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Game-Theoretic Model of SARS Precautions

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Abstract. Severe Acute Respiratory Syndrome (SARS) is a highly contagious viral disease with high mortality rate. There is no vaccine against SARS, but the spread can be limited by masking or social distancing. In this paper we implement a game-theoretic model of voluntary precautions against SARS. We build on the compartmental ODE model of the 2003 SARS epidemic. We assume that susceptible individuals can mask and/or limit contacts with others in order to decrease their chances of contracting SARS. Since the risk of SARS infection depends on the actions of others, this creates a public goods game. We find the Nash equilibrium, the solution of the game, which is the optimal voluntary level of precautions the individuals should take. We also study the effects of such actions on the spread of SARS and show that the effect significantly depends on the individual cost of the precautions. As soon as the cost rises above a critical threshold, the individuals will have no incentive to use any kind of voluntary precaution.

1. Introduction

Severe Acute Respiratory Syndrome (SARS) is a highly contagious airborne and nosocomial disease caused by the coronavirus SARS-CoV [50]. SARS was a major source of concern when it first emerged in 2003 [15]. Overall, SARS accounted for over 770 deaths and 8000 infections globally [44]. SARS has a variety of symptoms that are similar to those of influenza, including headaches, body aches, rash, and fever [39]. SARS has a high mortality (about 15%) especially among the elderly (about 50%) [39]. There is no current treatment or vaccine for SARS, but there are human vaccines in development and a successful animal vaccine has been tested [34, 43]. Although the last reported case of SARS was in 2004, the threat of its possible return cannot be understated, especially given the recent emergence of SARS-CoV2.

Mathematical models are now a common tool used to understand and control the spread of infectious diseases [2, 57]. There are many models for SARS control, including [28, 27, 18, 42, 53, 45, 60, 31, 63, 46] and there are thousands of models for COVID-19 [38].

In their seminal paper [7], the authors incorporated game theory into epidemics modeling. With the addition of voluntary disease prevention, the game-theoretic models study scenarios in which self-interested individuals take actions based on the decisions of the rest of the population. In extensive reviews [61], [59] and [16], it is argued that incorporating human behavior provides more insight and better predictions than through the standard compartmental models.

There are many ways in which the human behavior can be incorporated into the underlying epidemiological models [61]. They include population level mean-field games where the population is assumed homogeneous and the human decisions are based on (i) the perceived risks of contracting the disease; (ii) the actual spread of the disease; (iii) the perceived risks of vaccine-related side effects. For example, [22] considered the effects of information-dependent vaccine uptake in a standard SIR compartmental model. On the other hand, [6] used imitation dynamics to create a behavior-explicit model of vaccinating behavior while [8] created the behavior-explicit game-theoretic models of vaccinating behavior. In this manuscript we follow the the theoretical framework started in [8] and expanded in [7]. However, we note that there are many other models of vaccination in structured and networked populations, such as [24].

To our knowledge, no game-theoretic model of SARS transmission exists in the literature. Thus, the main objective of this paper is to fill this gap and implement a game-theoretic model of voluntary precautions against SARS. We follow the compartmental model of [28] and assume that susceptible individuals can wear masks and/or limit contacts with others in order to decrease their chances of contracting SARS. We study the effects of such actions on the spread of SARS.

Our approach is similar to [62] who considered strategic choices during disease outbreak and applied their work to SARS. They considered an infinite population divided into "local population" and "potential travelers". Each of the two subpopulations were further divided into the usual susceptible, infected, and recovered compartments. The potential travelers played a game in which they had to decide whether to travel or not. By analyzing the resulting system of ordinary differential equations, the authors were able to assign payoffs to the players. They solved the game for Nash equilibria and they found that the optimal strategy for an individual can sometimes coincide with the optimal strategy for the group. However, when the optimal strategies differ, the difference is very large and highly sensitive to changes in parameter values. The authors concluded that centrally imposed, rather than voluntary, travel restrictions are needed to prevent SARS outbreaks. Also, [29] and [30] investigated a vaccination game in an explicitly finite population. They applied their model to HIV/AIDS. In their game, the individuals engaged in safe or risky behavior and, if infected, they could buy treatment (if the treatment was available; the game also included a drug company as additional player). The players received an idealized payoff based on their choices. The authors constructed an extensive game which they solved by backward induction for optimal strategies.

Similarly to [29], we consider a game in which individuals can engage in safe (wear a mask and/or socially distance) or risky (do not wear a mask and do not socially distance) behavior. Unlike [62], where only part of the population played the game, in our model, every person is a player. Unlike [29], we do not consider other players (such as mask producers) and we evaluate the payoffs to the players based on the underlying epidemiological model of SARS transmission, similarly to what was done in [62].

2. Model

2.1. Compartmental ODE model

We follow the model from [28] which is an extension of the classical SIR model [20, 57, 13]. The population is divided into six classes or compartments:

(1) Susceptible (S); individuals without the SARS pathogen,
(2) Exposed asymptomatic (E); asymptomatic individuals who have been exposed to the virus, but have not yet developed clinical symptoms of SARS,
(3) Infectious symptomatic (I); individuals who developed symptoms and are not in isolation,
(4) Quarantined asymptomatic (Q); those are asymptomatic exposed individuals who are quarantined because of established epidemiological contact with a source of virus,
(5) Isolated symptomatic (J); individuals who developed clinical symptoms and are isolated (for example by hospitalization or staying at home),
(6) Recovered (R); individuals who were infected in the past and possess lasting immunity against SARS.

