Zhao S, Bauch CT, He D. 2018 Strategic decision making about travel during disease outbreaks: a game theoretical approach. J. R. Soc. Interface 20180515. http://dx.doi.org/10.1098/rsif.2018.0515

Strategic Decision-making about Travel during Disease Outbreaks: a Game Theoretical Approach

Shi Zhao[1], Chris T. Bauch[2,*] & Daihai He[1,*]

1 Department of Applied Mathematics, Hong Kong Polytechnic University, Hong Kong

2 Department of Applied Mathematics, University of Waterloo, Canada

* Correspondence to: `daihai.he@polyu.edu.hk` (D.H.) and `cbauch@uwaterloo.ca` (C.T.B.)

August 20, 2018

Abstract

Visitors can play an important role in the spread of infections. Here, we incorporate an epidemic model into a game theoretical framework to investigate the effects of travel strategies on infection control. Potential visitors must decide whether to travel to a destination that is at risk of infectious disease outbreaks. We compare the individually optimal (Nash equilibrium) strategy to the group optimal strategy that maximizes the overall population utility. Economic epidemiological models often find that individual and group optimal strategies are very different. In contrast, we find perfect agreement between individual and group optimal strategies across a wide parameter regime. For more limited regimes where disagreement does occur, the disagreement is (1) generally very extreme; (2) highly sensitive to small changes in infection transmissibility and visitor costs/benefits; and (3) can manifest either in a higher travel volume for individual optimal than group optimal strategies, or *vice versa*. The simulations show qualitative agreement with the 2003 Severe Acute Respiratory Syndrome (SARS) outbreak in Beijing, China. We conclude that a conflict between individual and group optimal visitor travel strategies during outbreaks may not generally be a problem, although extreme differences could emerge suddenly under certain changes in economic and epidemiological conditions.

Introduction

Visitors can play an important role in the transmission and spread of infectious diseases. They can serve as susceptible hosts and be infected while staying in one place and then act as mobile sources of case imports to other populations [1, 2, 3]. On the one hand, more visitors can lead to substantial benefits for the local economy and businesses. On the other hand, some infectious diseases spread aggressively in major tourism destinations (e.g., Hong Kong, New York, Singapore, Toronto, Beijing), and a large number of visitors can have unexpected impacts on public health [3, 4, 5]. For example, Severe Acute Respiratory Syndrome (SARS) was introduced to Beijing, China by a few infected visitors in early March 2003, resulting in a large epidemic[6, 7, 8, 9, 10, 11]. Other examples where visitors have played a role in regional or international spread include pandemic influenza [12, 13, 14], Ebola fever [15] and Middle East respiratory syndrome coronavirus (MERS-CoV) [16]. Enforcing restrictions on incoming visitors could be an efficient way to control local disease outbreaks [7, 17, 18, 19], but the decision to restrict visitors must be weighed carefully due to the economic and social repercussions.

Game theory attempts to analyse situations where individuals must make decisions in a group environment and where each individual's decision influences the payoff received by the others in the group [20]. Many interventions (such as vaccination and social distancing) create positive externalities, i.e., benefits to those who did not participate in the intervention, because of herd immunity generated by interruption of transmission. Hence, many previous models have illustrated the discrepancy between the optimal individual strategy that maximizes personal interest, and the strategy that serves the group best by minimizing the overall health burden on the population [21, 22, 23, 24, 25, 26]. Although several factors may alter this picture and have been explored in successive work — such as the beneficial effects of social norms and prosocial vaccination [51, 50] — these models often illustrate a conflict between group and individual optima across a very broad region of parameter space, covering most epidemiologically and economically relevant regimes [21, 22, 24, 25].

However, this previous research has been mostly concerned with individuals making decisions in a closed population where the disease is already established and is spreading [21, 22, 23, 24, 25, 27, 28, 29, 30, 31], and does not consider multipopulation interactions or the strategic considerations faced by a visitor deciding whether to travel to an affected area during an outbreak. In the context of travel decisions, game theory can be used to answer questions such as whether travelling or not travelling to a location is optimal according to a criterion of self-interest, and the answers it provides can be contrasted with optimal control strategy from the health authority perspective, in terms of maximizing overall population utility.

In this work, we incorporate an epidemic model (based on the classic Susceptible-Infectious-Recovered model) into a game theoretical framework to investigate the effects of strategic decisions about travel on local disease control. In contrast to many previous game theoretical analyses of decision making in epidemiological systems in a closed population, for this visitor's game we find perfect agreement between the individual and group optimal strategies for a range of epidemiologically and economically plausible parameter values. This agreement can be observed in two forms: individual and group optimal strategies both completely reject travelling when the real or perceived disease risk level are sufficiently high, or both strategies allow free travel when the real or perceived disease risk level is sufficiently low. However, disagreement (or conflict) between the individual visitor strategy and the group optimal strategy are observed in two forms: an overload or deficit of visitors compared to the group optimum. In regions where disagreement occurs, the disagreement between the individual optimum (corresponding to a "voluntary entrance" scheme) and the group optimum (corresponding to a "restricted entrance" scheme) is significant. During an outbreak, this conflict is likely to occur at any real or perceived disease risk level. More importantly, in this region, the model outcomes are highly sensitive to small changes in infection transmissibility and visitor costs/benefits. For certain parameter regimes, uncontrolled visitor inflow could result in unexpected large-scale outbreaks when the disease risk level suddenly increases by even a small amount, and local health authority's travel restrictions could effectively control disease outbreaks when visitor inflow is considered to be "overloaded" during epidemics. Interestingly, the faster the disease risk information is updated, the more likely a discrepancy will occur. Moreover, faster disease risk information updating could effectively prevent visitor inflow "overload" and therefore stop an outbreak.

The remaining parts of this work are organized as follows. In the next two sections, we establish a game theorerical framework including both travelling and local populations, to model the individual decision making process. In the subsequent section, the results are presented along with a detailed discussion.

Travelling Game

Our game is a population game where players are individuals in a homeland population (the "travelling population") deciding whether or not to travel to an affected destination. These individuals can move through the following states:

$$\text{individual in homeland} \rightarrow \text{potential visitor} \rightarrow \text{visitor outside} \rightarrow \text{visitor inside} \rightarrow \text{individual in homeland.} \tag{1}$$

A certain fraction of individuals in a homeland population are designated as potential visitors, who have the economic means and opportunities for travel. A potential visitor may adopt a strategy of travelling to the destination and leaves their homeland, becoming a "visitor outside". Upon arrival at the destination, they become a "visitor inside", and subsequently they become a "removed visitor" and re-join the homeland population, again as a potential visitor. A potential visitor corresponds to N_1 in Table 1, a visitor outside corresponds to ρN_1 in the term $f(\rho)$ in Eqns. 7, a visitor inside corresponds to $(S_1 + I_1 + R_1)$ in Supplementary Material S3, and an individual in homeland means that a visitor has been removed from the system and re-joins individuals in the homeland. More details of the steps individuals may take in travelling can be found in Supplementary Material S1. Fig. 1 presents the process of a "travelling" individual joining the epidemic system (i.e., from "potential visitor" to "individual in homeland").

For simplicity, we suppose that every individual receives the same information and picks strategies in the same way (i.e., with equivalent preferences and equivalent payoff for the same strategy). An individual can decide whether to travel (i.e., the "travelling" strategy) or not to travel (i.e., the "non-travelling" strategy) to their destination. We use r_1 to denote the perceived cost (negative payoff) of morbidity and/or mortality risk (i.e., the risk of disease, or as a term of "health cost") from infection. Similarly, we use r_0 to denote the perceived cost of the risk

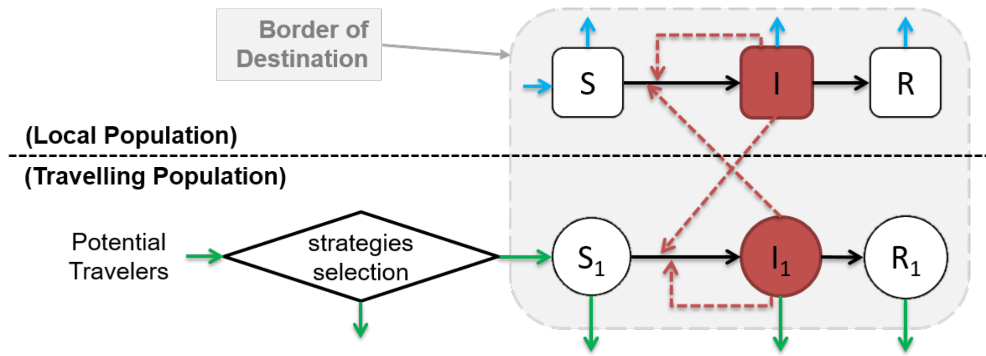

Figure 1: The epidemic model diagram. Black arrows represent infection status transition paths and red dashed arrows represent transmission paths. The light blue arrows represent natural births and deaths, and green arrows represent visitor entry and exit. Square compartments represent local classes, circular compartments represent visitor classes, and the diamond denotes the "decision" process of potential visitors. Red compartments represent infectious classes. The light grey area (surrounded by a grey dashed line) represents "inside border". The horizontal black dashed line separates the total population into "local population" (or local residents) and "travelling population" (as in Path 1).

of utility loss for adopting the "non-travelling" strategy, since those individuals lose economic or social opportunities. Therefore, we write the payoff for an individual following the travelling strategy as

$$E_1 = -\alpha \cdot \phi(\rho; P) \cdot r_1, \tag{2}$$

where α represents the probability that an epidemic occurs at the destination during a traveller's visit (or, $\alpha = 1$ for an ongoing epidemic that the traveller knows about before departure), $\phi(\rho; P)$ is the probability that a visitor is infected during the trip (to the epidemic destination) given that the pre-existing immunity level in the destination population is P, and ρ is the overall proportion of potential visitors who adopted the "travel" strategy.

To assess the risk of a visitor being infected during the trip, we need to know the basic reproduction number of the disease, $\mathcal{R}_0$, i.e., the expected number of secondary cases generated by a typical primary case during his/her infectious period in an otherwise susceptible population. In the case of $\mathcal{R}_0 > 1$, we have $\phi(\rho; P) = 0$ if $P \geqslant \left(1 - \frac{1}{\mathcal{R}_0}\right)$ (see Supplementary Material S2.1). This is called perfect herd immunity, i.e., an outbreak cannot occur when the population immunity level is greater than $\left(1 - \frac{1}{\mathcal{R}_0}\right)$ [32, 33]. We denote the payoff of an individual following the non-travelling strategy as

$$E_0 = -r_0, \tag{3}$$

Since this is a population game, we also define a mixed strategy (i.e., "p-strategy"), where players follow the travelling strategy with a probability p and follow the non-travelling strategy with a probability $(1 - p)$. The payoff function is then

$$
\begin{aligned}
E(p,\rho;P) &= pE_1 + (1-p)E_0 \\
&= -p\alpha r_1 \cdot \phi(\rho;P) - (1-p)r_0.
\end{aligned} \tag{4}
$$

The game remains unchanged if we scale the payoff function by a constant; thus, we eliminate one parameter in Eqn. 4 by leaving only the relative risk, $r = \frac{r_0}{r_1}$. Normally, we have $0 < r_0 \ll r_1$ since the payoff of utility loss, r_0 in Eqn. 3, should be less than that of health loss, r_1 in Eqn. 2, if the disease is severe or potentially deadly. Hence we assume $0 < r \ll 1$ in general. Furthermore, we have

$$
E(p,\rho;P) = p \cdot [r - \alpha\phi(\rho;P)] - r. \tag{5}
$$

For convenience, we denote $\phi(\rho;P)$ as $\phi(\rho)$ and $E(p,\rho;P)$ as $E(p,\rho)$ and fix P in the rest of this work. We can show that the individual equilibrium (p^*) of the game exists, is the unique Nash equilibrium, and is stably convergent (see Supplementary Material S2.1).

We formulate the (scaled) costs of all potential visitors (game players) as

$$
\Upsilon(\rho) = \rho\alpha \cdot \phi(\rho) + (1-\rho)r, \tag{6}
$$

where all terms have the same meaning as in Eqn. (5). More details are provided in Supplementary Material S2.2. We also define the group (Pareto) optimum ρ^* as the value of ρ for which the population average cost function $\Upsilon(\rho)$ of all potential visitors (i.e., all game players) is minimized.

