Supporting Information

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SI Text S1: Calculating Apparent R_0

R_0 is defined as the number of secondary infections caused by the index case in a completely susceptible population. Formally, this metric is defined only at the instance of disease emergence (1, 2). However, it is generally estimated on the basis of data that reflect postinfection disease dynamics (3). Accordingly, the correct approach to estimating R_0 depends on the underlying epidemiological processes (4). Multiple approaches to estimating R_0 have been developed to account for various epidemiological complexities, such as heterogeneous populations (e.g., age, spatial location) (5), thereby attempting to eliminate or reduce model specification error. However, none of this work accounts for the role of endogenous behavioral responses involving contact decisions. One of our objectives in the main text is to compare the true value of R_0 (which we derive analytically as R_0^i in the main text) with the value that is incorrectly estimated when adaptive behavior that depends on health status is ignored. Specifically, we estimate R_0 for the simulated epidemiological system Eqs. **1–3**, assuming homogeneous mixing [the classical susceptible–infected–recovered (SIR) compartmental model], but using the behavioral epidemiological-economic model as the data-generating mechanism (DGM). We refer to this as the *apparent* R_0, because it is what R_0 appears to be given the classical SIR framework.

The simplicity of the classical SIR model makes estimation of the R_0 for this model equally simple. Specifically, our estimation is based on the intrinsic growth rate of the infected population in the classical SIR model, r, measured from the initial epidemic phase (3). This general approach is commonly used in applied epidemiology (e.g., refs. 5 and 6). We take the initial epidemic phase as the period over which prevalence is increasing. Using a shorter initial period results in an apparent R_0 closer to R_0^i, but a simulated epidemic curve that, by visual inspection, is a poorer fit to the DGM.

R_0 is the dominant eigenvalue of the linearized system (2), Eqs. **1–3** in the main text, assuming homogeneous mixing,

$$R_0 = 1 + r/q, \qquad \textbf{[S1]}$$

where q is the expected time until recovery (calculated as $1/P^z$, where $P^z = 1 - e^{-v}$ from the main text and v is the recovery rate). The intrinsic growth rate, r, can be calculated assuming an initial exponential epidemic growth phase, where

$$y = Ae^{rt\varepsilon}, \qquad \textbf{[S2]}$$

and where y is the curve of incident cases at time t, A is a constant, and ε is a mean 1-lognormal error term. The value of r is estimated by linear regression following a log transformation of Eq. **S2**.

We take the epi-economic model with adaptive behavior as the DGM and record the implied daily cumulative new and unique cases during the initial epidemic growth phase. We use these data in the regression implied by Eq. **S2**. Then, the resulting estimate of r is used to calculate R_0 according to Eq. **S1**.

SI Text S2: Sensitivity Analysis

Model responses are summarized using four metrics: the minimum number of contacts made by susceptible individuals, peak prevalence, the apparent R_0, and cumulative proportion of the population infected (Table S1). The values for these four measures for the baseline epi-economic model are 3.65, 0.088, 1.8, and 0.641, respectively.

To conduct sensitivity analysis we individually increase parameters in the model by 10% (Table S1). When a 10% change in the parameter yields a 10% change in the absolute value of the response metric, we call this a unit elastic response because the change in response is proportional to the change in the parameter. When the response is greater (less) than 10% in absolute value, we say that metric is sensitive (insensitive) to the parameter. Peak prevalence was generally the most sensitive metric. The other metrics were generally unit elastic or insensitive. The apparent R_0 was very insensitive (<1% change in absolute value) to parameter changes that did not also change R_0^i.