The symbols S, E, I, Q, J, R will be used not only to denote the compartments, but also the number of individuals in the compartments. The total population size will be denoted by $N = S + E + I + Q + J + R$.

The individuals enter the population as susceptible at rate Π. This parameter represents a net inflow into the region at per unit time, including new births, immigration and emigration. Individuals in any class can die of natural and SARS unrelated causes at rate μ. As in [28], we ignore the net inflow of individuals in any other class.

Susceptible individuals become exposed when they come in contact with infectious symptomatic, exposed asymptomatic, quarantined asymptomatic, or isolated symptomatic individuals. Without any precautions, the transmission rates are β, $\varepsilon_E\beta$, $\varepsilon_Q\beta$, $\varepsilon_J\beta$, respectively; here β is the transmission coefficient which includes both the contact rate and the transmission probability and ε_X is a modification factor for the class $X \in \{E, J, Q\}$. However, the individuals may take some precautions such as wearing masks, social distancing, or avoiding gatherings. We denote such precaution efforts by e_{pop} and, similarly as in [21, 17, 14, 54] we assume that the transmission rate is reduced by the factor $(1 - e_{pop})$. The total force of infection is thus given by

$$\lambda = (1 - e_{pop})\beta \left(\frac{I}{N} + \varepsilon_E \frac{E}{N} + \varepsilon_Q \frac{Q}{N} + \varepsilon_J \frac{J}{N} \right). \tag{1}$$

The exposed asymptomatic individuals develop symptoms at rate κ_1. They can also enter quarantine at rate γ_1. As discussed in [28], in reality some susceptible individuals will likely enter quarantine, but for the purpose of our model we neglect this possibility and assume that all quarantined individuals were truly exposed and,

in time κ_2^{-1}, develop symptoms and become isolated symptomatic. This assumption means that our model slightly overestimates the number of susceptible individuals.

The infectious symptomatic individuals may recover at rate σ_1, enter isolation at rate γ_2, or die of SARS related causes at rate d_1. The isolated individuals recover at rate σ_2 but can also die of SARS related causes at rate d_2. We assume $d_1 > d_2$ and $\sigma_2 > \sigma_1$ because isolated individuals are more likely to receive treatment [12, 42].

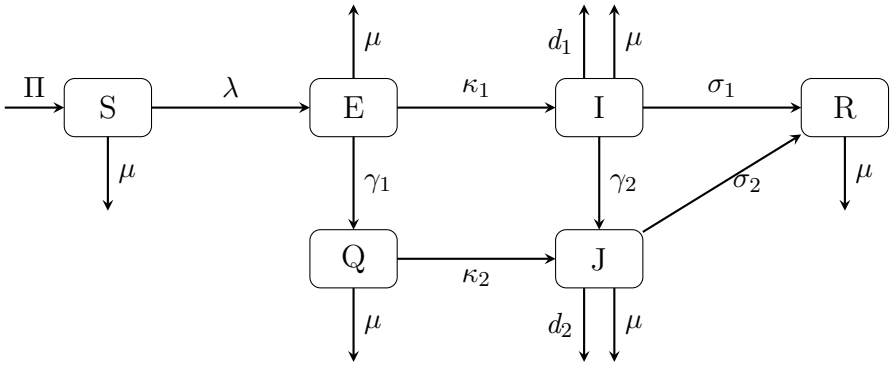


FIGURE 1. Scheme of the compartmental ODE model for SARS transmission. The compartments represent Susceptible (S), Exposed and asymptomatic (E), Infectious and symptomatic (I), Quarantined asymptomatic (Q), Isolated symptomatic (J), and Recovered (R). Solid arrows represent the transitions between compartments. The letters next to the arrows specify the per capita rates of the transitions. The force of infection λ is given by (1). The parameters are explained in Table 1.

The dynamics are summarized in Figure 1. The notation is summarized in Table 1. The parameter values are adopted from [28], specifically the Hong Kong outbreak [42].

TABLE 1. Model parameters. The rates are per capita per day. The values were all adopted from [28]. Ranges were estimated from the values.