Epidemic Model

Formulation of Epidemic Model

To specify the infection probability $\phi(\rho)$, we adopt the standard susceptible-infectious-removed (SIR) model. Individuals of the destination population (excluding visitors) are categorized as susceptible to the disease (S, those who may be infected), infectious (I, i.e., those capable of transmitting disease), or removed (R, these who are either recovered and immunized or died). Similarly, visitors are also categorized as susceptible (S_1), infectious (I_1), or removed (R_1). We use S, I, and R (or S_1, I_1 and R_1) to denote the proportions of susceptible, infectious and recovered individuals in the destination (visitor) populations, respectively. This patchy population structure was proposed previously in [1, 2, 34, 35]. Before taking the trip, visitors are assumed to be totally susceptible. We illustrate this "local-and-travelling population" interactive epidemic system in Fig. 1. We further assume that the susceptible visitors follow a logistic growth mechanism.

- The visitor population capacity (e.g., the number beds in hotels) of one place is finite and assumed to be a constant.
- Low (/high) volume of visitors will increase (/decrease) the recruitment effort of travellers for a business trip and decrease (/increase) the expense for a recreation trip.

Thus, logistic growth is a reasonable choice. After eliminating R' and R'_1 (see Supplementary Material S3 for details), we formulate the epidemic model as

$$
\begin{cases}
S' = \mu \cdot (1 - K_1 - S) - \beta S \cdot (I + I_1) \\
I' = \beta S \cdot (I + I_1) - (\gamma + \mu) I \\
S_1' = f_\rho \cdot \left[1 - \dfrac{S_1 + \left(1 + \frac{\gamma}{\nu}\right) I_1}{K_1} \right] - \beta S_1 \cdot (I + I_1) - \nu S_1 \\
I_1' = \beta S_1 \cdot (I + I_1) - (\gamma + \nu) I_1
\end{cases}
\tag{7}
$$

where $f_\rho = f(\rho) = \rho \lambda N_1$ represents the rate of incoming visitors, K_1 is the maximum visitor capacity that the destination is willing (or able) to accept, N_1 is the number of all players (i.e., all potential visitors), and players who adopt the "travel" strategy, travel from the homeland to the destination at a rate $\lambda = 1/3$ day^{-1} (see Supplementary Material S6.1). We express both K_1 and N_1 in units of proportion of the population threshold (destination population plus the maximum visitor capacity) and we fix N_1. We assume that all trips are three days long, hence visitors return at rate $\nu = 1/3$ day^{-1} (see Supplementary Material S6.3). We summarize all model parameters in Table 1.

The contact term β is a function of $\mathcal{R}_0$. Using the next generation matrix method [42], we derive the basic reproduction number of our epidemic model as

$$
\mathcal{R}_0 = \beta \cdot \left[\frac{(1 - K_1)}{\gamma + \mu} + \frac{K_1}{\gamma + \nu} \right], \tag{8}
$$

thus, $\beta \propto \mathcal{R}_0$ when the values of the other parameters are fixed.

Model Equilibria

We denote the disease-free equilibrium (DFE) as

$$
\mathcal{E}^{(1)} = \left(S^{(1)}, I^{(1)}, S_1^{(1)}, I_1^{(1)} \right) = \left((1 - K_1), 0, \frac{f_\rho K_1}{f_\rho + \nu K_1}, 0 \right),
$$

where $I = I_1 = 0$ and $S_1^{(1)} < K_1$. The DFE ($\mathcal{E}^{(1)}$) is globally stable when $\mathcal{R}_0 < 1$, whereas it is unstable when $\mathcal{R}_0 > 1$. When $\mathcal{R}_0 > 1$, there is an endemic, i.e., the visitor-absent endemic equilibrium ,

$$
\mathcal{E}^{(2)} = \left(S^{(2)}, I^{(2)}, S_1^{(2)}, I_1^{(2)} \right) = \left(\frac{\gamma + \mu}{\beta}, \mu \cdot \left(\frac{1 - K_1}{\gamma + \mu} - \frac{1}{\beta} \right), 0, 0 \right),
$$

where $S_1 = I_1 = 0$. Specifically, $S^{(1)} = \frac{\gamma+\mu}{\beta}$ is the reciprocal of $\mathcal{R}_0$ of the standard SIR model [33]. $\mathcal{E}^{(2)}$ can be realized when f_ρ in S_1' (see Eqn. 7) becomes 0 and it is locally stable. When $\mathcal{R}_0 > 1$, there also exists an endemic equilibrium corresponding to a mixed state of local and visitor infections (i.e., infected visitors), denoted as $\mathcal{E}^{(3)} = \left(S^{(3)}, I^{(3)}, S_1^{(3)}, I_1^{(3)} \right)$. The solution of $\mathcal{E}^{(3)}$ can be obtained explicitly by taking the nonnegative root of $[S', I', S_1', I_1']^T = \mathbf{0}$ ($\mathbf{0}$ represents the zero vector) with both $I \neq 0$ and $I_1 \neq 0$.

Table 1: Summary table of model parameters. The ranges of the parameters are used for the sensitivity analysis.

Parameter	Notation	Value	Range/Remark	Source(s)
Basic reproduction number	$\mathcal{R}_0$	$2.5^{\dagger}$	$[1.0, 10.0]$	[36, 37, 38, 39]
Mean duration that visitors are outside border	λ^{-1}	3 days	$[0.1, 10]$	S6.1
Ratio: $\frac{\text{travelling players}}{\text{population threshold}}$	N_1	7.5%	$[5.0\%, 15.0\%]$	assumed, S2.2 and S3
Ratio: $\frac{\text{visitors capacity}}{\text{population threshold}}$	K_1	7.0%	$[5.0\%, 15.0\%]$	S6.2
Mean infectious period	γ^{-1}	5 days	$[2.0, 10.0]$	[40]
Mean human lifespan	μ^{-1}	70 years	fixed	-
Mean duration that visitors are inside border	ν^{-1}	3 days	$[0.5, 15.0]$	S6.3
Relative risk (as in Eqn. 5)	$r = \frac{r_0}{r_1}$	10^{-3}	$[10^{-4}, 10^{-2}]$	S6.4
Probability of travelling	p	-	$[0.0, 1.0]$	Eqn. 4
Optimal probability of travelling	p^*	-	$[0.0, 1.0]$	S2.1
Proportion of visitors	ρ	-	$[0.0, 1.0]$	Eqn. 2
Optimal proportion of visitors	ρ^*	-	$[0.0, 1.0]$	Eqn. (6) and S2.2
Cost of all game players	Υ	-	-	S2.2
Difference between group and individual optima	$\Delta\rho$	$\rho^* - p^*$	$[-1.0, 1.0]$	Eqn. (10)
Probability that disease outbreak occurs	α	$0.01^{\ddagger}$	$[0.001, 0.02]$	assumed

The point values of the disease parameters reflect influenza, and the ranges of the parameters reflect a broad range of other infectious diseases.
The values and ranges of the parameters related to travel (i.e., K_1, r, ν^{-1} and λ^{-1}) reflect Hong Kong as the default destination.
† One can determine the function $\beta(\mathcal{R}_0)$ explicitly from Eqn. 8, and $\mathcal{R}_0 = 2.5$ is also applicable to the 2003 SARS epidemic according to [6, 7, 9, 10, 11, 41]
‡ $\alpha = 1.0$ during epidemics.

Probability of Visitors becoming Infected

Given the model in Eqn. 7 and the assumption that all individuals in a compartment leave it at the same rate regardless of how long they have been there, we may take the probability of a visitor becoming infected during the trip to be equal to the ratio of the rate at which susceptible visitors (S_1) are infected to the rate at which susceptible visitors (S_1) leave the destination [22],

$$\begin{aligned} \phi(\rho) &= \frac{\beta S_1^{(3)}(I^{(3)} + I_1^{(3)})}{\beta S_1^{(3)}(I^{(3)} + I_1^{(3)}) + \nu S_1^{(3)}} = 1 - \frac{\nu}{\beta(I^{(3)} + I_1^{(3)}) + \nu}, \\ \text{and thus,} \quad \alpha\phi(\rho) &= \alpha - \frac{\nu\alpha}{\beta(I^{(3)} + I_1^{(3)}) + \nu}. \end{aligned} \tag{9}$$

We present the numerical results of the relationship between $\phi(\rho)$ and ρ in Supplementary Material S2.1. Given the relationship between β and $\mathcal{R}_0$, one may derive the relationship between $\mathcal{R}_0$ and $\phi(\rho)$ explicitly.

Results and Discussion

Individual Equilibrium and Travelling Optimum

We first explore how the predicted travel strategies depend on the basic reproduction number ($\mathcal{R}_0$) and the relative risk (r). Many factors, including seasonal (climatic) factors and the evolution of viruses, could affect $\mathcal{R}_0$. Additionally, media coverage of the risk and relevant educational programs [44, 45, 46, 47, 48, 49] could influence visitors' perception of the risk, thus changing r_1 and r (Eqn. 5). During an ongoing epidemic ($\alpha = 1$), we find that both r and $\mathcal{R}_0$ significantly influence the individual equilibrium p^* and the group optimum ρ^* (Fig. 2). (The values of the other parameters are fixed and listed in Table 1, and small variations in their values do not dramatically change the trends of these relationships.) We observe that both the individual and population optima have the same qualitative relationship with $\mathcal{R}_0$ and r: both optima are monotonically decreasing functions of $\mathcal{R}_0$ and monotonically increasing functions of r. This behaviour is expected, since an increasing transmissibility should reduce both the individual incentive to travel and the group optimal rate of travelling, while a decline in the relative risk of travelling should encourage travel, both individually and as a group. More surprisingly, the sudden transition of the individual optimum from 0 to 1 (as shown in panel a) is steeper than that of the population optimum (as shown in panel b).

(a) individual equilibrium, p^* (b) travelling optimum, ρ^*

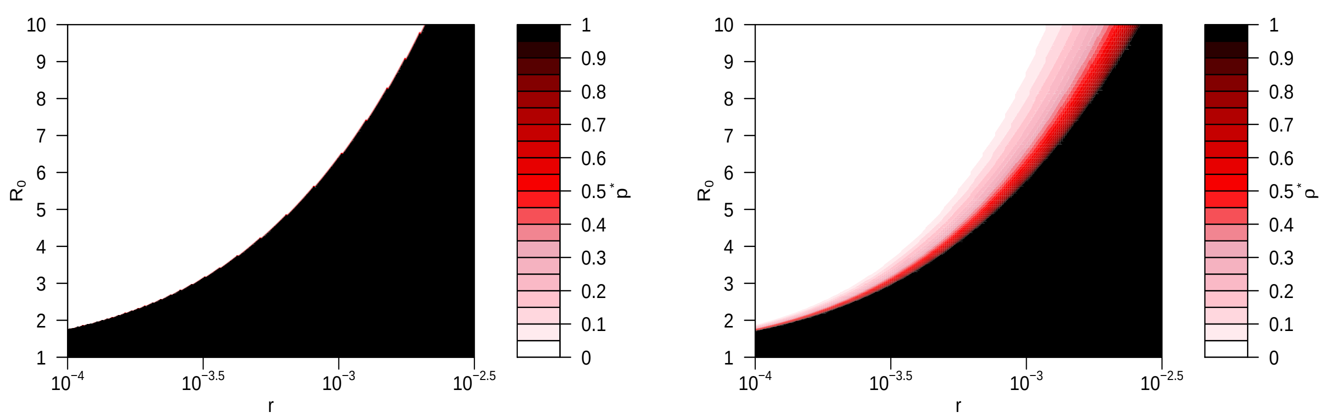

Figure 2: Individual and population optima as functions of the basic reproduction number $\mathcal{R}_0$ and the relative risk r during an epidemic ($\alpha = 1$). Panel (**a**) shows the Nash equilibrium proportion of travellers p^*; panel (**b**) shows the group optimal proportion of travelllers ρ^*, with colour codes to indicate magnitude. The range of $\mathcal{R}_0$ and the values of the other parameters are listed in Table 1.

To further explore the relationship between the individual and group optimum, we study their difference:

$$\Delta\rho = \rho^* - p^* \tag{10}$$

More details are given in Supplementary Material S2. A plot of $\Delta\rho$ versus the population optimum ρ^* and the individual equilibrium p^* during an ongoing epidemic ($\alpha = 1$) show that they agree perfectly for most of the parameter space (Fig. 3). For most of the parameter region, $\rho^* = p^* = 0$

or 1 (i.e., the white area in Fig. 3). These two situations can occur when both the disease risk (reflected by $\mathcal{R}_0$) and perceived risk are (1) either considerably high, i.e., $\rho^* = p^* = 0$, in which case no one intends to travel and complete border entrance restrictions are implemented, or (2) considerably low, i.e., $\rho^* = p^* = 1$, in which case all individuals intend to travel and border entrance is completely unrestricted. Variations in the values of the other parameters do not change the trends of these relationships (Table 1).

