larger τ increases $V_{t+1}(s)$, as there is more time to gain benefits either by remaining healthy or from having more time to recover. The net effect of a larger τ on the contact decision depends on the response of $V_{t+1}(s) - V_{t+1}(i)$ to a larger τ .

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Results

We simulate a disease outbreak using the epi-economic model to show the effect of adaptive behavior (Mathematica 7.0; Wolfram). In the absence of appropriate behavioral data, we calibrate the baseline epi-economic model to be consistent with a flu-like pathogen ($b^h = 10$ when $h \neq i$, $b^i = 6.67$, $a^i = 1.826$, $\gamma^h = 0.25$, $\delta = 0.99986$ corresponding to a 5% annual discount rate, $\beta = 0.0925$, $\nu = 0.1823$, $\tau = 12$). These baseline parameters generate an *estimated* or *apparent* $R_0 = 1.8$ (28) and an expected per-individual infection period of 6 d (29). The apparent R_0 is the estimated value of R_0 (SI Text S1) from outbreak data assuming a classical epidemiological model, but where the data-generating mechanism (DGM) is actually the epi-economic model.

The dotted curve in Fig. 14 presents results of the model assuming individuals do not react to disease risks or to infection; i.e., $C_t^i = c = 0.5b^*$ $\forall h \in Y, \forall t$. For susceptible and infected individuals these behavioral assumptions likely hold only before disease introduction. We call this the ex ante classic susceptible–infected–recovered (SIR) model. This simulation results in 90% of the population becoming infected. The projected course of the disease for the baseline adaptive epi-economic model, where behavior responds to changes in disease states, is illustrated by the solid curve in Fig. 14. This simulation results in 64% of the population becoming infected. Comparing the solid and dotted curves indicates behavioral responses can substantially alter the disease course. Adaptive changes in C_t^{i*} and C_t^{s*} in the epi-economic model reduce the peak prevalence level, in addition to the reduction in cumulative cases. Reductions in C_t^{i*} also delay new infections.

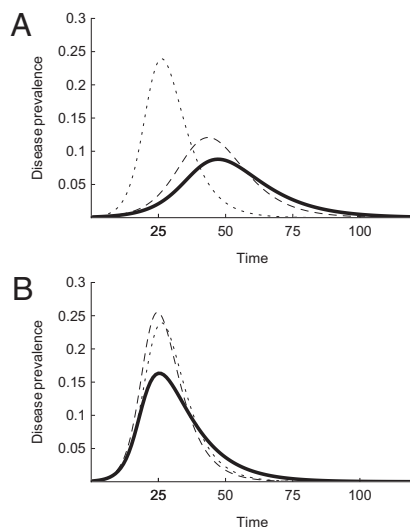


Fig. 1. (A and B) The disease courses for the adaptive epi-economic model (solid line), the classic ex ante model using the baseline parameters (dotted line), and the classic ex post model, based on the apparent R_0 (dashed line).

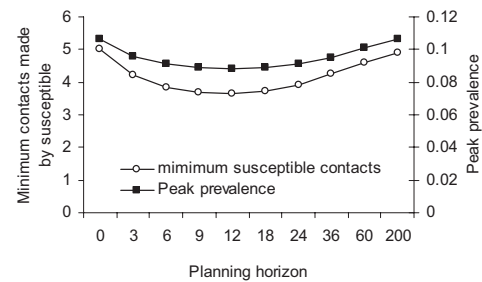


Fig. 2. The minimum number of contacts made by susceptible individuals over the course of the epidemic and the peak prevalence of the epidemic for various susceptible individual planning horizons.

Fig. 1*B* illustrates results when infected individuals find it optimal not to respond to disease; $b^i = b^z = 10$ so that $C_t^i = C_t^z$. The nonresponse by infected individuals increases risk to susceptible individuals, and susceptible individuals respond more strongly. The minimum number of contacts that susceptible individuals make is 2.49 when $b^i = b^z$ (as in Fig. 1*B*), vs. 3.65 when $b^i = 2b^z/3$ (as in Fig. 1*A*). Peak prevalence and cumulative cases are greater in Fig. 1*B*, even with a stronger susceptible response. The result is that the disease course with adaptive behavior is closer to the ex ante results, but still differs substantially.

Eq. 8 suggests that behavior is sensitive to the individual's planning horizon, τ (Fig. 2). However, the impact of longer horizons is not monotonic. A small increase in τ causes $V_{t+1}(s)$ to increase faster than $V_{t+1}(i)$, which increases the expected marginal damages of disease in Eq. 7. Therefore, susceptibles make fewer contacts, resulting in reduced peak prevalence. Greater increases in τ can have the opposite effect, reducing expected marginal damages and increasing contacts and peak prevalence. See *SI Text S2* for additional sensitivity analysis.

We simulate how behavior responds to a public policy intervention that changes the payoff structure of contacts. Consider the baseline adaptive behavior case (Fig. 14). Assume a social distancing policy reduces the payoff to making contacts (e.g., increases the cost of accessing public transportation). Fig. 3 shows two policy interventions that proportionally reduce $b^h \forall h \in Y$ for 2 wk starting on day 35 of the epidemic (e.g., following outbreak detection). The two interventions differ by the magnitude of the proportional reduction. The first intervention, a 7% reduction in b^h , maximizes societal utility (*SI Text S3*). In the second intervention b^h is reduced by 30% to further reduce infections. Policies that change the payoffs to contacts alter behavior and can cause multiple epidemic peaks, even in a fixed population. This result is consistent with conclusions in ref. 30.