Symbol	Meaning	Value	Range
Π	Net inflow of susceptible individuals	221	$[100, 300]$
β	Transmission rate	0.15	$[0.1, 0.25]$
μ	Natural death rate	$\frac{1}{80*365}$	$\left[\frac{1}{90*365}, \frac{1}{70*365}\right]$
κ_1	Rate at which not quarantined exposed individuals develop symptoms	0.1	$[0.05, 0.15]$
κ_2	Rate at which quarantined exposed individuals develop symptoms	0.125	$[0.1, 0.15]$
γ_1	Rate of quarantining of people who have been in contact with an infected individual	0	$[0, 0.15]$
γ_2	Rate at which symptomatic individuals seek medical attention and are put into isolation	0	$[0, 0.75]$
σ_1	Recovery rate of infectious individuals	0.0337	$\left[\frac{1}{40}, \frac{1}{20}\right]$
σ_2	Recovery rate of isolated individuals	0.0386	$\left[\frac{1}{40}, \frac{1}{20}\right]$
d_1	SARS-induced death rate of I	0.0079	$[0.001, 0.02]$
d_2	SARS-induced death rate of J	0.0068	$[0.001, 0.02]$
ε_E	Transmission coefficient for E	0	$[0, 0.18]$
ε_Q	Transmission coefficient for Q	0	$[0, 0.18]$
ε_J	Transmission coefficient for J	0	$[0, 0.84]$
e_{pop}	Precaution level in the population	variable	
C	Cost of precautions (relative to the cost of SARS)	variable	

The model yields the following system of differential equations.

$$\frac{dS}{dt} = \Pi - \mu S - (1 - e_{pop}) \left(\frac{\beta I + \beta \varepsilon_E E + \beta \varepsilon_Q Q + \beta \varepsilon_J J}{N} \right) S \tag{2}$$

$$\frac{dE}{dt} = (1 - e_{pop}) \left(\frac{\beta I + \beta \varepsilon_E E + \beta \varepsilon_Q Q + \beta \varepsilon_J J}{N} \right) S - (\mu + \gamma_1 + \kappa_1) E \tag{3}$$

$$\frac{dQ}{dt} = \gamma_1 E - (\mu + \kappa_2) Q \tag{4}$$

$$\frac{dI}{dt} = \kappa_1 E - (\mu + \gamma_2 + d_1 + \sigma_1) I \tag{5}$$

$$\frac{dJ}{dt} = \kappa_2 Q + \gamma_2 I - (\mu + d_2 + \sigma_2) J \tag{6}$$

$$\frac{dR}{dt} = \sigma_1 I + \sigma_2 J - \mu R \tag{7}$$

Above, we assume that e_{pop} is constant in time.

2.2. Equilibria of the dynamics

The disease-free equilibrium (DFE) the system (2) – (7) is given by

$$(S^0, E^0, Q^0, I^0, J^0, R^0) = \left(\frac{\Pi}{\mu}, 0, 0, 0, 0, 0\right). \tag{8}$$

The effective reproduction number, $\mathcal{R}$, is derived in A.1 and is given by

$$\mathcal{R} = (1 - e_{pop})\mathcal{R}_0 \tag{9}$$

where

$$\mathcal{R}_0 = \frac{\beta}{D_E}\left(\varepsilon_E + \varepsilon_Q \frac{\gamma_1}{D_Q} + \frac{\kappa_1}{D_I} + \varepsilon_J \frac{\gamma_1\kappa_2}{D_J D_Q} + \varepsilon_J \frac{\gamma_2\kappa_1}{D_I D_J}\right) \tag{10}$$

is the basic reproduction number (in a population without any precautionary efforts) and

$$D_E = \mu + \kappa_1 + \gamma_1, \tag{11}$$

$$D_I = \mu + \sigma_1 + d_1 + \gamma_2, \tag{12}$$

$$D_J = \mu + \sigma_2 + d_2, \tag{13}$$

$$D_Q = \mu + \kappa_2. \tag{14}$$

In the endemic equilibrium (EE), as derived in A.2, the force of the infection can be expressed as

$$\lambda^* = \frac{(\mathcal{R} - 1)D_E D_I D_J D_Q}{T} \tag{15}$$

where

$$\begin{aligned} T = {} & D_I D_J D_Q + \kappa_1 D_J D_Q + \gamma_1 D_I D_J + \kappa_2\gamma_1 D_I + \kappa_1\gamma_2 D_Q + \frac{\sigma_1\kappa_1}{\mu} D_J D_Q \\ & + \frac{\sigma_2\kappa_2\gamma_1}{\mu} D_I + \frac{\sigma_2\kappa_1\gamma_2}{\mu} D_Q. \end{aligned} \tag{16}$$

The endemic equilibrium is then given by

$$S^* = \frac{\Pi}{\mu + \lambda^*}, \tag{17}$$

$$E^* = \lambda^* \frac{S^*}{D_E}, \tag{18}$$

$$Q^* = \lambda^* \frac{S^* \gamma_1}{D_E D_Q}, \tag{19}$$

$$I^* = \lambda^* \frac{S^* \kappa_1}{D_E D_I}, \tag{20}$$

$$J^* = \lambda^* \frac{S^*}{D_E} \left(\frac{\kappa_2 \gamma_1}{D_J D_Q} + \frac{\kappa_1 \gamma_2}{D_I D_J} \right), \tag{21}$$

$$R^* = \lambda^* \frac{S^*}{D_E} \left(\frac{\sigma_1 \kappa_1}{\mu D_I} + \frac{\sigma_2 \kappa_2 \gamma_1}{\mu D_J D_Q} + \frac{\sigma_2 \kappa_1 \gamma_2}{\mu D_I D_J} \right). \tag{22}$$

While we do not focus on stability of the equilibria, it follows directly from [58] that the DFE is locally asymptotically stable if $\mathcal{R} < 1$. Also, by (15), the EE exists only if $\mathcal{R} > 1$. Based on the numerical simulations, we believe that DFE is globally asymptotically stable if $\mathcal{R} \leq 1$ and EE is globally asymptotically stable if $\mathcal{R} > 1$.