However, despite the broad agreement across the parameter plane, the region where ρ^* and p^* are discrepant reveals interesting findings. During an epidemic, most locations are expected to receive fewer visitors (with limited visitor entrance) than usual when there is no epidemic. But the model predicts parameter regimes where the group optimal solution requires a higher volume of travel than what is individually optimal: in the blue region of the parameter plane, $\Delta\rho > 0$, meaning $p^* < \rho^*$ (Fig. 3a). In this regime, the health authority would wish to encourage more travel than actually occurs. However, if either the disease risk $\mathcal{R}_0$ or the perceived payoff of disease risk r_1 decline even slightly (for instance, due to seasonal factors and/or changing media coverage) the situation is reversed, and the discrepancy in interests $\Delta\rho$ could change from $\Delta\rho > 0$ to $\Delta\rho < 0$ (red region in Fig. 3a). When $\Delta\rho < 0$, a health authority restriction on visitors is desired and only $\frac{\rho^*}{p^*}$ of the visitors should be allowed to enter in order to achieve the population optimum ρ^*. In summary, Fig. 3 shows a surprising contrast to many game theoretical models comparing individual and group optimal outcomes: in large parts of the parameter space, there is no discrepancy. However, when a discrepancy does emerge, it can emerge very quickly with small changes in parameter values, and moreover, the individual optimal travel rate could exceed the group optimal rate, or *vice versa*.

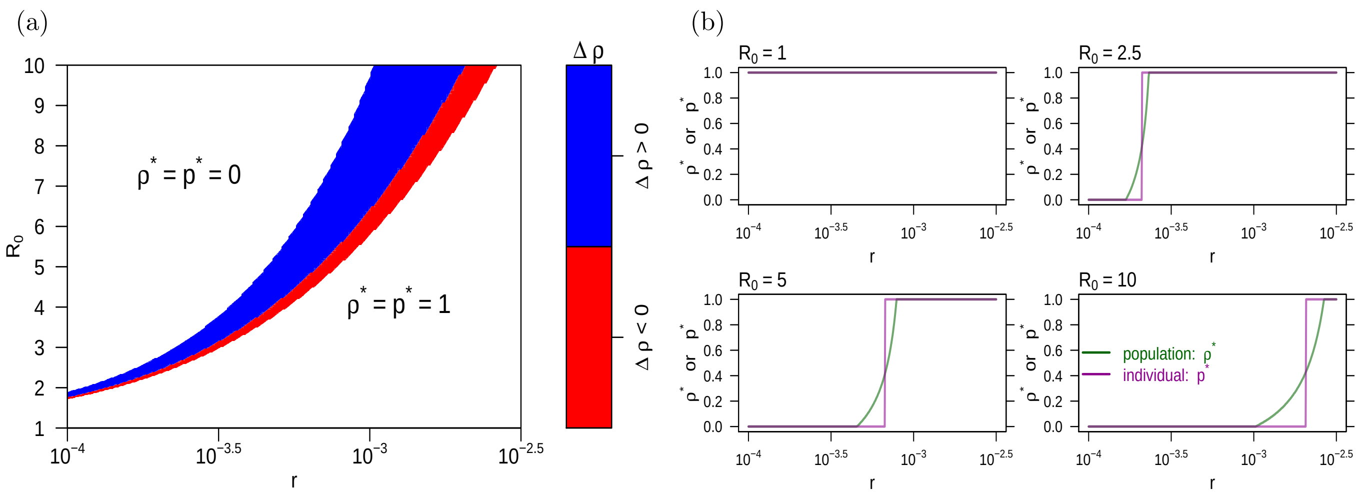

Figure 3: Discrepancy between individual and population optima as a function of the basic reproduction number $\mathcal{R}_0$ and relative risk r, during an epidemic (i.e., $\alpha = 1$). Panel **a** shows the relationship among r (Eqn. 5), $\mathcal{R}_0$ and $\Delta\rho$ (Eqn. 10); and panel **b** shows the relationship between r and $\Delta\rho$ for $\mathcal{R}_0 = 1.0, 2.5, 5.0, 10.0$. In panel **a**, the colour code quantifies $\Delta\rho$. The white area represents $\Delta\rho = 0$ under the two cases that $\rho^* = p^* = 0$ or 1. In panel **b**, ρ^* is in green, and p^* is in purple. In both panels, the range of $\mathcal{R}_0$ and the values of the other parameters are listed in Table 1. Please refer to the electronic version for the figure with color.

Example of the 2003 SARS Outbreak in Beijing

The epidemic patterns predicted by our model under a manipulation of the group optimal strategy ρ are qualitatively similar to the epidemic curve during the 2003 SARS outbreak in Beijing, China, resulting from the timing of certain travel-related events during the outbreak. Fig. 4a (adapted from Ref. [11]) shows weekly reported cases in Beijing during the outbreak. Data are available from the electronic supplementary material. The time point when knowledge of the epidemic was first made public, e.g., "*SARS made reportable (Apr 10)*" in Fig. 1 of Ref. [11], refers to the date of news press [52]. The time point of the official start of restrictions on travel refers to the events "*outbreak announced publicly by government (Apr 20)*" and "*fever check at airport begin (Apr 22)*" in Fig. 1 of Ref. [11]. We note that these two events resulted in almost no one travelling to Beijing, i.e., $\rho = 0$, until the end of the SARS epidemic [53].

We also note that, although the Beijing SARS outbreak was initially sparked by travellers, the proportion of cases in Beijing caused by travellers over the entire outbreak is thought to be small, especially after fever screening began [54]. Also, the United States Centers for Disease Control suggests that travellers to SARS-affected destinations take precautions to avoid infection, suggesting a nontrivial infection risk for travellers [55]. The latter two features of the Beijing SARS outbreak are consistent with our model assumptions.

Fig. 4b shows a model-simulated epidemic curve that largely matches the observed epidemic curve. To generate this curve we focus on changes in $\mathcal{R}_0$ (disease transmissibility) and ρ (proportion of players adopting the "travel" strategy). We decrease ρ from 0.5 to 0.25 at the time indicated by the blue dashed vertical line in Fig. 4b. This decrease is associated with the start of public awareness of the SARS risk in Beijing after it was revealed to the public [52]. Similarly, the decrease in $\mathcal{R}_0$ from 2.5 to 1.75 as also indicated by the blue dashed vertical line would correspond to an accompanying reduction of the effective contact rate due to the onset of public awareness of SARS. (The effective contact rate is defined as the product of the contact rate and transmission probability per contact. It is believed, and is modelled, to be negatively, or at least non-positively, related to reported disease incidence [34, 45, 46, 47, 56].) The time lag, i.e., the gap between the pairs of vertical solid and dashed lines of the same colour in Fig. 4, is fixed at three days due to the mixed effects of the incubation period (or the latent period) of SARS infection and the delay of human reaction to the outbreak. The model simulation largely captures the observed SARS epidemic between March and May 2003, as shown in Fig. 4a-b and Fig. 8 of [57].

The model-predicted outcome of an earlier implementation of travel restrictions (see blue and red dashed lines in Fig. 4b) are obtained by fixing the combinations of $\mathcal{R}_0$ and N_1, and setting $\rho = 0$ (i.e., nobody is able or willing to enter due either to travel restrictions or cautious behaviour due to SARS risk). We found that the earlier the travel restrictions are implemented, the more effectively the disease outbreak level is reduced. By contrast, an uncontrolled and sudden increase in the proportion of visitors (e.g., increasing ρ from 0.5 to 0.75) could yield a larger outbreak, as indicated by the gold dashed lines in Fig. 4b.

We note that our objective in Figure 4 is to convey how the model framework applies during an unfolding epidemic where travel restrictions are put in place partway through the epidemic. Hence, although the starting value of $\mathcal{R}_0$ is epidemiologically plausible for SARS [58, 59], the parameters were chosen for convenience rather than being fitted systematically. However, slight changes in the parameter values away from this parameter regime do not change the outcomes.

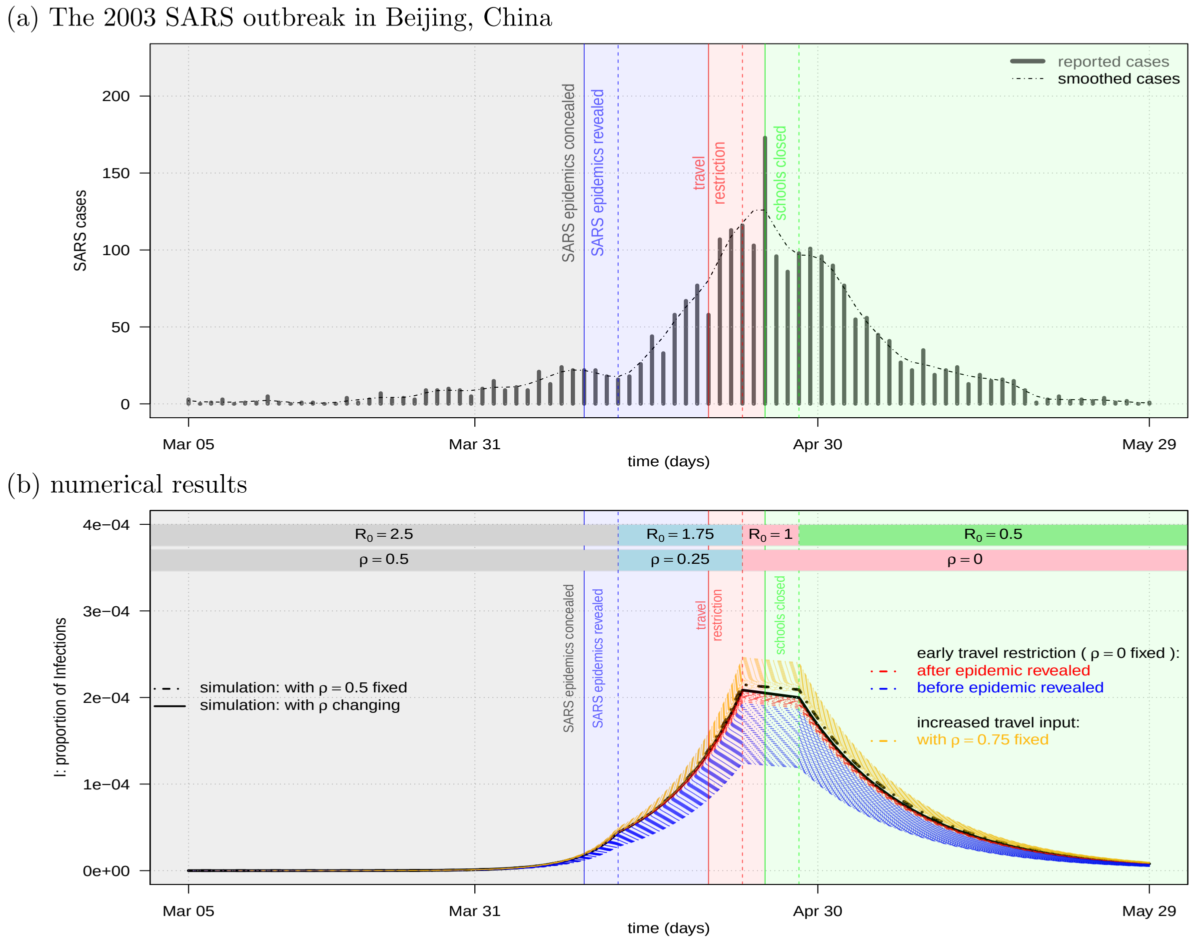

Figure 4: The 2003 SARS outbreak in Beijing, China. Panel **a** shows the reported cases during 2003 SARS outbreak in Beijing, China (adapted from Ref. [11]); and panel **b** shows the numerical results of the epidemic model (see Eqns. 7). In both panels, the vertical lines represent the starting points of events, and the vertical dashed lines represent the time points with lag of three days. In panel **a**, the SARS epidemic and government intervention are given on timeline from Mar 05 to May 29, 2003. The back dashed line is the time series smoothed by using the *LOESS* function (`R` version 3.4.3). In panel **b**, the initial states are set as $[S(0), I(0), S_1(0), I_1(0)] = [(1 - K_1), 0, (K_1 - 1 \times 10^{-8}), 1 \times 10^{-8}]$, with $\mathcal{R}_0 = 2.5$, $N_1 = 15\%$ and $\rho = 0.5$ (see grey parts of the bars on the top). The blue and red dashed lines are the simulations under "what if" scenarios in which travel restriction policies were implemented earlier. The black and gold dashed lines are under "what if" scenarios in which travel restriction (or reduction) failed and travel input suddenly increased respectively. The values of the other parameters are assumed to be the same as those in Table 1, and the changes in parameters are marked at the top of the panel. Note that the timelines are the same in panels **a** and **b**.

Also, additional numerical results for wider parameter variations in Supplementary Material S4 show the range of possible dynamics exhibited by the model.

Additional Sensitivity Analysis

The sensitivity analysis of the baseline model (Supplementary Material S5) shows that the results are most sensitive to the relative risk (r), basic reproduction number ($\mathcal{R}_0$), and the rate at which individuals leave the destination (ν). More detailed discussion of the influence of these model parameters on model predictions are given in Supplementary Material S7.

