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Individual preferences, government policy, and COVID-19: A game-theoretic epidemiological analysis

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ABSTRACT

Purpose: The ongoing COVID-19 pandemic imposes serious short-term and long-term health costs on populations. Restrictive government policy measures decrease the risks of infection, but produce similarly serious social, mental health, and economic problems. Citizens have varying preferences about the desirability of restrictive policies, and governments are thus forced to navigate this tension in making pandemic policy. This paper analyses the situation facing government using a game-theoretic epidemiological model.
Methodology: We classify individuals into health-centered individuals and freedom-centered individuals to capture the heterogeneous preferences of citizens. We first use the extended Susceptible-Exposed-Asymptomatic-Infectious-Recovered (SEAIR) model (adding individual preferences) and the signaling game model (adding government) to analyze the strategic situation against the backdrop of a realistic model of COVID-19 infection.
Findings: We find the following: 1. There exists two pooling equilibria. When health-centered and freedom-centered individuals send anti-epidemic signals, the government will adopt strict restrictive policies under budget surplus or balance. When health-centered and freedom-centered individuals send freedom signals, the government chooses not to implement restrictive policies. 2. When governments choose not to impose restrictions, the extinction of an epidemic depends on whether it has a high infection transmission rate; when the government chooses to implement non-pharmacological interventions (NPIs), whether an epidemic will disappear depends on how strict the government's restrictions are.
Originality/value: Based on the existing literature, we add individual preferences and put the government into the game as a player. Our research extends the current form of combining epidemiology and game theory. By using both we get a more realistic understanding of the spread of the virus and combine that with a richer understanding of the strategic social dynamics enabled by game theoretic analysis. Our findings have important implications for public management and government decision-making in the context of COVID-19 and for potential future public health emergencies.

Introduction

The ongoing COVID-19 pandemic continues to impose major health costs worldwide. Although the dominance of the Omicron variant and widespread vaccination have decreased the severity of infection and mortality rates, the problem of

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"long COVID" and the prevalence of reinfection remain serious concerns. There is increasing evidence that people face two significant problems: reinfection and sequelae. Antibodies produced by Omicron early in infection could not neutralize the variant Omicron [1,2]. As a result, people face cycle after cycle of reinfection. Reinfection will significantly increase the probability of COVID-19 sequelae [3–6]. Some people most affected by COVID-19 are protesting [7]. Non-drug measures, if implemented for a long time, will lead to economic downturn, human rights violations, and lower people's mental health [8–11]. Anti-lockdown protests have erupted in several cities of the world in the past [12–14]. As a result, there is a massive contradiction between those most affected by COVID-19 and those affected by non-pharmacological interventions (NPIs). What strategies the government should adopt to balance and moderate the contradiction between these two groups of people with different preferences is a burning research topic for study.

The detailed literature review presented in section 2 indicates that the existing literature has some limitations. They did not take individual preferences into account. The heterogeneity of preferences has not been studied in the past literature. Different preferences can have a big impact on decisions. The formulation of social mechanisms is greatly influenced by individual preferences. Individual preference is an important variable and can significantly affect government decisions and policies. Serious individual preference conflicts can even lead to demonstrations and social unrest. Therefore, this paper extends the theory based on the existing literature. The main purpose of this paper is to study the impact of penalty and subsidy mechanisms on individual behavior during COVID-19.

Our paper, based on the work of [15], extends their theoretical model. [15] studied optimal individual strategies for vaccination and social distancing. They combined the epidemiological Susceptible-Infected-Recovered (SIR) model with game theoretic analysis. They used the dynamic epidemiology results as players' payoff in the game model to study the optimal strategy of individual vaccination and social distancing. They found that when vaccination and social distancing are available to a population, social distancing is more likely to be the dominant strategy. They made a vital conclusion: social distancing is more likely to be an optimal choice for individuals than vaccination. However, they only analyzed individual behavior. In particular, they only from the perspective of individual incentives to do the relevant research for social distancing. Another significant incentive for social distancing comes from the government. Therefore, incorporating government into the model can better study NPIs, COVID-19, and individual behavior.