2.3. Game-theoretic framework

We incorporate the game-theoretic component into the above transmission dynamics as done in [7].

A game is played by susceptible individuals who decide how many precautions they should take. All individuals are assumed to be rational, have complete information about the SARS epidemic, and act in their own interest. They evaluate costs and benefits of their own action and the actions of others. They choose the action that maximizes their own net payoffs (benefits minus costs).

We focus on a single individual using a strategy e when the rest of the population uses a strategy e_{pop}. If the population is large enough, the decision of a single individual will not have a significant impact on the steady state of the dynamics. Thus, the force of infection will be either 0 if $\mathcal{R} < 1$ and the population is in the disease-free equilibrium, or it is given by (1) if $\mathcal{R} > 1$.

The focal individual uses e instead of e_{pop} and thus their rate of becoming infected is $\lambda = \frac{1-e}{1-e_{pop}} \lambda^*$. Hence, the probability of a susceptible individual becoming exposed is given by

$$\pi_{S \to E} = \begin{cases} \dfrac{\frac{1-e}{1-e_{pop}} \lambda^*}{\frac{1-e}{1-e_{pop}} \lambda^* + \mu} = \dfrac{(1-e)\lambda^*}{(1-e)\lambda^* + (1-e_{pop})\mu}, & \text{if } \mathcal{R} > 1, \\ 0, & \text{if } \mathcal{R} \leq 1. \end{cases} \tag{23}$$

There is a cost associated with social distancing [51]. Let C be the cost of complete prevention ($e = 1$) relative to the cost of getting infected by SARS. Let us assume

that the cost of the precautionary efforts is linear in e. The payoff to an individual using e in a population where everyone else uses e_{pop} is given by

$$P(e, e_{pop}) = -Ce - \pi_{S\to E}. \tag{24}$$

This equation was developed in [7] and specifies that an individual using strategy e has to pay a cost Ce, and even with this level of precaution, the individual can still contract the disease (and pay a unit cost 1) with probability $\pi_{S\to E}$. The expected payoff (expressed in the units of the disease cost) is given by (24).

In accordance with the static vaccination game model in [7], it is assumed that all individuals make their choices once, simultaneously, and based on the payoffs in the equilibrium of the dynamics (2)–(7). This is a standard assumption successfully used in many scenarios, including models of influenza epidemics [25, 52]. As a result, e_{pop} can be treated as a constant. There are ways to consider a more dynamical approach and explicitly model the time evolution of the protective strategies; see for example [6, 41, 9, 19, 10, 49, 5, 1].

3. Analysis

The focal individual tries to maximize $P(e, e_{pop})$ given in (24). If the basic reproduction number, $\mathcal{R}_0$, given by (10), is such that $\mathcal{R}_0 < 1$, then the population will reach disease free equilibrium even if nobody uses any precautions. Hence, $e = 0$ is the Nash equilibrium of the game.

However, for SARS, $\mathcal{R}_0 > 1$. Let us denote

$$e_{HI} = 1 - \frac{1}{\mathcal{R}_0}, \tag{25}$$

the minimal precautionary efforts needed to achieve $\mathcal{R} \le 1$. If $e_{pop} > e_{HI}$, then $\mathcal{R} < 1$ and the optimal action for the focal individual is $e = 0$.

Hence, we will assume $e_{pop} < e_{HI}$. We have

$$\frac{\partial}{\partial e} P(e, e_{pop}) = -C + \frac{\partial}{\partial e}\pi_{S\to E} = -C + \lambda^* \frac{(1 - e_{pop})\mu}{\big((1-e)\lambda^* + (1 - e_{pop}\mu)\big)^2} \tag{26}$$

$$\frac{\partial^2}{\partial e^2} P(e, e_{pop}) = 2(\lambda^*)^2 \frac{(1 - e_{pop})\mu}{\big((1-e)\lambda^* + (1 - e_{pop}\mu)\big)^3} > 0. \tag{27}$$

Consequently, the function $P(e, e_{pop})$ has a maximum either at $e = 0$ or $e = 1$ and the Nash equilibrium is a solution of

$$P(0, e_{pop}) = P(1, e_{pop}), \tag{28}$$

i.e., the solution of

$$\frac{\lambda^*}{\lambda^* + (1 - e_{pop})\mu} = C. \tag{29}$$

As derived in A.3, the Nash equilibrium is given by

$$e_{NE} = \begin{cases} 1 - \dfrac{(1-C)\frac{D_E D_I D_J D_Q}{T}}{\mathcal{R}_0(1-C)\frac{D_E D_I D_J D_Q}{T} - \mu C}, & \text{if } C < C_{crit}, \\ 0, & \text{otherwise.} \end{cases} \tag{30}$$

where

$$C_{crit} = \begin{cases} \dfrac{(\mathcal{R}_0 - 1)\frac{D_E D_I D_J D_Q}{T}}{(\mathcal{R}_0 - 1)\frac{D_E D_I D_J D_Q}{T} + \mu}, & \text{if } \mathcal{R}_0 > 1, \\ 0, & \text{otherwise.} \end{cases} \tag{31}$$

4. Results

For any parameter values, the Nash equilibrium e_{NE}, i.e., the optimal precautionary level is given by (30). The dependence of e_{NE} on C is illustrated in Figure 2(a). When $C = 0$, $e_{NE} = e_{HI}$. As C increases, e_{NE} decreases. When C reaches the critical value C_{crit}, $e_{NE} = 0$.