In the baseline model, for simplicity, we assume that visitors do not bring infection back to their home country. To amend this shortcoming, we introduce an additional probabilistic case importation risk level into an extended model (see parameters in Table 1). Under this extension, our main results are unchanged. Please refer to Supplementary Material S9 for a detailed discussion. We also included pre-existing immunity among visitors in an extended model, and also found that our main results were unchanged. A detailed discussion can be found in Supplementary Material S8.

Model Limitations and Future Research

In this subsection, we discuss possible model extensions and some limitations. In the baseline model, we assume individuals have accurate knowledge of the real basic reproduction number $\mathcal{R}_0$. However, an imbalance between the perceived and actual $\mathcal{R}_0$ could exist [60, 61, 62]. We denote $\widetilde{\mathcal{R}_0}$ as the perceived $\mathcal{R}_0$. We expect the perceived $\widetilde{\mathcal{R}_0}$ to correlate positively with the actual $\mathcal{R}_0$. Thus, we assume $\widetilde{\mathcal{R}_0}(\mathcal{R}_0)$ is a nondecreasing function of $\mathcal{R}_0$. Given the perceived disease risk $\widetilde{\mathcal{R}_0}$, the payoff of the disease risk $r_1\left(\widetilde{\mathcal{R}_0}\right)$, i.e., r_1 as a function of $\widetilde{\mathcal{R}_0}$ given in Eqn. 2, is a nondecreasing function of $\widetilde{\mathcal{R}_0}$ and a nondecreasing function of $\mathcal{R}_0$. One of the simplest forms of $r_1\left(\widetilde{\mathcal{R}_0}\right)$ is $r_1 \propto \widetilde{\mathcal{R}_0}$ with a positive scalar. Future research should explore the impact of such a difference between $\mathcal{R}_0$ and $\widetilde{\mathcal{R}_0}$.

In addition, travelling players may not always be informed about outbreak events in a timely manner. Thus, a time delay between $\mathcal{R}_0$ and $\widetilde{\mathcal{R}_0}$ could exist. We denote $\widetilde{\mathcal{R}_0}(t;\tau) = \widetilde{\mathcal{R}_0}(\mathcal{R}_0(t-\tau))$, where $\tau \geqslant 0$ is the time lag between the occurrence of infection risk and the perception of infection risk. If we set $\tau = 0$ for all t by assuming humans receive accurate knowledge of a risk when it emerges, we have $\lim_{\tau\to 0^+} \widetilde{\mathcal{R}_0}(t;\tau) = \widetilde{\mathcal{R}_0}(\mathcal{R}_0(t))$. In this work, we consider a limiting case of $\tau = 0$. In reality, this assumption can be relaxed, and a reasonable estimate can be used. The value of τ depends on the impacts of the risk and the efficiency of the media and relevant programs (e.g., news press coverage [22, 34, 45, 47], education programs [22, 49, 63], communication effectiveness in social networks [48, 49, 64, 65, 66, 67] and pre-existing public health awareness [14, 48, 65]).

In this work, we assumed the same information availability and the same strategic response for the entire visitor population (see Eqns. 2 and 3). However, different groups of people could have different risk perceptions or risk preferences, hence the payoffs could differ between individuals. This has been demonstrated in previous game theoretical models to lead to different equilibria and optima regarding the human response to epidemics [26, 68]. Consider the situation where $E_1 = E_0$ (see Eqns. 2 and 3). In this case, some individuals may prefer the travelling strategy (i.e., risk-seeking preference), while others may prefer the non-travelling strategy (i.e., risk-averse preference).

Future models including a heterogeneous population could improve the realism of the model and help test the robustness of our predictions. One way this could be done is by allowing the disease natural history and economic parameters to vary between individuals (as noted in the foregoing paragraph), to reflect varying health conditions and socio-economic status. Another way to account for heterogeneity at a larger scale is to allow for a patchy environment [1] where different sub-populations are subject to different conditions. Under such circumstances, we expect that the boundaries in Fig. 2) would probably become less sharp, although it is not clear *a priori* how large the effect would be. We expect that most forms of heterogeneity would not change our finding that the individual and group optima tend to agree in this kind of game theoretical framework, although the regime shifts implied by Fig. 2) would probably be less dramatic if heterogeneity were included.

Conclusions

Many game theoretical studies of closed socio-epidemiological systems find a significant discrepancy between individual and group (Pareto) optima in a broad range of economic and epidemiological parameters. In this work, we studied an open socio-ecological system in which visitors decide whether to travel to a location with an ongoing outbreak. Surprisingly, we found perfect agreement between the individual and group optimal strategies for broad ranges of parameter values. When a disagreement between the individual and group optimal strategies occurs, the discrepancy was very large and highly sensitive to small changes in disease transmissibility and visitor costs/benefits. For instance, if disease transmissibility increases by even a small amount, the uncontrolled incoming visitors are capable of causing an unexpected outbreak. This suggests that a discrepancy between the individual and group optima could emerge suddenly in real-world settings, provided that slight changes in economic and epidemiological factors (parameters) occur. However, timely implementation of travel restrictions by health authorities may effectively prevent large-scale outbreaks.

Data Accessibility

The 2003 SARS cases time series in Beijing are obtained from Ref. [11] and are available from the electronic supplementary material.

Author Contributions

All authors conceived and carried out the study, drafted the manuscript and gave final approval for publication.

Competing interests

We have no competing interests.

Acknowledgements

We are grateful to reviewers for valuable comments.

Ethics

Ethical issue is not applicable.

Funding

S.Z. and D.H. were supported by an Early Career Scheme grant from the Hong Kong Research Grant Council (PolyU 251001/14M).

References

[1] Wang W, Zhao XQ. An epidemic model in a patchy environment. Mathematical biosciences. 2004;190(1):97-112.

[2] Brauer F, van den Driessche P. Models for transmission of disease with immigration of infectives. Mathematical Biosciences. 2001;171(2):143-54.

[3] Cui JA, Takeuchi Y, Saito Y. Spreading disease with transport-related infection. Journal of theoretical biology. 2006;239(3):376-90.

[4] Epstein JM, Goedecke DM, Yu F, Morris RJ, Wagener DK, Bobashev GV. Controlling pandemic flu: the value of international air travel restrictions. PloS one. 2007;2(5):e401.

[5] He D, Chiu AP, Lin Q, Cowling BJ. Differences in the seasonality of MERS-CoV and influenza in the Middle East. International journal of infectious diseases: IJID: official publication of the International Society for Infectious Diseases. 2015;40:15.

[6] Ruan S, Wang W, Levin SA. The effect of global travel on the spread of SARS. Mathematical Biosciences and Engineering. 2006;3(1):205.

[7] Bauch CT, Lloyd-Smith JO, Coffee MP, Galvani AP. Dynamically modeling SARS and other newly emerging respiratory illnesses: past, present, and future. Epidemiology. 2005;16(6):791-801.

[8] Lipsitch M, Cohen T, Cooper B, Robins JM, Ma S, James L, Gopalakrishna G, Chew SK, Tan CC, Samore MH, Fisman D. Transmission dynamics and control of severe acute respiratory syndrome. Science. 2003;300(5627):1966-70.

[9] Gumel AB, Ruan S, Day T, Watmough J, Brauer F, Van den Driessche P, Gabrielson D, Bowman C, Alexander ME, Ardal S, Wu J. Modelling strategies for controlling SARS outbreaks. Proceedings of the Royal Society of London B: Biological Sciences. 2004;271(1554):2223-32.

[10] Chowell G, Fenimore PW, Castillo-Garsow MA, Castillo-Chavez C. SARS outbreaks in Ontario, Hong Kong and Singapore: the role of diagnosis and isolation as a control mechanism. Journal of theoretical biology. 2003;224(1):1-8.

[11] Pang X, Zhu Z, Xu F, Guo J, Gong X, Liu D, Liu Z, Chin DP, Feikin DR. Evaluation of control measures implemented in the severe acute respiratory syndrome outbreak in Beijing, 2003. Jama. 2003;290(24):3215-21.

[12] Grais RF, Ellis JH, Glass GE. Assessing the impact of airline travel on the geographic spread of pandemic influenza. European journal of epidemiology. 2003;18(11):1065-72.

[13] Poletti P, Ajelli M, Merler S. The effect of risk perception on the 2009 H1N1 pandemic influenza dynamics. PLoS One. 2011;6(2):e16460.

[14] Cornforth DM, Reluga TC, Shim E, Bauch CT, Galvani AP, Meyers LA. Erratic flu vaccination emerges from short-sighted behavior in contact networks. PLoS Computational Biology. 2011;7(1):e1001062.

[15] Funk S, Knight GM, Jansen VA. Ebola: the power of behaviour change. Nature. 2014;515(7528):492-.

[16] Jack A. Why the panic? South Korea's MERS response questioned. BMJ: British Medical Journal (Online). 2015;350.

[17] He D, Chiu AP, Lin Q, Yu D. Spatio-temporal patterns of proportions of influenza B cases. Scientific reports. 2017;7:40085.

[18] Hollingsworth TD, Ferguson NM, Anderson RM. Will travel restrictions control the international spread of pandemic influenza? Nature medicine. 2006;12(5):497-9.

[19] Apolloni A, Poletto C, Ramasco JJ, Jensen P, Colizza V. Metapopulation epidemic models with heterogeneous mixing and travel behaviour. Theoretical Biology and Medical Modelling. 2014;11(1):3.

[20] Von Neumann J, Morgenstern O. Theory of games and economic behavior. Princeton university press; 2007.

[21] Bauch CT, Galvani AP, Earn DJ. Group interest versus self-interest in smallpox vaccination policy. Proceedings of the National Academy of Sciences of the United States of America. 2003;100(18):10564-7.

[22] Bauch CT, Earn DJ. Vaccination and the theory of games. Proceedings of the National Academy of Sciences of the United States of America. 2004;101(36):13391-4.

[23] Fine PE, Clarkson JA. Individual versus public priorities in the determination of optimal vaccination policies. American journal of epidemiology. 1986;124(6):1012-20.

[24] Chen FH. A susceptible-infected epidemic model with voluntary vaccinations. Journal of mathematical biology. 2006;53(2):253-72.

[25] Codeco CT, Luz PM, Coelho F, Galvani AP, Struchiner C. Vaccinating in disease-free regions: a vaccine model with application to yellow fever. Journal of The Royal Society Interface. 2007;4(17):1119-25.

[26] Reluga TC, Bauch CT, Galvani AP. Evolving public perceptions and stability in vaccine uptake. Mathematical biosciences. 2006;204(2):185-98.

[27] Wang Z, Andrews MA, Wu ZX, Wang L, Bauch CT. Coupled disease!Vbehavior dynamics on complex networks: A review. Physics of life reviews. 2015;15:1-29.

[28] Bauch C, d'Onofrio A, Manfredi P. Behavioral epidemiology of infectious diseases: an overview. In Modeling the Interplay Between Human Behavior and the Spread of Infectious Diseases. Springer New York. 2013;(pp. 1-19).

[29] Molina C, Earn DJD. Game theory of pre-emptive vaccination before bioterrorism or accidental release of smallpox. Journal of The Royal Society Interface. 2015;12(107):20141387.

[30] Funk S, Salathe M, Jansen VA. Modelling the influence of human behaviour on the spread of infectious diseases: a review. Journal of the Royal Society Interface. 2010:rsif20100142.

[31] Wang Z, Bauch CT, Bhattacharyya S, d'Onofrio A, Manfredi P, Perc M, Perra N, Salathe M, Zhao D. Statistical physics of vaccination. Physics Reports. 2016;664:1-13.

[32] Allen LJ, Brauer F, van den Driessche P, Wu J. Mathematical epidemiology. Berlin: Springer; 2008.

[33] Keeling MJ, Rohani P. Modeling infectious diseases in humans and animals. Princeton University Press; 2008.

[34] Gao D, Ruan S. An SIS patch model with variable transmission coefficients. Mathematical biosciences. 2011;232(2):110-5.

[35] Wang W, Mulone G. Threshold of disease transmission in a patch environment. Journal of Mathematical Analysis and Applications. 2003;285(1):321-35.

[36] Germann TC, Kadau K, Longini IM, Macken CA. Mitigation strategies for pandemic influenza in the United States. Proc Natl Acad Sci U S A. 2006;103(15):5935-40.

[37] Mills CE, Robins JM, Lipsitch M. Transmissibility of 1918 pandemic influenza. Nature. 2004;432(7019):904-6.

[38] White LF, Wallinga J, Finelli L, Reed C, Riley S, Lipsitch M, *et al.* Estimation of the reproductive number and the serial interval in early phase of the 2009 influenza A/H1N1 pandemic in the USA. Influenza Other Respir Viruses. 2009;3:267-76.