First, consider the effects of epidemiological parameters, β and v. The parameter β affects the susceptible individual's trade-off calculus the same way as increases in I; i.e., increasing β places susceptible individuals at greater disease risk. However, susceptible individuals do not fully offset that risk with behavioral adjustments, and peak prevalence is sensitive and increasing to changes in β. Other metrics were generally unit elastic or insensitive. The recovery rate v had effects in the opposite direction of β, and with the exception of peak prevalence, metrics were generally unit elastic to changes in v. We note that given our interest in behavior, the initial calibration of v is based on the expected *infection* period (how long an individual is expected to experience symptoms). For many pathogens the *infection* period may differ from the *infectious* period (how long an individual can infect others), but modeling both effects would have required a greater number of compartments.

Now consider changes in the utility parameters b^h and γ. The greatest change in response metrics was seen with changes to b^i. Peak prevalence was almost as sensitive to changes in b^i as it was to changes in β. The other metrics were unit elastic or insensitive to changes in b^i. Only minimum contacts were unit elastic to changes in b^h, $h \neq i$; other metrics were insensitive. This result suggests that social distancing policies aimed at the susceptible population are unlikely to be very effective or efficient. The metrics considered were generally insensitive to changes in γ, which determines how sharp the peak of the utility function is.

The model was insensitive to changes in the annual discount rate. Sensitive analysis to the individual's planning horizon is presented in the main text.

We also investigated a permanent utility loss from infection. A 10% permanent utility loss from infection was implemented by reducing the utility of the recovered individual by 10%. This permanent utility loss from infection resulted in a 5% decrease in the minimum susceptible contacts. Peak prevalence was reduced by 3% and cumulative cases were reduced by 1.5%. The possibility of sustained utility losses from infection provides an incentive for contact reductions and averting behavior, but the health benefits are disproportionally small relative to the utility loss.

Our analysis focuses on a fixed population with no birth, death, or migration because we are interested in single epidemic outbreaks of infectious disease with rapid dissemination (e.g., influenza, SARS). Furthermore, in our model recovered individuals acquire permanent immunity against future infection. The entrance of new susceptible individuals to the population can have two effects in our model. First, it can perpetuate the epidemic, a common effect in the epidemiology literature. Second, changes in population, particularly the possibility of death, change the payoff structure associated with contacts. This is a unique difference in our model.

Assuming new individuals enter the population as susceptible at the same rate that individuals leave the population (i.e., N remains constant, a common assumption in the mathematical epidemiology literature), then Eqs. **1–3** in the main text can be modified to

$$\dot{S} = DI + M - C(\cdot)\beta SI/N - MS \quad \textbf{[S3]}$$

$$\dot{I} = (C(\cdot)\beta SI/N) - vI - (D + M)I \quad \textbf{[S4]}$$

$$\dot{Z} = vI - MZ, \quad \textbf{[S5]}$$

where D is the disease-induced mortality rate, and M is the rate that individuals leave the population either through non-disease–related deaths or through emigration. We have already discussed how scaling $u^z < u^s|_{I=0}$, which represents an individual's expectation of permanent disutility or death from becoming infected, reduces contacts, peak prevalence, and cases.

Assume the disease-induced mortality rate is $D = 3.3 \times 10^{-4}$, which is based on the per-capita daily mortality rate associated with influenza reported in ref. 7. If there is no effect on expectations of future utility, then this value of D is sufficiently small so that there are no qualitative effects on the model. The minimum number of contacts made by susceptible individuals, peak prevalence, and the apparent R_0 remain virtually unchanged (with entry into and exit from the population it is not straightforward to compute the proportion of the population that becomes infected because the population exposed to disease is greater than N).

Finally, consider disease-independent exit where $M = 0.01$. This quantity results in a high rate of population turnover that enables the pathogen to remain endemic (Fig. S1). Prevalence peaks at 0.083 (5.7% less than in the base epi-economic model) and eventually stabilizes at ≈0.019. During the initial outbreak the minimum number of contacts made by susceptible individuals is 3.71 (1.7% more than in the base model), but contact levels remain depressed relative to the no-disease level in the long run, stabilizing at ≈4.69 (a 6.2% reduction relative to the case when disease dies out and contact levels return to 5.0). Adaptive behavior by susceptible individuals smoothes the secondary peaks in the epidemic that commonly occur with entry and exit of individuals into and out of the population (Fig. S1). This result suggests that if adaptive behavior is important in epidemics, then incentive changes may be more important than population turnover in creating multiple epidemic waves.