Choi and Shim [15] provide suggestions in the discussion section about NPIs. "*Additionally, if the severity of a disease increases, and government interventions, such as school closures and travel restrictions, are present, individuals may choose to increase their social distancing level*" ([15], p.11). "*Furthermore, the herd immunity threshold for social distancing cannot be achieved through voluntary participation alone (*e.g.*, school or work attendance and public transportation). Therefore, public health agencies should establish policies considering both social distancing and vaccination, such as subsidized vaccination, school closure, and cancelation of public events*" ([15], p. 10). However, those suggestions are based on individual incentives. From the government's perspective, the effectiveness of these suggestions is worthy of study. Therefore, adding the government in their model and classifying individual preferences further: freedom-centered and health-centered, is an essential contribution to our paper.

At the same time, this paper is also an extension of Zhou, et al. [16]. They used a tripartite evolutionary game to analyze decisions made by governments, businesses, and citizens in response to COVID-19. They found that different levels of punishment and reward lead to different evolutionary stability points. In other words, the difference between the reward and the punishment led to different decisions among the three players. The government's high penalties and low incentives policy can encourage businesses and citizens to fight the epidemic. At the same time, high penalties will create incentives for the government to fight the pandemic. The high level of punishment will encourage the three parties to fight the virus together. However, the article did not analyze the impact of joint or no response on the epidemic.

Therefore, we expand the model and add new variables based on their work. We introduce government and individual preferences. We analyze the strategies the government should adopt by analyzing the demands of individuals with different preferences. Our research emphasizes the impact of individuals with different preferences on government anti-epidemic policies and epidemic development. We note the following two research questions:

(1) Is there some equilibrium that can reach an agreement between the government and individuals with different preferences?
(2) What impact will government action have on the epidemic?

The above two research questions are closely related. Research question 1 studies the existence of equilibrium. When a certain equilibrium exists, governments and individuals with different preferences can reach an agreement, which means that policies can be implemented smoothly. Moreover, when such policies are implemented, they can substantially impact the spread and severity of the virus. Together, these two research questions address our research problem.

This research contributes to the literature in a number of ways. Theoretically, we extend the theoretical model from Choi and Shim [15] by adding the government sector and heterogeneous preferences. This adds political economy as well as game theory to epidemiological analysis. From a practical perspective, this allows us to consider a broader range of factors and expands the knowledge base on which pandemic policy can be made. Our findings can be applied not only to COVID-19 but also to potential future public health emergencies.

Following the introduction, the reminder of the paper is structured as follows: Section 2 presents the literature review. Section 3 establishes the epidemiological and signaling game models. The results are analyzed mathematically and then real

Omicron parameters are used to predict the epidemic development in section 3. We make a discussion for our results and analyzes their practical significance in section 4. Finally, we make conclusions in section 5.

Literature review

Many past studies have worked on the combination of epidemiological models and game theory models [17,18]. Methodologically, this literature is mainly based on the SIR model. Substantially, it mainly concerns individual behaviours such as individual social distancing and masking rather than policy choices by governments.

From the perspective of bounded rationality, some studies have used evolutionary game theory to study individual behavior. Tori and Tanimoto [19] used a combination of evolutionary game theory and the SEIR model to study the effects of mask-wearing on self-isolation and its potential risks to others. They found that the optimal state of society is one in which individuals would rather be infected than wear a mask because the cost of infection might be the same as the cost of preventing infection. They observed that the Nash equilibrium has a kind of "inertial effect," which makes individuals passive to take preventive measures, and partially inhibits the spread of disease. Amaral, et al. [20] studied individual risk perception and infection risk by combining evolutionary game theory with SIR Model. They argued that the individual's strategy depends on the risk of infection, the cost of quarantine, and the risk of infection. They found that an individual's risk of repeated infection stemmed from a low level of disease awareness. Martcheva et al. [21] also used evolutionary game theory and epidemiological models to study the impact of social distance on disease control and economic growth. They found that the epidemic could end if everyone practiced complete social distancing. Kabir et al. [22] have also applied the same approach. They studied the behavior of individuals wearing masks. They assume that individuals do not want to wear masks but want others to wear them. They found that whether an individual wears a mask depends on a trade-off between the cost and the risk of infection. Wei et al. [23] found through an evolutionary game that the emergency strategy adopted by the government at the early stage of the epidemic could effectively control the spread of the epidemic.