[39] Lessler J, Reich NG, Cummings DA. Outbreak of 2009 pandemic influenza A (H1N1) at a New York City school. N Engl J Med. 2009;361(27):2628-36.

[40] Centers for Disease Control and Prevention (CDC), webpage of "Key Facts About Influenza (Flu)". `https://www.cdc.gov/flu/keyfacts.htm`. Accessed on Nov 2017.

[41] Riley S, Fraser C, Donnelly CA, Ghani AC, Abu-Raddad LJ, Hedley AJ, Leung GM, Ho LM, Lam TH, Thach TQ, Chau P. Transmission dynamics of the etiological agent of SARS in Hong Kong: impact of public health interventions. Science. 2003;300(5627):1961-6.

[42] van den Driessche P, Watmough J. Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission. Mathematical biosciences. 2002;180(1):29-48.

[43] Ben-Shachar R, Koelle K. Minimal within-host dengue models highlight the specific roles of the immune response in primary and secondary dengue infections. Journal of the Royal Society Interface. 2015;12(103):20140886.

[44] Tchuenche JM, Dube N, Bhunu CP, Smith RJ, Bauch CT. The impact of media coverage on the transmission dynamics of human influenza. BMC Public Health. 2011;11(1):S5.

[45] Cui J, Tao X, Zhu H. An SIS infection model incorporating media coverage. Rocky Mountain J. Math. 2008;38(5):1323-34.

[46] Liu R, Wu J, Zhu H. Media/psychological impact on multiple outbreaks of emerging infectious diseases. Computational and Mathematical Methods in Medicine. 2007;8(3):153-64.

[47] Cui J, Sun Y, Zhu H. The impact of media on the control of infectious diseases. Journal of dynamics and differential equations. 2008;20(1):31-53.

[48] Bauch CT. Imitation dynamics predict vaccinating behaviour. Proceedings of the Royal Society of London B: Biological Sciences. 2005;272(1573):1669-75.

[49] Bauch CT, Bhattacharyya S. Evolutionary game theory and social learning can determine how vaccine scares unfold. PLoS computational biology. 2012;8(4):e1002452.

[50] Tamer O, Thampi V, and Bauch CT. The influence of social norms on the dynamics of vaccinating behaviour for paediatric infectious diseases. Proc. R. Soc. B. 2014; 281:20133172.

[51] Shim E, Chapman, GB, Townsend JP, Galvani AP. The influence of altruism on influenza vaccination decisions. Journal of The Royal Society Interface. 2012; rsif20120115.

[52] News press of the reveal of 2003 SARS epidemic in Beijing, China: http://www.lifeweek.com.cn/2003/0729/5582.shtml (in Chinese) and http://news.eastday.com/epublish/gb/paper148/20030530/class014800003/hwz953568.htm.

[53] The news press of National Tourism Administration (see (in Chinese) http://www.people.com.cn/GB/jingji/1038/1970533.html) and WHO travel advice (see http://www.who.int/csr/sars/archive/2003_04_23/en/).

[54] Wu J, Xu F, Zhou W, Feikin D, Lin CY, He Z et al. Risk factors for SARS among persons without known contact with SARS patients, Beijing, China. Emerging infectious diseases. 2004; 10(2):210.

[55] US Centers for Disease Control. Guidance for Persons Traveling to Areas Where SARS Cases Have Been Reported. https://www.cdc.gov/sars/travel/advice.html

[56] Yu D, Lin Q, Chiu AP, He D. Effects of reactive social distancing on the 1918 influenza pandemic. PLoS One. 2017;12(7): e0180545. https://doi.org/10.1371/journal.pone.0180545

[57] Wang W, Ruan S. Simulating the SARS outbreak in Beijing with limited data. Journal of theoretical biology. 2004;227(3):369-79.

[58] Wallinga J, Teunis P. Different epidemic curves for severe acute respiratory syndrome reveal similar impacts of control measures. American Journal of epidemiology. 2004;160(6):509-16.

[59] Bauch CT, Lloyd-Smith JO, Coffee MP, Galvani AP. Dynamically modeling SARS and other newly emerging respiratory illnesses: past, present, and future. Epidemiology. 2005;16(6):197-801.

[60] Stratton K, Gable A, Shetty P, McCormick M, Institute of Medicine (US) Immunization Safety Review Committee. Immunization safety review: measles-mumps-rubella vaccine and autism. Washington, DC: The National Academies Press. 2004.

[61] Basu S, Chapman GB, Galvani AP. Integrating epidemiology, psychology, and economics to achieve HPV vaccination targets. Proceedings of the National Academy of Sciences. 2008;105(48):19018-23.

[62] Shim E, Chapman GB, Townsend JP, Galvani AP. The influence of altruism on influenza vaccination decisions. Journal of The Royal Society Interface. 2012:rsif20120115.

[63] Mbah ML, Liu J, Bauch CT, Tekel YI, Medlock J, Meyers LA, Galvani AP. The impact of imitation on vaccination behavior in social contact networks. PLoS computational biology. 2012;8(4):e1002469.

[64] Perisic A, Bauch CT. Social contact networks and disease eradicability under voluntary vaccination. PLoS computational biology. 2009;5(2):e1000280.

[65] Funk S, Gilad E, Watkins C, Jansen VA. The spread of awareness and its impact on epidemic outbreaks. Proceedings of the National Academy of Sciences. 2009;106(16):6872-7.

[66] Gross T, D'Lima CJ, Blasius B. Epidemic dynamics on an adaptive network. Physical review letters. 2006;96(20):208701.

[67] Shaw LB, Schwartz IB. Fluctuating epidemics on adaptive networks. Physical Review E. 2008;77(6):066101.

[68] Reluga TC. An SIS epidemiology game with two subpopulations. Journal of Biological Dynamics. 2009;3(5):515-31.

Supplementary Information

S1 More Explanation of the Travelling Procedure

S2 Individual Equilibrium and Group Optimum

S2.1 Individual Equilibrium

S2.2 Group Optimum

S3 Simplification of the Epidemic Model: Elimination of R and R_1

S4 Some Numerical Examples

S5 Sensitivity Analysis of Payoffs

S6 Interpretation and Value of Some Model Parameters

S6.1 Rate of visitors moving from outside status to inside status λ

S6.2 Visitor capacity at destination K_1

S6.3 Rate of visitors leaving destination ν

S6.4 Relative risk r

S7 Further Discussion of Model Parameters

S8 Pre-existing immunity among visitors

S9 Risk of visitors bringing the disease back to their home country

S10 Repored SARS cases in Beijing in 2013

Supplementary Information

Shi Zhao[1], Chris T. Bauch[2,*] & Daihai He[1,*]

1 Department of Applied Mathematics, Hong Kong Polytechnic University, Hong Kong

2 Department of Applied Mathematics, University of Waterloo, Canada

***** Correspondence to: `daihai.he@polyu.edu.hk` (D.H.) and `cbauch@uwaterloo.ca` (C.T.B.)

August 20, 2018

Contents

S1 More Explanation of the Travelling Procedure

In this work, the game players are potential visitors who have a plan to visit a travel destination. Potential visitors will end up in two categories: those who take the trip and those who cancel the trip (see path (1) in main text). Any individual of the home country of visitors may become a potential visitor at any time. The number of potential visitors (or N_1 as the ratio in the model, see Table 1 in main text) is mainly dependent on the travelling pattern (or seasonality) of the destination. A potential visitor may decide to travel (i.e., become a "visitor outside") according to his/her knowledge on the disease risk at the very moment the decision being made. Thus, there are three cases regarding to the different travelling decisions.

- A potential visitor decides to travel and successfully completes the trip. Since the trip is short (three days), we assume that the visitor does not change his/her travel decision. Finally, s/he returns to his/her home population after the trip.
- A potential visitor decides to travel but fails to complete it due to travel restriction at the destination. In this case, the visitor returns to his/her home population.
- A potential visitor voluntarily cancels the trip and stays at his/her home population.

Therefore, in any case, the decision making process of the proposed travelling game follows the sequential game scheme (i.e., the decision is "renewable" for every participant in this game). We note that local "travel restriction" only has its effects on these potential visitors who decide to travel; and the potential visitors is mainly influenced by the travelling pattern to the destination.

S2 Individual Equilibrium and Group Optimum

S2.1 Individual Equilibrium

We assume that a proportion ε $(0 < \varepsilon < 1)$ of potential visitors will take the trip with a probability p (i.e., playing p strategy) and the rest of potential visitors $(1 - \varepsilon)$ will take the trip with probability q, where $q \neq p$. Then, the overall proportion of visitors $(\bar{\rho})$ who will take the trip among all game players is

$$\bar{\rho} = \varepsilon p + (1 - \varepsilon)q. \tag{S1}$$

Therefore, the payoff to individuals playing p-strategy and q-strategy are $E(p, \bar{\rho})$ and $E(q, \bar{\rho})$, respectively. The payoff gain (or loss if negative) of an individual playing p strategy against q strategy is the difference of two payoff functions,

$$\Delta E = E(p, \bar{\rho}) - E(q, \bar{\rho}) = (p - q)\left[r - \alpha\phi(\bar{\rho})\right]. \tag{S2}$$

where the parameters have the same meaning as in the main text.

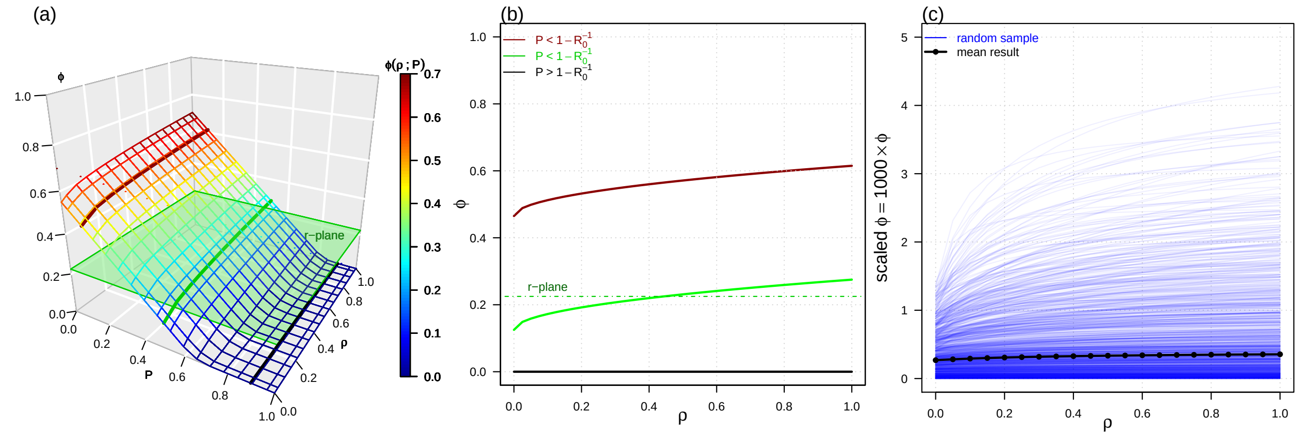

Figure S1: Schematic diagram of Nash equilibria under three "existence" situations (panel (a) and (b)) and numerical results of the relation between $\phi(\rho)$ and ρ (panel (c)). In order to have a clear demonstration of three kinds of Nash equilibria, panel **(a)** and **(b)** show the trends of $\phi(\rho; P)$ against ρ and P. Panel **(c)** shows the relation between scaled $\phi(\rho)$ and ρ. The scaled $\phi(\rho) = 1000 \times \phi(\rho)$. In panel **(c)**, the transparently blue line are from 1,000 random samples with parameter sets, and the black dotted line is the result with fixed parameter values. The parameters' values and ranges can be found in Table 1.

Existence of Nash Equilibria The probability of a visitor becomes infected during the trip ($0 < \phi(\rho) < 1$) must increase strictly (which is in line with [**?**], as explained in Epidemic Model section) when a proportion ρ of game players choose the travelling strategy (see Fig. S1). Hence, when P is fixed, the minimum of $\phi(\rho)$ occurs at $\rho = 0$ and the maximum of $\phi(\rho)$ occurs at $\rho = 1$. Here, we show the existence of the unique Nash equilibria by achieving $\Delta E > 0$ in Eqn. (S2) under three situations.

- If $\alpha \cdot \min\{\phi(\rho)\} = \alpha\phi(\rho = 0) \geqslant r$, $\alpha\phi(\rho) > r$ for all $0 < \rho < 1$, so for any $0 < \varepsilon < 1$ of Eqn. (S1), $\Delta E > 0$ for any $q \neq p$ if and only if $p = 0$ (such that $p - q < 0$ for all $0 < q < 1$), thus, $p^* = 0$ is the unique Nash equilibrium.