Similarly, we can investigate the value of the reproduction number at the population that uses the Nash equilibrium level of precautions. This is shown in Figure 2(b). When $C \approx 0$, $\mathcal{R} \approx 1$ but still larger than 1. As C increases, $\mathcal{R}$ increases and reaches $\mathcal{R}_0$ when $C \geq C_{crit}$.

We performed uncertainty and sensitivity analysis using the Latin hyper-cube sampling with partial rank correlation coefficient (LHS-PRCC) scheme [11, 55, 47, 37]; see also A.4.

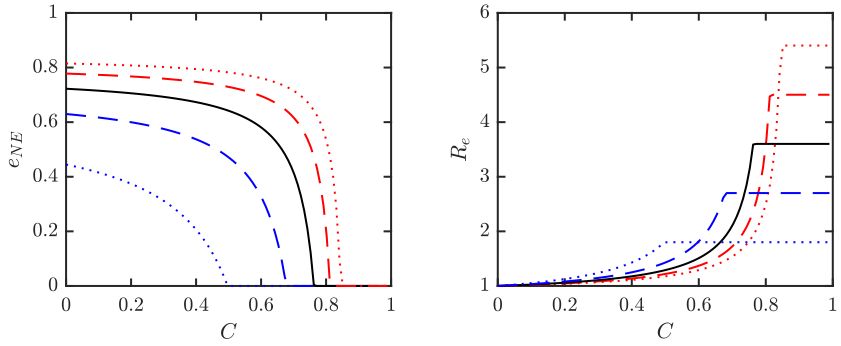
Figure 3(a) shows the results of uncertainty analysis, i.e., the distribution of e_{NE} among all the sampled parameter values. The most frequent values of e_{NE} are between 0.5 and 0.7 and the average value is approximately 0.47.

Figure 3(b) shows the sensitivity of e_{NE} on various parameters. There is a strong negative correlation between the optimal voluntary precaution level e_{NE} and the cost of the precautions, C. As seen above in Figure 2, increasing C decreases e_{NE}. Increasing recovery rate, σ_2, or isolation rate, γ_2, also causes e_{NE} to decline. On the other hand, increasing the transmission rate, β, or the transmission coefficient from isolated individuals, ε_J, causes e_{NE} to increase. The influence of other parameters on e_{NE} is relatively small.

Figure 3(c) shows the results of uncertainty analysis and the distribution of C_{crit}. The most frequent values of C_{crit} are between 0.5 and 0.8; the average value is approximately 0.54. Figure 3(d) shows the sensitivity of C_{crit} on various parameters. The sensitivity indices are very similar to the sensitivity of e_{NE}.

5. Discussion and Limitations

Our model has limitations connected to the assumption of infinite homogeneous population. While this assumption is common in epidemiological modeling, other approaches are possible.



(A) (B)

FIGURE 2. (a) Values of the optimal individual precautionary levels e_{NE} as a function of the relative cost of the precautions C. (b) Values of the effective reproduction number $\mathcal{R}$ when the population uses e_{NE}. Parameter values are as specified in Table 1; the rate at which non-quarantined cases develop symptoms varies from $\kappa_1 = 0.05$ (blue dotted), $\kappa_1 = 0.075$ (blue dashed), $\kappa_1 = 0.1$ (black solid), $\kappa_1 = 0.125$ (red dashed), $\kappa_1 = 0.15$ (red dotted).

For example, the use of simulations, such as in [33, 35, 36, 40, 4, 3, 32], could allow for flexibility and realism in the modeling approach, taking into account both geographical and social heterogeneity of an explicitly finite population.

In our game-theoretic setup, we assumed that the cost is linear. As demonstrated in [54], the assumption is not necessary. If the cost is assumed to be an increasing and concave down function, then the analysis and results would be qualitatively similar. It is unclear how much the result would change if no assumptions about the second derivative were made.

Finally, to evaluate the payoffs of the game, we assumed that the underlying transmission dynamics reached an equilibrium. This assumption can be relaxed by considering adaptive dynamics as done in [6]. This dynamical approach can yield more complex outcomes. For example, temporal oscillations in the use of protective strategies can emerge; this was demonstrated for example on the use of insecticide treated nets in the prevention of visceral leishmaniasis [23] and malaria [41].