Others analyze individual behavior using traditional game theory (which makes the assumption of perfect rationality) combined with epidemiological models. Jia et al. [24] analyzed potential mismatched priorities among levels of policymakers (e.g., federal, state, and local governments) using a multi-level game theory model of hierarchical decision-making. They analyzed government policies and implementation strategies during the epidemic from factors such as whether policies were implemented, implementation costs, and implementation priorities. They found free riding and unfair cost distribution in the policy implementation process. Özkaya and Izgi [25] used game theory to study three distinct phases of an outbreak: the beginning, spread, and end of a pandemic. Their study of South Korea, Italy, and Turkey found that isolation significantly impacted infection and pandemic transmission at all levels and that the phenomenon was similar in the countries studied.

Soltanolkottabi et al. [26] used the public goods game model to investigate the social dynamics of vaccination and virus transmission in epidemic outbreaks. They found that the more attention individuals paid to the epidemic, the fewer infections there were. Nevertheless, this emphasis significantly impacts the number of people vaccinated. When the incubation period is extended, more infected people and fewer people get vaccinated. Similarly, Deka et al. [27] also studied public attitudes toward vaccination. By comparing the cost of vaccination, they found that vaccination rates increased when an individual's risk perception evolved with deaths from vaccination or disease. Vaccination at the beginning and later in an epidemic outbreak minimizes the optimal cost. Wang et al. [28] used SIR Model and Monte Carlo simulation to study group imitation behavior during the epidemic. They found that herd mimicry can suppress herd immunity. This means that if one person is unwilling to wear a mask or self-isolate, others will copy. Therefore, such behavior is not conducive to herd immunity.

Through a review of existing studies, we found that the existing literature has done an excellent job of studying the strategies individuals should adopt during an epidemic. They comprehensively analyzed the importance of individual decisions such as social distancing and wearing masks. However, we find that some limitations are also reflected. They only analyzed individual strategies. Using individuals and the epidemic as a set of participants, they analyzed the impact of individual actions on the epidemic. The critical factor of government also needs to be taken into account. As a game player, the government can form non-cooperative (cooperative) games with individuals. Therefore, putting the government as a participant in the participant set is a topic worthy of study. We also find that the existing literature does not consider the heterogeneity of individual preferences. Since preferences over individual behaviours and government policies differ among individuals, differentiating individuals by type is an important consideration.

Zhou et al. [16] conducted a study on the limitations of the above literature that did not consider the heterogeneity of preferences. They used tripartite evolutionary game theory to analyze whether governments, businesses, and citizens were resilient. They divide civic preferences into health-centered and freedom-centered. Individuals with different preferences have different influences on restrictive policies. They found that governments must adopt a high-penalty mechanism if both players prevent the epidemic. The severe punishment mechanism can not only bring incentives for citizens and enterprises to fight the epidemic but also bring financial revenue to the government to guarantee the government's incentive to fight the epidemic. For the first time, they used citizens' preferences to analyze player decisions during an epidemic. However, they did not analyze the impact of these decisions on the pandemic. Based on their classification of citizen preferences, we use a combination of epidemiological models and traditional game theory further to analyze the impact of decision-making on the epidemic.

In terms of the game between the government and individuals, Khan et al. [29] further complements the limitations of previous scholars. Their topic is fascinating. They look at the cost of mandatory isolation for individuals. On a shoestring budget, the government pays for mandatory isolation for individuals. They used dynamic equations to study the balance between the budget of an individual case and the risk of infection. They found that the peak of infection decreased as government interventions increased. Government restrictions can reduce the prevalence sufficiently. At the same time, their research fills in another gap: the impact of government restrictions on social welfare. They developed a new concept: the Social Efficiency Deficit (SED). It measures the potential gain in utility or fitness from an actor moving from evolutionary equilibrium to social optimum [30]. That index can better measure the gap between the optimum social payoff and equilibrium payoff. They explained the game relationship between government and individuals from the social welfare perspective. Other scholars not only studied the strategic interaction between government policies and individuals in the case of a single strain but also used the average field game theory to study the strategic interaction between participants in the case of different strains. This research further expands the theoretical basis of emergency public events management. The cutting-edge models and research of epidemiology and game theory can be referred to Tanimoto's book [31].