- If $\alpha \cdot \max\{\phi(\rho)\} = \alpha\phi(\rho = 1) \leqslant r$, $\alpha\phi(\rho) < r$ for all $0 < \rho < 1$, so for any $0 < \varepsilon < 1$ of Eqn. (S1), $\Delta E > 0$ for any $q \neq p$ if and only if $p = 1$ (such that $p - q > 0$ for all $0 < q < 1$), thus, $p^* = 1$ is the unique Nash equilibrium.

- If $\alpha \cdot \max\{\phi(\rho)\} = \alpha\phi(\rho = 1) > r > \alpha\phi(\rho = 0) = \alpha \cdot \min\{\phi(\rho)\}$, there exist one and only one p^* such that $\alpha\phi(\rho = p^*) = r$. For all $q < p$, we have $\bar{\rho} < p$ (according to Eqn. (S1)) for any $0 < \varepsilon < 1$ and, similarly, for all $q > p$, we have $\bar{\rho} > p$ for any $0 < \varepsilon < 1$. Hence, for $\alpha\phi(\rho = 1) > r > \alpha\phi(\rho = 0)$, we always have $\Delta E > 0$ for all $q \neq p$ if and only if $p = p^*$, so p^* is the unique Nash equilibrium such that $\alpha\phi(p^*) = r$.

These different situations of the relationship between $\alpha\phi(\rho)$ and r are due to different values of the pre-existing immunity level (i.e., P, Fig. S1) and different values of parameters (Table 1).

Convergent Stability Follow the previous work [3], let p be closer to p^* than q (i.e., the unique Nash equilibrium of Eqn. (S2)), which means $q < p \leqslant p^*$ or $q > p \geqslant p^*$ (note that p is not necessarily equal to p^*). Given $\phi(\rho)$ increases with respect to ρ, if $q < p \leqslant p^*$, $(r - \alpha\phi(\bar{\rho})) > 0$ for all ε in Eqn. (S1), we have $\Delta E > 0$. Similarly, we can also have $\Delta E > 0$ if $q > p \geqslant p^*$ as desired. Therefore, the Nash equilibria in all of the three scenarios are convergently stable.

S2.2 Group Optimum

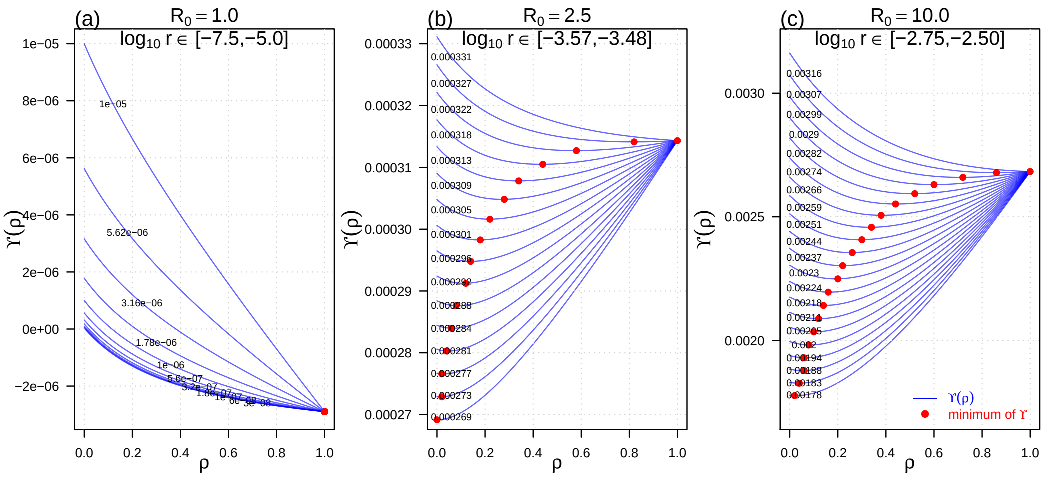

Figure S2: The optimal proportion of travelling-players becoming visitors (i.e., ρ^* corresponding to Eqn. (S4)) during epidemic (i.e., $\alpha = 1$). Panel (a)-(c) corresponds to $\mathcal{R}_0 = 1.0, 2.5$ and 10.0 respectively. Blue lines are $\Upsilon(\rho)$ in Eqn. (S4) with respect to different values of r and red dots are the minima (when $\rho = \rho^*$) of $\Upsilon(\rho)$, of which $\rho \in [0, 1]$. The values of r are shown on each blue line. The range of $\mathcal{R}_0$ and values of other parameters are on Table 1.

For all potential visitors, we aim to minimize the overall cost (negative payoff) of all players, which also appears to be the goal of governmental control. We further ignore the possibility that infected visitors bring the disease back to their home population. We can express the expected cost in term of ρ (i.e., the overall proportion of all players who choose to travel),

$$\Upsilon(\rho) = N_1 \cdot [\rho\alpha \cdot \phi(\rho) \cdot r_1 + (1 - \rho) r_0] \,. \tag{S3}$$

Here, N_1 is the ratio of total number of players to the total local population capacity (i.e., sum of local visitors capacity and number of population). The other terms have the same meaning as in Eqn. (S2). We further scale $\Upsilon(\rho)$ by eliminating N_1 (because N_1 can be fixed as a constant) and one risk term (replacing r_0 and r_1 by $r = \frac{r_0}{r_1}$ [2, 6]. Thus, the (scaled) cost of potential visitors is

$$\Upsilon(\rho) = \rho\alpha \cdot \phi(\rho) + (1 - \rho) r, \tag{S4}$$

where all terms have the same meaning as in Eqn. (S2). The optimal travelling proportion is the optimal ratio of successful visitors over all game players, which is denoted by ρ^*. ρ^* can be obtained by minimizing $\Upsilon(\rho)$ (see Fig S2 as numerical examples).

S3 Simplification of the Epidemic Model: Elimination of R and R_1

Based on the framework of the standard SIR compartmental model (see "Epidemic Model" section in the main text), we write the original epidemic model as:

$$\begin{cases} S' = \mu \cdot (1 - K_1 - S) - \beta S \cdot (I + I_1), \\ S_1' = f_\rho \cdot \left(1 - \dfrac{S_1 + I_1 + R_1}{K_1}\right) - \beta S_1 \cdot (I + I_1) - \nu S_1, \\ I' = \beta S \cdot (I + I_1) - (\gamma + \mu) I, \\ I_1' = \beta S_1 \cdot (I + I_1) - (\gamma + \nu) I_1, \\ R' = \gamma I - \mu R, \\ R_1' = \gamma I_1 - \nu R_1. \end{cases} \tag{S5}$$

Here, $f_\rho = f(\rho) = \rho \lambda N_1$ represents the rate of incoming visitors. K_1 is the ratio of maximum capacity of visitors to the total population capacity. K_1 controls the upper bound of the magnitude of visitors in the model system (thus, generally, K_1 is fixed) S6.2. N_1 has the same meaning as in Table 1 and Eqn. (S3). N_1 is the ratio of total number of potential visitors (i.e., travelling-players) to the total population capacity (i.e., the sum of maximum visitors capacity and the size of local population, see Table 1, $(S + I + R + K_1)$ in model (S5)) For simplicity, we fix N_1 in this work. Model parameters are summarized in Table 1 in the main text.

Most visitors stay inside border (i.e., in the destination) for a considerably short period (three days, ν^{-1} in Table 1 and S6.3). Since $(S + I + R) + K_1 \equiv 1$ (i.e., the total population capacity, is scaled to unity) and $S_1 + I_1 + R_1 \leqslant K_1 < 1$, we have $(S + I + R) + (S_1 + I_1 + R_1) \leqslant 1$.

Under the quasi-steady-state assumption, which is widely adopted in within-host modelling studies [7, 5], we replace the term $\frac{S_1 + I_1 + R_1}{K_1}$ (in model (S5)) by $\frac{S_1 + \left(1 + \frac{\gamma}{\nu}\right) I_1}{K_1}$ (by forcing $R_1' = 0$) in order to eliminate equation of R_1. This approximation can be interpreted as that all R_1 come from I_1 and only $\frac{\gamma}{\gamma + \nu}$ of I_1 could transit to R_1 at any time (other part of I_1 simply leaving the system at rate ν). Thus, $R_1 \leqslant \frac{\gamma}{\gamma + \nu} I_1 \leqslant \frac{\gamma}{\nu} I_1$ (both γ and ν are positive), and then, $S_1 + I_1 + R_1 \leqslant S_1 + \left(1 + \frac{\gamma}{\nu}\right) I_1$. Since infected (I_1) visitors will quickly join R_1 class at the rate γ and the proportion of recovered visitors are relatively small, term $S_1 + I_1 + R_1$ is very close to $S_1 + \left(1 + \frac{\gamma}{\nu}\right) I_1$. Note that $\frac{\gamma}{\nu} I_1$ is simply the upper bound of R_1, and, after all, the effects of both I_1 and R_1 are little (compared with S_1) regarding to the visitors input.

After eliminating R' and R_1', we reformulate the epidemic model as,

$$\begin{cases} S' = \mu \cdot (1 - K_1 - S) - \beta S \cdot (I + I_1), \\ I' = \beta S \cdot (I + I_1) - (\gamma + \mu) I, \\ S_1' = f_\rho \cdot \left[1 - \dfrac{S_1 + \left(1 + \frac{\gamma}{\nu}\right) I_1}{K_1}\right] - \beta S_1 \cdot (I + I_1) - \nu S_1, \\ I_1' = \beta S_1 \cdot (I + I_1) - (\gamma + \nu) I_1. \end{cases}$$

This version is used in the main text.

For mathematical convenience, we fix $(S + I + R) + K_1 \equiv 1$ (i.e., the population threshold, or the total population capacity, is scaled to unity, 1). We also let $S_1 + (1 + \gamma/\nu)I_1 \leqslant K_1$, thus, $S_1 + I_1 + R_1 \leqslant K_1$ is guaranteed. Therefore, we have $(S + I + R) + (S_1 + I_1 + R_1) < 1$ in our complete model (see S3).

S4 Some Numerical Examples

The epidemics could be amplified by the uncontrolled visitor inflow, even when the basic reproduction number is low. Fig. S3(a) shows an epidemic becoming out of control with $\mathcal{R}_0$ declines (from 2.5 to 2.4) while the incoming visitor restriction fails (red line). The disease outbreaks can be controlled if the incoming visitors are restricted (i.e., by holding $\rho = 0.1$ unchange, see the green line). Since ρ^* is sensitive in a narrow range of $\mathcal{R}_0$ and r (see section "Results of Individual Equilibrium and Travelling Optimum" in main text), ρ^* could have very large change (e.g., from 0.1 to 0.99) with **slight** change on $\mathcal{R}_0$ (e.g., from 2.5 to 2.4 in Fig. S3(a)). The large variation in ρ^* could lead to the discrepancy between ρ^* and p^*. The decline of disease risk ($\mathcal{R}_0$) could avoid this discrepancy (by achieving $p^* = \rho^* = 1$). The increase of disease risk ($\mathcal{R}_0$) might also avoid this discrepancy (by achieving $p^* = \rho^* = 0$).

When the risk of disease (in term of $\mathcal{R}_0$) is higher than the perceived risk (i.e., the perceived risk is low), the local government is suggested to restrict visitor entrance. Otherwise, the actual proportion of the incoming visitor is likely to be greater than the optimal level (ρ^*). Fig. S3(b) shows the epidemic becoming out of control when $\mathcal{R}_0$ slightly rises and incoming visitors are not controlled (green line). The disease outbreak can be controlled by visitors entrance restriction (red and purple lines).

Fig. S3(c) shows the similar trend as the early stage of SARS epidemic (in Jan - Feb, 2003). The rapid increasing could be mainly due to the increased visitors during Chinese new year (see Fig. 2(a) of Ref. [10]). Namely the increase of visitors could lead to a disease outbreak.

S5 Sensitivity Analysis of Payoffs

Partial rank correlation coefficient (PRCC) analysis is deployed to assess the dependence of the model results on the parameters [7, 8, 9]. The ranges of model parameters used for the sensitivity analysis are summarized in Table 1 in the main text.