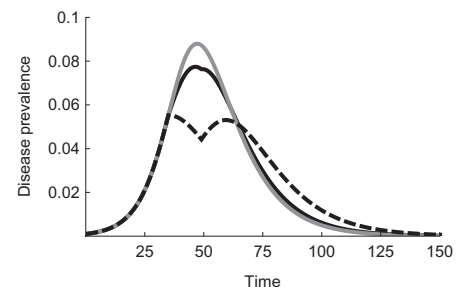


Fig. 3. Disease course with interventions. The shaded line is a no intervention case with baseline parameters. The solid line presents the optimal 2-wk decrease in b^1 (7%) starting at day 35. The dashed line presents an alternative intervention with greater case reductions, but at a net cost (b^0 reduced by 30%).

Adaptive behavior has implications for estimating disease parameters and projecting disease courses. It might be possible to estimate contact rates *ex ante*, before a disease outbreak. However, we have shown that the *ex ante* model does not yield accurate predictions. Commonly, estimation of disease parameters occurs after an outbreak. Suppose the adaptive epi-economic model is the DGM underlying the outbreak. A standard approach is to take data from the early stages of the outbreak and estimate R_0 on the basis of the assumptions of the classical SIR model ($C^h_t = c \forall h, \forall t$) (*SI Text S1*). This is the *apparent* R_0 . Next, the apparent R_0 is used to construct an *ex post* model of disease transmission on the basis of classical SIR assumptions. The dashed curve in Fig. 1 presents results of the estimated *ex post* model.

For the baseline case in Fig. 1A the apparent $R_0 = 1.8$, whereas $R_0^i = 1.67$. The apparent R_0 is closer to R_0^i than to the ex ante value $R_0^c = 2.5$. Thus, the ex post model is a better fit than the ex ante model. However, the ex post model still yields biased and inconsistent estimates of epidemiological parameters because it ignores behavioral responses and does not estimate behavioral and biological parameters jointly (31). Unless behavior is explicitly accounted for, an important omitted variables bias persists. Consistent estimates require simultaneous estimation of epidemiological and economic parameters (e.g., ref. 32 in other coupled systems; ref. 30 gives an uncoupled, but joint, estimation of disease and reduced-form behavioral parameters). The interpretation of apparent R_0 is unclear, but it is not the number of secondary infections caused by the index case.

At the instant R_0 is revealed, only the adaptive behavior of infected individuals affects disease dynamics, as is reflected by R_0^i . If individuals maintain preinfection behaviors postinfection (Fig. 1B), then $R_0^i = R_0^c = 2.5$ (shown in Fig. 1B by the overlap of the solid and dotted curves in the initial period). In this case, the apparent $R_0 = 2.6$ is closer to R_0^i than in the baseline case. Still the differences in peak prevalence between the estimated model and the DGM are greater in Fig. 1B than in Fig. 1A. This result implies that a closer estimate of R_0 does not necessarily lead to better prediction; behavior matters. The “true” value of R_0 and the implications for forecasting are increasingly muddled if multiple stages of infection or increased behavioral variability related to population heterogeneity are modeled.

Individuals acting in their own self-interest are expected to respond to disease risks by forgoing valuable contacts to protect themselves from infection. Individuals are unlikely to stop making

contacts altogether, but rather balance the expected incremental benefits and costs of additional contacts. These behavioral responses feed back into the disease transmission process and alter epidemiology dynamics and future disease risks.

Acknowledging adaptive behavior requires a shift in thinking about epidemiological processes and parameters. Adaptive behavior implies disease transmission rates change as disease risks and the private payoffs of alternative behaviors change. Epidemiological and behavioral parameters need to be estimated jointly, and traditional metrics such as R_0 may provide little policy insight once a disease has emerged. Future research is needed to determine how to recover structural parameters of a joint behavioral epi-economic model.

Jointly analyzing behavior and disease dynamics facilitates analysis of social distancing incentives. Social distancing is about changing person-to-person contact rates. Models that inform social distancing must explicitly consider the determinants of the contact rate. Most traditional epidemiological models can devise a target $C(\cdot)$, but provide insufficient guidance for reducing contacts. Extreme measures, e.g., public shutdowns that attain $C(\cdot) = 0$, can quickly end an epidemic and reduce peak prevalence, but come at a cost of forgone benefits from contacts. Reductions in the contact rate to control an epidemic can result in greater economic losses than those from the epidemic itself (33). Our policy simulation suggests that the greatest case reduction does not lead to the greatest social well-being. Our modeling framework is able to simultaneously project the epidemiological and economic effects of a social distancing policy. Public health officials must have information about both effects to evaluate whether a given strategy is worth the cost.

Understanding the incentives for contacts is critical to forming effective social distancing policies. Epidemiological models will be most useful if the contact functions they use reflect the trade-offs that people make. Mechanistic understanding of contact functions and tradeoffs can improve the cost effectiveness of disease control and help health authorities avoid unintended consequences (e.g., a school closure that results in infected children mixing in other public places where they interact with and infect more people). A mechanistic argument for the nature of the contact function allows a full analysis of the actual policy choices and provides a framework for making trade-offs so that the cure is not worse than the disease.

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