Methodology

Expanded SEAIR model

Based on the model structure of Choi and Shim [15], we further expand the SIR model. Our model is divided into two sectors: Individuals and the government. Suppose an epidemiology state space $\Omega = (S(t),\ F(t),\ E(t),\ A(t),\ I(t),\ R(t),\ \mathcal{T},\ H(t), P_{hc}(t),\ \lambda(t);\{\eta_i\}_{i\in\mathcal{T}},\ \delta,\ \mu,\ k,\ \gamma)$. Most factors in this epidemiology state space are based on the assumptions from [15]: susceptible ($S(t) \subset \mathbb{R}^+$), exposed ($E(t) \subset \mathbb{R}^+$), symptomatic ($I(t) \subset \mathbb{R}^+$), asymptomatic ($A(t) \subset \mathbb{R}^+$), and recovered ($R(t) \subset \mathbb{R}^+$). $N(t)$ represents the total population, and $N(t) = S(t) + H(t) + F(t) + E(t) + I(t) + A(t) + R(t)$.

We defined the following basic parameters as the same with Choi and Shim [15]: reduction contact rate $\{\eta_i\}_{i\in\mathcal{T}} \in [0,\ 1]$; recover rate for asymptomatic and symptomatic individuals $\gamma \in [0,\ 1]$; Natural death rate $\mu \in \mathbb{R}^+$; There is probability p of exposed individuals becoming symptomatic individuals and probability $1 - p$ of becoming asymptomatic individuals.

$\mathcal{T} = \{h,\ f\}$ represents the individuals' type set. This is the first novel contribution of this paper. We divide the population into two types according to their preference: health-centered individuals and freedom-centered individuals [16], denoted as $H(t) \subset \mathbb{R}^+$ and $F(t) \subset \mathbb{R}^+$, respectively. Health-centered individuals give priority to health. In the context of an epidemic, they see health as an overriding concern. Therefore, as long as they think the epidemic will severely threaten their health, they will increase the reduction contact rate $\eta_h \in (\frac{1}{2},\ 1]$ to maintain social distancing and prevent infection. In contrast, freedom-centered individuals put freedom first. In the context of an epidemic, they will minimize the reduction contact rate $\eta_f \in [0,\ \frac{1}{2})$ to ensure adequate freedom. They are opposed to government restrictions. These two groups of individuals correspond to the situation we introduced in the introduction.

Although the vaccine has led to a decline in severity and mortality rates, the number of actual infections and deaths is still rising [32]. Government therefore has a continued interest in controlling the spread of the virus through public health measures. However, this must be balanced against the economic and mental health costs of restrictive policies as well as the preferences of freedom-centered citizens who regard many public health policies as overly restrictive [13,33–36].

In symptomatic individuals, hospitalization rate $\nu \in [0,\ 1]$. Our research subjects are individuals who need to express their own type. Therefore, we define the type expression rate as $\{\alpha_i\}_{i\in\mathcal{T}} \in [0,\ 1]$ (a number of susceptible individuals expressing ith type per unit of time). The individuals who express their type (health-centered or freedom-centered) to the government through media, comments, socializing, etc. Thus, we define the expression as a rate. When $\alpha_i = 0$, it means no one express their type. When $\alpha_i = 1$, it means Individual types are fully expressed. When $\alpha_i \in (0,\ 1)$, it means only some individuals express their type. We define the natural infection rates as follows Choi and Shim [15]:

$$\frac{\beta_0(I(t) + bA(t))}{N(t)} = \lambda(t) \tag{2.1}$$

Where $\beta_0 \in \mathbb{R}^+$ is the transmission infection rate and $b \in \mathbb{R}^+$ is the rate of relative infectiousness of asymptomatic cases compared to symptomatic cases [15]. Appendix B shows the description of the parameters and variables. Different types of individuals have different potential distribution functions. According to the above assumptions, the dynamic equation of individual epidemiology can be expressed as follows:

$$\dot{S}(t) = \Lambda N(t) - \left(\alpha_h + \alpha_f + \mu\right)S(t) \tag{2.2}$$

$$\dot{H}(t) = \alpha_h S(t) - [\mu + (1 - \eta_h)\lambda(t)]H(t) \tag{2.3}$$

$$\dot{F}(t) = \alpha_f S(t) - \left[\left(1 - \eta_f\right)\lambda(t) + \mu\right]F(t) \tag{2.4}$$

$$\dot{E}(t) = (1 - \eta_h)\lambda(t)H(t) + \left(1 - \eta_f\right)\lambda(t)F(t) - (k + \mu)E(t) \tag{2.5}$$