6. Conclusions

In this paper, we adapted the compartmental model of SARS transmission developed by [28]. We added the game-theoretic component incorporating voluntary precautions such as masking or limiting contacts with others. The model shows that the optimal level of precautions significantly depends on the individual costs

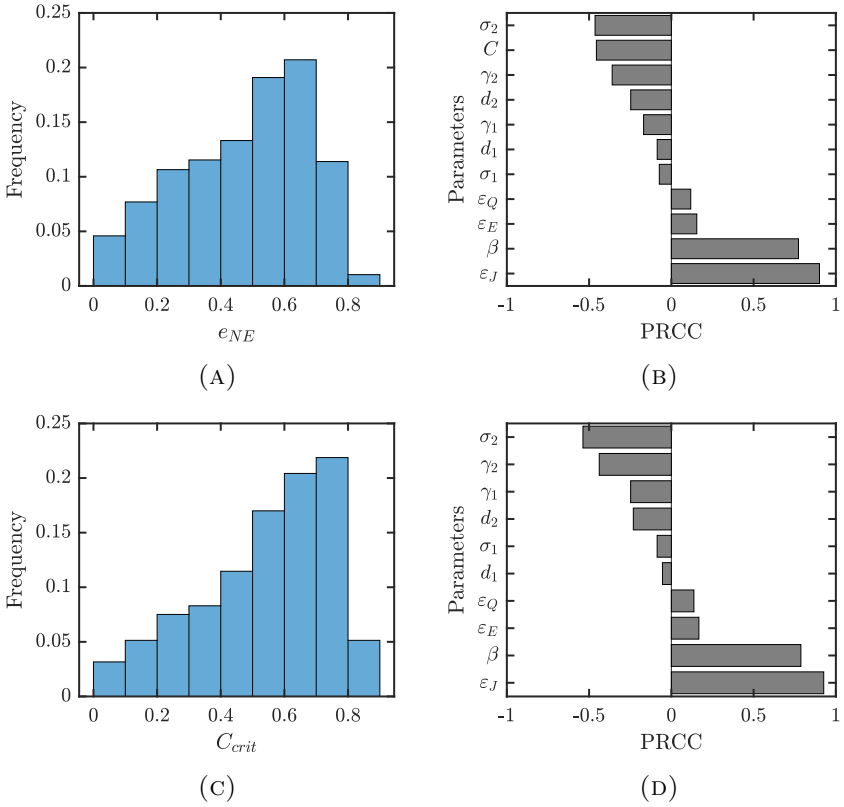


FIGURE 3. Uncertainty and sensitivity analysis. (a) Uncertainty of e_{NE}, the average is around 0.36, (b) sensitivity of e_{NE}, (c) uncertainty of C_{crit}, the average is about 0.4, and (d) sensitivity of C_{crit} . The parameter ranges are as in Table 1. Only parameters with sensitivity over 0.05 are shown in figures (b) and (d).

of such actions relative to the cost of SARS. The more costly those actions are (in true or perceived costs), the lower the level of precautions the individuals will adopt. As soon as the costs rise above a relatively low threshold, the individuals will not voluntarily use any precautions. Since the cost is expressed relative to the cost of SARS, a decrease of the actual or perceived SARS infection has the same effect as the increase of the cost of precautions.

This finding is in agreement with what we see happening for COVID-19. When COVID-19 first appeared and was considered very serious, people isolated and

masked as much as possible. However, as the perceived negative effects of COVID-19 continued to decline, the recommended levels of precautions were also dropping.

Another result of our model is that voluntary precautions alone are not enough to reduce the reproduction number to a values less than 1, i.e., voluntary precautions alone cannot eliminate SARS nor mitigate the risks of outbreaks. That is in agreement with the general game considered in [26] as well as models for specific diseases, e.g., yellow fever [14]. In these cases, the cost of prevention is high relative to the cost of the disease. However, and this is seen from our model as well, when the relative cost is quite low, voluntary precautions may lower the reproduction number close enough to 1 so that the disease incidence or prevalence is no longer a public health concern. This phenomenon was already observed in models of many vector-borne diseases, including dengue [21], and lymphatic filariasis [54].

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Author contribution

L.J.C.A - writing - original draft, methodology, supervision, conceptualization
A.C. - writing - original draft, formal analysis
R.M.D. - writing - original draft, formal analysis
B.L. - writing - original draft, formal analysis
H.O. - writing - original draft, writing - review and editing, methodology, supervision, conceptualization, formal analysis, funding acquisition
J.R. - writing - original draft, writing - review and editing, software, visualization, methodology, supervision, conceptualization, resources
D.T. - writing - original draft, writing - review and editing, methodology, supervision, conceptualization, formal analysis, visualization, funding acquisition

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Appendix A. Calculations

A.1. Reproduction number

The reproduction number is derived using the next-generation matrix method [58]. We order the compartments with the infections as E, I, Q and J. The new infections only appear in E, and thus

$$\mathcal{F} = \begin{bmatrix} \frac{S}{N}\beta(1-e_{pop})(I+\varepsilon_E E+\varepsilon_Q Q+\varepsilon_J J) \\ 0 \\ 0 \\ 0 \end{bmatrix} \tag{32}$$

The vector $\mathcal{V}$ considers the other transfers out of these infectious compartments minus all the transfers coming into the compartments other than new infections.