Fig. S4 shows the PRCCs between model parameters and individual payoff (E, see Eqn. (5)), and population risk level (Υ, Table 1 and S2.2) respectively. The ranges of model parameters are given in Table 1. Since "payoff" (the term in Fig. S4(a)) is the defined as the opposite number of "risk level" (the term in Fig. S4(b)), some model parameters have symmetric PRCC result with respect to level "0" (see the vertical grey dashed line in Fig. S4) on both panels. The PRCCs show that the results are most sensitive to the group of the relative risk (r), the basic reproduction number ($\mathcal{R}_0$), and the rate at which individuals leave the destination country (ν). Hence, these parameters should be the focus of data collection efforts during outbreaks when a travel policy must be decided. In Fig. S4(b), the basic reproduction number ($\mathcal{R}_0$) and relative risk (r) is

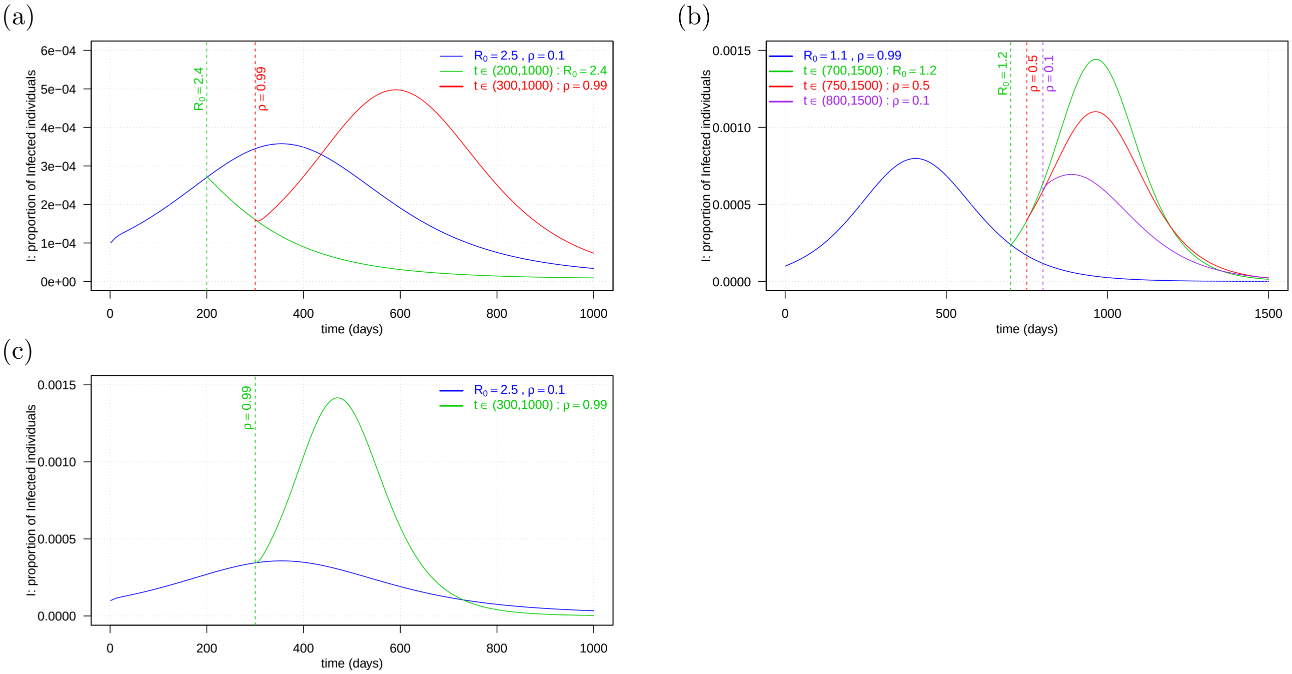

Figure S3: The simulation results of local infections (I) of epidemic model (panel (a) and (b), see Eqns. (S3)) and the SARS epidemic of China in 2002-03 (panel (c)). The baseline scenario contains that initial states are set as $[S(0), I(0), S_1(0), I_1(0)] = \left[\frac{1}{\mathcal{R}_0}, 1\times 10^{-4}, \left(K_1 - 5\times 10^{-6}\right), 5\times 10^{-6}\right]$; with $\mathcal{R}_0 = 2.5$ and $\rho = 0.1$ for panel **(a)** and **(c)**, and $\mathcal{R}_0 = 1.1$ and $\rho = 0.99$ for panel **(b)**. Values of other parameters are on Table 1. In panel **(a)**, the blue line is the simulation results under baseline scenario of panel **(a)**; the green line is of basic reproduction number ($\mathcal{R}_0$) decreasing to 2.4 since the 201-st day (vertical green dashed line); based on the change of green line, the red line is of travelling proportion (ρ) increasing to 0.99 since the 301-st day (vertical red dashed line). In panel **(b)**, the blue line is the simulation results under baseline scenario of panel **(b)**; the green line is of basic reproduction number ($\mathcal{R}_0$) increasing to 1.2 since the 701-st day (vertical green dashed line); based on the change of green line, the red line is of travelling proportion (ρ) decreasing to 0.50 since the 751-st day (vertical red dashed line); based on the change of red line, the purple line is of travelling proportion (ρ) continually decreasing to 0.10 since the 801-st day (vertical purple dashed line). In panel **(c)**, the blue line is the simulation results under baseline scenario of panel **(c)**; the green line is of travelling proportion (ρ) increasing to 0.99 since the 301-st day (vertical green dashed line).

strongly positively related to the population risk level (Υ), and the visitors leaving rate (ν) is negatively related to Υ. Opposite results can be seen in Fig. S4(a) for the individual payoff.

S6 Interpretation and Value of Some Model Parameters

S6.1 Rate of visitors moving from outside status to inside status λ

The value of the mean period of a traveler stay outside border (λ^{-1}) can be estimated by referring to the "deadline" of cancellation of hotel room, flight or even car-rent for travelling usage. For example, according to cancellation policies of Airbnb (`https://www.airbnb.com/home/`

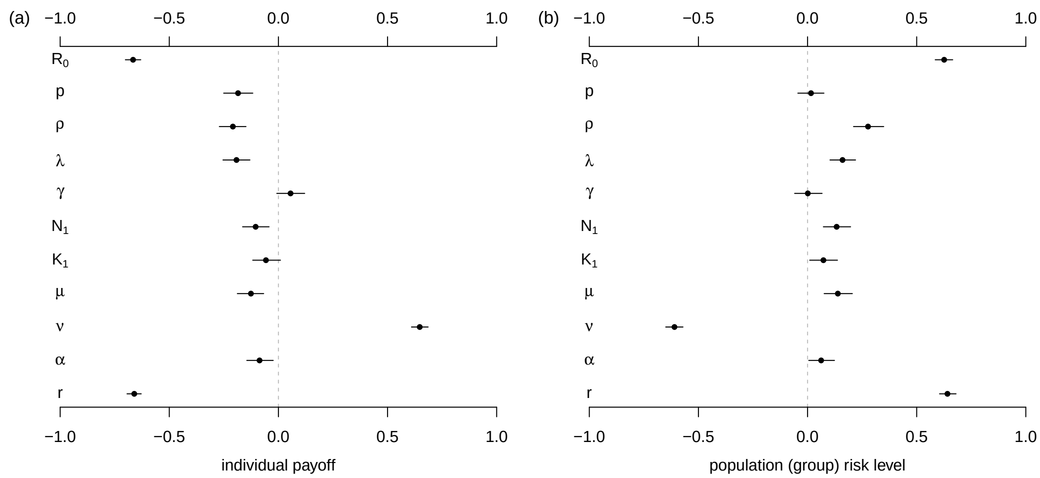

Figure S4: Sensitivity analysis results of (PRCCs) between model parameters and individual payoff (panel (a), see Eqn. (5)), and population risk level (Υ, in panel (b), see Table 1). The black dots are the estimated correlations and the bars represent 95% C.I.s. The ranges of model parameters are summarized on Table 1.

`cancellation_policies`), the waiver of refund charges can be considered for room-cancellation at least 1 day (with "flexible" policy) or 5 days (with "moderate" policy) in advance, thus we can make a rough estimation that $\lambda^{-1} \approx \frac{1}{2} \times (1+5) = 3$ days. According to Hong Kong Airline refund policies (`http://www.hongkongairlines.com/en_HK/flight/refund`), $\lambda^{-1} > 2$ days.

S6.2 Visitor capacity at destination K_1

According to the monthly travelling statistics (`http://partnernet.hktb.com/en/research_statistics/latest_statistics/index.html`) and travelling summary sheet (`http://partnernet.hktb.com/en/research_statistics/index.html`) from PartnerNet—Hong Kong tourism website for travel trade partners, there were approximate 58,000,000 travelers in Hong Kong of 2015 or 2016, and the local hotel room occupancy is roughly 87% over the whole period of time. Provided the information in S6.3, the local tavelling capacity of Hong Kong can be estimated as $\mathcal{N}K_1 = \frac{(58000000/87\%)\times 3}{365} \approx 550000$, here $\mathcal{N}$ denotes the number of total population capacity in Hong Kong (i.e., the sumation of upper bound of the number of travelers and local population, $\mathcal{N} = \mathcal{N}K_1 + \mathcal{N}\cdot(S+I+R)$). Given the popualtion statistics from World Bank (`https://data.worldbank.org/indicator/SP.POP.TOTL?locations=HK`), 7,300,000 is the number of local population in Hong Kong in 2015-16, thus $\mathcal{N} = \mathcal{N}K_1 + \mathcal{N}\cdot(S+I+R) = 550000 + 7300000 = 7850000$, and $K_1 = \frac{550000}{7850000} \approx 7.0\%$.

S6.3 Rate of visitors leaving destination ν

Referring to immigration department of the government of Hong Kong (`http://www.immd.gov.hk/eng/services/visas/visit-transit/visit-visa-entry-permit.html`), **Chinese** citizens can stay in Hong Kong for at maximal 7 days, and the majority of **non-Chinese** citizens can stay for roughly at maximal 15 days. According to the monthly travelling statistics from PartnerNet -

Hong Kong tourism website for travel trade partners (`http://partnernet.hktb.com/en/research_statistics/latest_statistics/index.html`), averagely, 75% of travelers are from mainland China and 25% are from other regions; for **Chinese** travelers, 50% of them are overnight passengers (expected to stay for $\frac{1}{2} \times (7+1) = 4$ days) and 50% them are one day visitors (expected to stay for $\frac{1}{2} \times (0+1) = 0.5$ day); for **non-Chinese** travelers, 66.67% of them are overnight passengers (expected to stay for $\frac{1}{2} \times (15+1) = 8$ days) and 33.33% them are one day visitors (expected to stay for $\frac{1}{2} \times (0+1) = 0.5$ day). Therefore, on average, one random-selected traveler would be expected to stay in Hong Kong for $\nu^{-1} = 75\% \times (50\% \times 4 + 50\% \times 0.5) + 25\% \times (\frac{2}{3} \times 8 + \frac{1}{3} \times 0.5) \approx 3$ days (thus, $\nu^{-1} = 3$ days).

S6.4 Relative risk r

The range of relative risk (r) can be approximated by simply checking the claim settlement odds of the travel insurance corresponding to the target place. For an example, according to travel insurance premium and coverage websites of Hang Seng Bank (`https://bank.hangseng.com/1/2/personal/insurance/travel-leisure/travel-insurance/travel-premium` and `https://bank.hangseng.com/1/2/personal/insurance/travel-leisure/travel-insurance/travel-coverage`), $r \approx 10^{-3}$.

S7 Further Discussion of Model Parameters

Relative risk $r = \frac{r_0}{r_1}$ (see Eqn. (S2) and Table 1) is the ratio of the "non-travelling" payoff ($E_0 = -r_0$, see main text) to the upper bound of the "travelling" payoff (i.e., $E_1 = -r_1$, see main text). The range of r could be obtain by referring to the claim-settlement-odds of the travel insurance with regard to the travelling destination (normally, $r \approx 10^{-3}$, see S6.4).

Number of visitors N_1 is the ratio of total number of potential visitors (i.e., game players) to the total population capacity. Provided total population capacity can be fixed in short term, the magnitude of N_1 is proportional to the number of potential visitors. We fix N_1 in this work. However, the number of potential visitors could be affected by seasonal factors (such as weather, school terms, holidays, etc.) and economic and politic factors (such as traffic expenditures, hotel fees, travelling policies [1], etc.), thus N_1 could be time-dependent in reality.

Agreement and conflict between ρ and p In Eqns. (S5) (and the epidemic model in the main text), $\frac{f_\rho}{\lambda} = \rho N_1$ is the proportion of visitors (outside border and about to be inside border shortly) to the total population capacity. ρ (see Table 1) is the proportion of potential visitors eventually becoming visitors correspond to the optimal travelling strategy selection. Therefore, we have $\rho = p^*$ (where p^* is individual's optimal travelling probability) under normal scenario (i.e., no serious disease outbreak, of which no restriction on travelling entry). However, during a serious disease outbreak, the local government will consider restricting travelling entry (in order to lower the number of visitors inside border) according to population's optimal travelling proportion (i.e., ρ^*), and this would change $\rho = \min\{p^*, \rho^*\}$. Numerical examples of local governmental intervention on travelling entry (i.e., ρ) are discussed in section S4. Note that, under governmental intervention scenario, ρ should only equal to ρ^* if $\rho^* < p^*$ (otherwise $\rho^* \geqslant p^*$, $\rho = p^*$ is equivalent to normal scenario).