For the ex ante SIR model it is possible to compute the peak prevalence, 0.239, and cumulative cases, 0.90, to provide a comparison. Incorporating adaptive behavior yields a 63% reduction in peak prevalence and a 29% reduction in cumulative cases. The sensitivity of these metrics to the inclusion of adaptive behavior is substantially larger than the sensitivity of these metrics to moderate changes in the parameter values in the epi-economic model.

SI Text S3: Using a Policy Intervention to Maximize Societal Utility

We consider a policy maker who aims to maximize social utility (i.e., aggregate utility of the population) over a 200-d time horizon:

$$\sum_{t=0}^{199} \delta^t (S_t u_t^s + I u_t^i + Z u_t^z). \quad \textbf{[S6]}$$

Our economic behavioral model takes on a reduced form where the parameters b^h and γ summarize prices, budget constraints, quality, and preference parameters. As noted in the main text a fully developed economic behavioral model would require more parameters to differentiate preference, prices, quality, and budget constraints. Implicitly, the policy intervention that reduces b^h is a change in prices that alters incentives, which alters behavior. Incentive changes that are exacted through price changes represent a wealth transfer to other members of society or the government. However, this wealth is not lost from the system, and the wealth remains so individuals potentially could be compensated for losses associated with the transfer of wealth. Following Freeman (8), we compute welfare with the original price, but at the policy-induced contact choice. This method simulates a policy that satisfies the potential compensation criteria commonly used in economics. We conduct a line search over proportional reductions in b^h for the reduction that maximizes expression S6.

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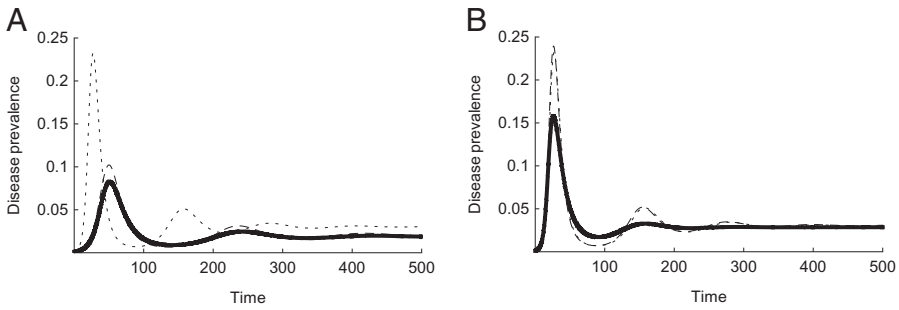

Fig. S1. The disease courses for the adaptive epi-economic model (solid line), the classic ex ante model, with no behavioral adaptation, using the parameters as given (dotted line), and the classic ex post model, with no behavioral adaptation, based on the apparent R_0 (dashed line) including population turnover.

Table S1. Baseline parameterization for simulation model and sensitivity analysis

		Percentage change in results for a 10% increase in indicated parameter value (holding other values constant)			
Parameter	Definition	Minimum contacts, %	Peak prevalence, %	Apparent R_0, %	Cumulative proportion of the population infected, %
b^s and b^z	Utility parameter (susceptible and recovered individuals)	9.4	1.2	0.1	0.4
b^i	Utility parameter (infected individuals)	−9.7	22.9	9.5	7.4
γ^h, $h \neq i$	Utility curvature parameter	3.2	1.9	0.0	0.9
δ	Discount factor*	0.0	0.0	0.0	0.0
β	Pathogen infectiousness	−10.5	24.5	9.6	7.8
v	Recovery rate	10.4	−25.2	−9.1	−10.1

*Sensitivity analysis conducted as a 10% increase to the annual 5% discount rate.