Thus,

$$\mathcal{V} = \begin{bmatrix} (\mu+\gamma_1+\kappa_1)E \\ (\mu+\kappa_2)Q-\gamma_1 E \\ (\mu+d_1+\gamma_1+\gamma_2+\sigma_1)I-\kappa_1 E \\ (\mu+d_2+\sigma_2)J-\gamma_2 I-\kappa_2 Q \end{bmatrix} \tag{33}$$

The Jacobian matrix of $\mathcal{F}$ evaluated at DFE is given by

$$F = \beta(1-e_{pop}) \begin{bmatrix} \varepsilon_E & \varepsilon_Q & 1 & \varepsilon_J \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix} \tag{34}$$

and the Jacobian matrix of $\mathcal{V}$ is

$$V = \begin{bmatrix} \mu+\gamma_1+\kappa_1 & 0 & 0 & 0 \\ -\gamma_1 & \mu+\kappa_2 & 0 & 0 \\ -\kappa_1 & 0 & \mu+d_1+\gamma_2+\sigma_1 & 0 \\ 0 & -\kappa_2 & -\gamma_2 & \mu+d_2+\sigma_2 \end{bmatrix}. \tag{35}$$

Let us denote

$$D_E = \mu+\kappa_1+\gamma_1, \tag{36}$$

$$D_I = \mu+\sigma_1+d_1+\gamma_2, \tag{37}$$

$$D_J = \mu+\sigma_2+d_2, \tag{38}$$

$$D_Q = \mu+\kappa_2. \tag{39}$$

We get

$$V^{-1} = \frac{1}{D_E} \begin{bmatrix} 1 & 0 & 0 & 0 \\ \frac{\gamma_1}{D_Q} & \frac{D_E}{D_Q} & 0 & 0 \\ \frac{\kappa_1}{D_I} & 0 & \frac{D_E}{D_I} & 0 \\ \frac{D_I\gamma_1\kappa_2+D_Q\gamma_2\kappa_1}{D_I D_J D_Q} & \frac{D_E\kappa_2}{D_J D_Q} & \frac{D_E\gamma_2}{D_I D_J} & \frac{D_E}{D_J} \end{bmatrix} \tag{40}$$

Thus, the effective reproduction number $\mathcal{R}$, calculated as the spectral radius of the next-generation matrix, FV^{-1}, is given by

$$\mathcal{R} = \frac{\beta(1-e_{pop})}{D_E}\left(\varepsilon_E + \varepsilon_Q\frac{\gamma_1}{D_Q} + \frac{\kappa_1}{D_I} + \varepsilon_J\frac{\gamma_1\kappa_2}{D_J D_Q} + \varepsilon_J\frac{\gamma_2\kappa_1}{D_I D_J}\right). \tag{41}$$

We note that [28] used an alternative, direct approach that yields the same formula for $\mathcal{R}$.

A.2. Endemic equilibrium

Setting the time derivative to 0 in (2)–(7) yields

$$0 = \Pi - \mu S - \lambda S \tag{42}$$

$$0 = \lambda S - D_E E \tag{43}$$

$$0 = \gamma_1 E - D_Q Q \tag{44}$$

$$0 = \kappa_1 E - D_I I \tag{45}$$

$$0 = \kappa_2 Q + \gamma_2 I - D_J J \tag{46}$$

$$0 = \sigma_1 I + \sigma_2 J - \mu R \tag{47}$$

where

$$\lambda = (1 - e_{pop})\beta \left(\frac{I}{N} + \varepsilon_E \frac{E}{N} + \varepsilon_Q \frac{Q}{N} + \varepsilon_J \frac{J}{N} \right). \tag{48}$$

Starting at (42) and going one equation at a time, we get

$$S = \frac{\Pi}{\mu + \lambda} \tag{49}$$

$$E = \frac{\lambda S}{D_E} \tag{50}$$

$$Q = \frac{\gamma_1 E}{D_Q} = \frac{\lambda \gamma_1 S}{D_E D_Q} \tag{51}$$

$$I = \frac{\kappa_1 E}{D_I} = \frac{\lambda \kappa_1 S}{D_E D_I} \tag{52}$$

$$J = \frac{\kappa_2 Q + \gamma_2 I}{D_J} = \frac{\lambda S}{D_E} \left(\frac{\kappa_2 \gamma_1}{D_J D_Q} + \frac{\kappa_1 \gamma_2}{D_I D_J} \right) \tag{53}$$

$$R = \frac{\sigma_1 I + \sigma_2 J}{\mu} = \frac{\lambda S}{D_E} \left(\frac{\sigma_1 \kappa_1}{\mu D_I} + \frac{\sigma_2 \kappa_2 \gamma_1}{\mu D_J D_Q} + \frac{\sigma_2 \kappa_1 \gamma_2}{\mu D_I D_J} \right). \tag{54}$$