Period of visitors staying outside the border λ^{-1} is defined as the mean period for a visitor used to get inside the border (see Table 1). We stepwise the "visiting" population as in Path (1) in main text. The λ^{-1} is the mean period for a visitor evolving from a "visitor outside" border to a "visitor inside" border. Note that a "potential visitor" can only become a "visitor outside" if he has finished his final travelling decision (see S1). The knowledge of the range of λ^{-1} can be learnt by referring to the "deadline" of withdrawal of various travelling "services" (e.g., hotel, flight, etc., see S6.1). Therefore, the speed of health information spread could be related to λ^{-1} because that the updating of relevant information can "renew" individual's final decision (i.e., re-choose strategy). Therefore, higher speed of information spread is corresponding to lower value of λ^{-1}.

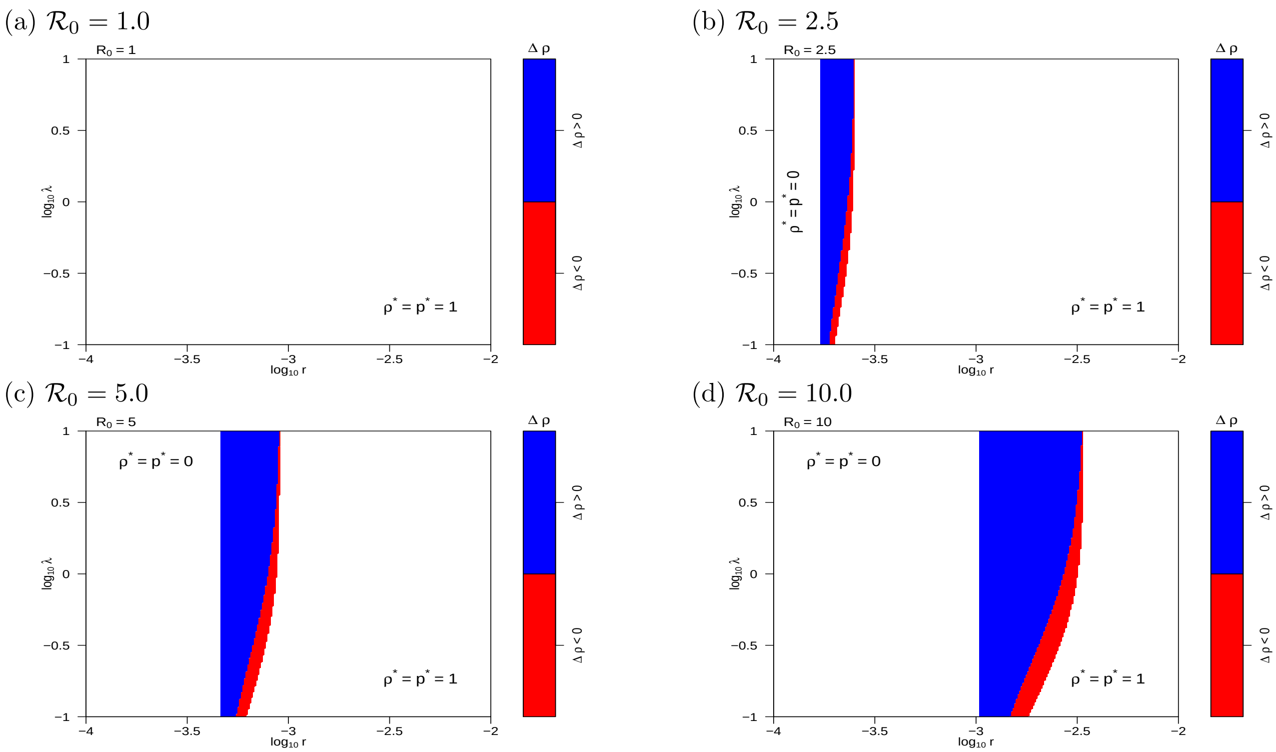

Figure S5: The relations among r, λ and $\Delta\rho$ (see main text) during epidemic (i.e., $\alpha = 1$) with $\mathcal{R}_0 = 1.0, 2.5, 5.0, 10.0$ for panel (a)-(d) respectively. The values of r and λ are in "$\log_{10}$" form. The color code of the difference of individual and population strategy, $\Delta\rho$, is shown on the color key. The white area (in each panel) represents $\Delta\rho = 0$ under two situations that $\rho^* = p^* = 0$ or 1. The values of other parameters are on Table 1.

Fig. S5 shows the relations among relative risk (r), rate of visitors pass border (λ) and $\Delta\rho$ during an epidemic. When λ increases, the discrepancy ($\Delta\rho$) of individual (p^*) and group optimum (ρ^*) appears under a wider range of relative risk (r). The discrepancy ($\Delta\rho$) shifts leftwards (the direction r increases) as $\mathcal{R}_0$ increasing. Particularly, p^* and ρ^* meet agreement (i.e., no discrepancy as $\rho^* = p^* = 1$) when $\mathcal{R}_0 = 1.0$ (which means disease cannot spread).

S8 Pre-existing immunity among visitors

For the model in the main text, we assume that all visitors are susceptible when entering the travel destination. In reality, this is not true. Pre-existing immunity of visitors could exist (e.g., the health authority of the visitors' home could recommend vaccination for visitors planning to go to a certain region where an epidemic is ongoing). The immunity level of the visitor population is dependent on a number of factors, including previous outbreaks, vaccination program policy and coverage, and the infection or vaccination history of the visitors, and may be estimated if the information is available. Thus, we denote

- P_{T} as the immunity level of the visitor population of a country;
- P_{O} as the immunity level of the rest of the population of the home countries of the visitors;
- P_{D} (i.e., term P in main text) as the immunity level of the local population of travel destination.

Then, the assumption $P_{\mathrm{D}} = P_{\mathrm{T}}$ or $P_{\mathrm{D}} = P_{\mathrm{O}}$, i.e., the immunity levels of local and origin populations are uniform, is unnecessary and can be relaxed. Moreover, the assumption $P_{\mathrm{T}} = P_{\mathrm{O}}$ can also be relaxed. In reality, $P_{\mathrm{T}} > P_{\mathrm{O}}$ could be common because (i) health authority of the visitors' home could recommend vaccination for visitors planning to go to an epidemic region; and (ii) vaccinated visitors are more likely to travel to an epidemic region.

After including P_{T}, the revised epidemic model becomes:

$$\begin{cases} S' = \mu \cdot (1 - K_1 - S) - \beta S \cdot (I + I_1), \\ S_1' = (1 - P_{\mathrm{T}}) f_\rho \cdot \left[1 - \dfrac{S_1 + I_1 + R_1}{(1 - P_{\mathrm{T}})K_1}\right] - \beta S_1 \cdot (I + I_1) - \nu S_1, \\ I' = \beta S \cdot (I + I_1) - (\gamma + \mu) I, \\ I_1' = \beta S_1 \cdot (I + I_1) - (\gamma + \nu) I_1, \\ R' = \gamma I - \mu R, \\ R_1' = \gamma I_1 - \nu R_1, \end{cases}$$

with all the terms remaining unchanged, except for inclusion of $(1-P_{\mathrm{T}})$ in $(1-P_{\mathrm{T}}) f_\rho \cdot \left[1 - \frac{S_1+I_1+R_1}{(1-P_{\mathrm{T}})K_1}\right]$.

We note that we could include one more equation,

$$X_1' = P_{\mathrm{T}} f_\rho \cdot \left[1 - \frac{S_1 + I_1 + R_1 + X_1}{(1 - P_{\mathrm{T}})K_1}\right] - \nu X_1,$$

where the additional state X_1 denotes visitors being protected against the disease, and the term $\frac{S_1+I_1+R_1}{(1-P_{\mathrm{T}})K_1}$ (in the revised model) should originally be written as $\frac{S_1+I_1+R_1+X_1}{K_1}$ (the same as in Eqn. X_1'). Since the magnitudes of both I_1 and R_1 are relatively small with respect to S_1 and X_1, we ignore the effects of I_1 and R_1 on the incoming visitors rate. Thus we have

$$S_1' \approx (1 - P_{\mathrm{T}}) f_\rho \cdot \left[1 - \frac{S_1 + I_1 + R_1 + X_1}{K_1}\right] - \nu S_1.$$

We can easily see that P_{T} of f_ρ joins in X_1, $(1-P_{\mathrm{T}})$ of f_ρ joins in S_1, and the leaving rates of X_1 and S_1 are the same as ν. To eliminate term X_1, we have $X_1 \approx \frac{P_T S_1}{(1-P_T)}$; therefore,

$$\frac{S_1+I_1+R_1+X_1}{K_1} \approx \frac{S_1+I_1+R_1}{(1-P_T)K_1},$$

as shown in the above revised model.

The term $(1-P_{\mathrm{T}})$ can be interpreted to mean that the protected visitors (P_{T}) are directly removed from the system (not by joining R_1, but by being "completely" removed from the model system), and the effect on the visitor input rate is partly reflected by "reducing" the local visitor capacity (i.e., replacing K_1 by $(1-P_{\mathrm{T}})K_1$). In this work, P_{T} is fixed to 0. Then, a new simplified model can be derived (from the revised model) by following the same method in S3 (by eliminating R and R_1). Since we regard P_{T} as a fixed nonzero constant (i.e., $P_T \neq 0$) during a short time period, and mathematically speaking, the effect of P_{T} can be transformed into a reduction of the magnitudes of f_ρ and K_1 [11], the main results in this work will hold for the revised epidemic model.

S9 Risk of visitors bringing the disease back to their home country

For the analysis in main text, for simplicity, we assume that visitors do not bring diseases back to their home country. This assumption is clearly overly optimistic. To amend this shortcoming, we may introduce one additional probabilistic factor of the risk level and obtain an improved travelling risk function

$$\Upsilon = \Upsilon(\rho,\pi) = N_1 \cdot \left[\rho \cdot \alpha\phi(\rho) \cdot (1+\pi \cdot \frac{\varrho}{r_1}) \cdot r_1 + (1-\rho) r_0\right],$$

where π is the average probability that the disease is brought back to the home country of a traveller, and ϱ is the average payoff of the disease spreading in a randomly selected home country.

Generally, we note that $\varrho > r_1$, since the consequences of a disease spreading in a region are presumed to be more serious than the consequences of a single individual being infected from a utilitarian point of view. We fix the ratio of $\frac{\varrho}{r_1}$ and use a similar idea as $r = \frac{r_0}{r_1}$. We view $(1+\pi \cdot \frac{\varrho}{r_1})$ as a scaler and assign a value to π. Thus the results of our original framework still hold, namely, the epidemic risk level of the travelling population, as listed in Table 1 in main text is a simplified version when $\pi = 0$.

References

[1] Cui JA, Takeuchi Y, Saito Y. Spreading disease with transport-related infection. Journal of theoretical biology. 2006;239(3):376-90.

[2] Bauch CT, Galvani AP, Earn DJ. Group interest versus self-interest in smallpox vaccination policy. Proceedings of the National Academy of Sciences of the United States of America. 2003;100(18):10564-7.

[3] Bauch CT, Earn DJ. Vaccination and the theory of games. Proceedings of the National Academy of Sciences of the United States of America. 2004;101(36):13391-4.

[4] Ciupe SM, Heffernan JM. In-host modeling. Infectious Disease Modelling. 2017.

[5] Ben-Shachar R, Koelle K. Minimal within-host dengue models highlight the specific roles of the immune response in primary and secondary dengue infections. Journal of the Royal Society Interface. 2015;12(103):20140886.

[6] Molina C, Earn DJD. Game theory of pre-emptive vaccination before bioterrorism or accidental release of smallpox. Journal of The Royal Society Interface. 2015;12(107):20141387.

[7] Ciupe SM, Heffernan JM. In-host modeling. Infectious Disease Modelling. 2017.

[8] Wu J, Dhingra R, Gambhir M, Remais JV. Sensitivity analysis of infectious disease models: methods, advances and their application. Journal of The Royal Society Interface. 2013;10(86):20121018.

[9] Gao D, Lou Y, He D, Porco TC, Kuang Y, Chowell G, Ruan S. Prevention and control of Zika as a mosquito-borne and sexually transmitted disease: a mathematical modeling analysis. Scientific reports. 2016;6:28070.

[10] Feng D, De Vlas SJ, Fang LQ, Han XN, Zhao WJ, Sheng S, Yang H, Jia ZW, Richardus JH, Cao WC. The SARS epidemic in mainland China: bringing together all epidemiological data. Tropical Medicine & International Health. 2009;14(s1):4-13.

[11] Earn DJ, Rohani P, Bolker BM, Grenfell BT. A simple model for complex dynamical transitions in epidemics. Science. 2000;287(5453):667-70.