Thus, we get

(55)

$$N = S + E + Q + I + J + R$$

(56)

$$= S \left(1 + \frac{\lambda}{D_E} \left[1 + \frac{\gamma_1}{D_Q} + \frac{\kappa_1}{D_I} + \frac{\kappa_2 \gamma_1}{D_J D_Q} + \frac{\kappa_1 \gamma_2}{D_I D_J} + \frac{\sigma_1 \kappa_1}{\mu D_I} + \frac{\sigma_2 \kappa_2 \gamma_1}{\mu D_J D_Q} + \frac{\sigma_2 \kappa_1 \gamma_2}{\mu D_I D_J} \right] \right)$$

Setting

(57)

$$T = D_I D_J D_Q + \kappa_1 D_J D_Q + \gamma_1 D_I D_J + \kappa_2 \gamma_1 D_I + \kappa_1 \gamma_2 D_Q + \frac{\sigma_1 \kappa_1}{\mu} D_J D_Q + \frac{\sigma_2 \kappa_2 \gamma_1}{\mu} D_I + \frac{\sigma_2 \kappa_1 \gamma_2}{\mu} D_Q,$$

(56) yields

$$N = S \left(1 + \frac{\lambda}{D_E} \frac{T}{D_I D_J D_Q} \right). \tag{58}$$

By (58) and (50)–(54), (48) becomes

(59)
$$\lambda = \frac{\lambda}{1+\frac{\lambda}{D_E}\frac{T}{D_I D_J D_Q}}(1-e_{pop})\frac{\beta}{D_E}\left(\kappa_1+\varepsilon_E+\varepsilon_Q\frac{\gamma_1}{D_Q}+\varepsilon_J\left\{\frac{\kappa_2\gamma_1}{D_J D_Q}+\frac{\kappa_1\gamma_2}{D_I D_J}\right\}\right)$$

(60)
$$= \frac{\lambda}{1+\frac{\lambda}{D_E}\frac{T}{D_I D_J D_Q}}\mathcal{R}.$$

Hence,

$$\lambda = \frac{(\mathcal{R}-1)D_E D_I D_J D_Q}{T}. \tag{61}$$

A.3. Nash equilibrium

As derived in the main text, the Nash equilibrium has to solve

$$\frac{\lambda^*}{\lambda^*+(1-e_{pop})\mu} = C. \tag{62}$$

By (61) and (9),

$$\lambda^* = \big((1-e_{pop})\mathcal{R}_0 - 1\big)k \tag{63}$$

where

$$k = \frac{D_E D_I D_J D_Q}{T}. \tag{64}$$

Thus, (62) becomes

$$\frac{\big((1-e_{pop})\mathcal{R}_0-1\big)k}{\big((1-e_{pop})\mathcal{R}_0-1\big)k+(1-e_{pop})\mu} = C. \tag{65}$$

This is equivalent to

$$\big((1-e_{pop})\mathcal{R}_0-1\big)k = \big((1-e_{pop})\mathcal{R}_0-1\big)kC+(1-e_{pop})\mu C \tag{66}$$

and solving for e_{pop} yields

$$e_{NE} = 1-\frac{(1-C)k}{(1-C)k\mathcal{R}_0-\mu C}. \tag{67}$$

Moreover, the left-hand side of (65) can be rearranged as

$$\frac{1}{1+\frac{\mu}{\left(\mathcal{R}_0-\frac{1}{1-e_{pop}}\right)k}} \tag{68}$$

which is decreasing in e_{pop}. So, the maximum occurs at $e_{pop}=0$ and it is

$$C_{crit} = \frac{(\mathcal{R}_0-1)k}{(\mathcal{R}_0-1)k+\mu}. \tag{69}$$

When $C > C_{crit}$, the equation (65) does not have a positive solution and thus $e_{NE}=0$.

Also, all of the above calculations made sense for $\mathcal{R}_0 > 1$. When $\mathcal{R}_0 \leq 1$, the dynamics tends to the disease-free equilibrium and thus it makes sense to consider $C_{crit} = 0$.

To summarize, there is a critical value C_{crit} for the cost C given by

$$C_{crit} = \begin{cases} \frac{(\mathcal{R}_0-1)k}{(\mathcal{R}_0-1)k+\mu}, & \text{if } \mathcal{R}_0 > 1, \\ 0, & \text{otherwise.} \end{cases} \tag{70}$$

A.4. LHS-PRCC

This section contains basic information on the uncertainty and sensitivity analysis using the LHS-PRCC scheme [11, 55]. The full description of the LHS-PRCC scheme is in [47] and the MATLAB and R implementations is in [37]. A shorter description such as the one below, can be found, for example, in [56].

For the Latin Hyper-cube Sampling (LHS), the parameter ranges are divided into intervals of equal probability. Those intervals are then sampled, independently for each parameter. LHS is also called a stratified sampling without replacement. The sampling yields an unbiased estimate of the expected model output. Compared to a simple random sampling, it needs fewer samples to achieve the same accuracy [48].

PRCC, the partial rank correlation coefficient, between a model parameter P and the model output O is defined by

$$r_{R_P,R_O} = \frac{\mathrm{Cov}(R_P, R_O)}{\sqrt{\mathrm{Var}(R_P)\mathrm{Var}(R_O)}} \tag{71}$$

where R_P and R_O are residuals of the rank-transformed linear regression models for P and O.