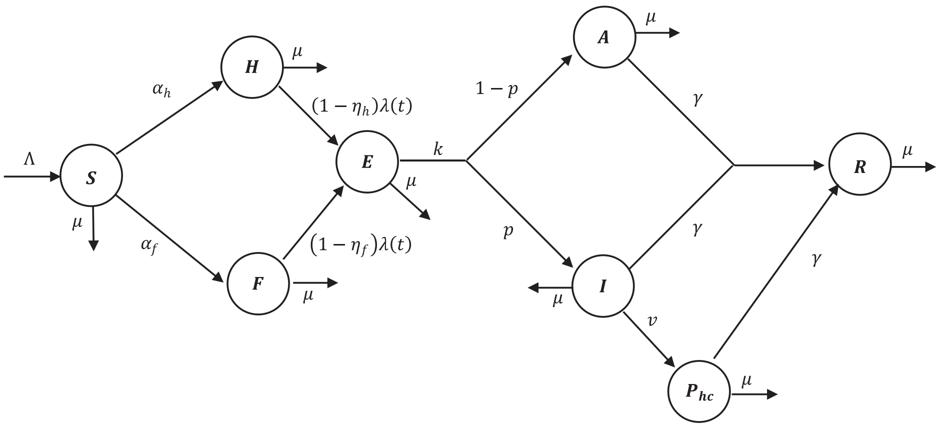

Fig. 1. Dynamic process of individuals and the government epidemiology.

$$\dot{I}(t) = kpE(t) - (\gamma + \mu + v)I(t) \tag{2.6}$$

$$\dot{P}_{hc}(t) = vI(t) - (\mu + \gamma)P_{hc}(t) \tag{2.7}$$

$$\dot{A}(t) = k(1-p)E(t) - (\gamma + \mu)A(t) \tag{2.8}$$

$$\dot{R}(t) = \gamma A(t) + \gamma P_{hc}(t) + \gamma I(t) - \mu R(t) \tag{2.9}$$

Fig. 1 shows the dynamic process of individual and the government epidemiology. To reduce the dimension of the model (let $h(t) = \frac{H(t)}{N(t)}$, $i(t) = \frac{I(t)}{N(t)}$, $r(t) = \frac{R(t)}{N(t)}$, $a(t) = \frac{A(t)}{N(t)}$, $e(t) = \frac{E(t)}{N(t)}$ and $f(t) = \frac{F(t)}{N(t)}$), we re-express the equation (2.2)–(2.9) as follows:

$$\dot{h}(t) = \alpha_h(1 - e(t) - a(t) - i(t) - r(t) - p_{hc}(t) - f(t)) - [\mu + (1-\eta_h)\lambda(t)]h(t) \tag{2.10}$$

$$\dot{\beta}(t) = \alpha_f(1 - e(t) - a(t) - i(t) - \beta(t) - p_{hc}(t) - h(t)) - \left[\left(1-\eta_f\right)\lambda(t) + \mu\right]\beta(t) \tag{2.11}$$

$$\dot{e}(t) = (1-\eta_h)\lambda(t)h(t) + \left(1-\eta_f\right)\lambda(t)f(t) - (k+\mu)e(t) \tag{2.12}$$

$$\dot{i}(t) = kpe(t) - (\gamma + \mu + v)i(t) \tag{2.13}$$

$$\dot{p}_{hc}(t) = vi(t) - (\mu + \gamma)p_{hc}(t) \tag{2.14}$$

$$\dot{a}(t) = k(1-p)e(t) - (\gamma + \mu)a(t) \tag{2.15}$$

$$\dot{\gamma}(t) = \gamma a(t) + \gamma p_{hc}(t) + \gamma i(t) - \mu\gamma(t) \tag{2.16}$$

We can define the disease-free equilibrium[1] (DFE) as follows: $(h_0(t), f_0(t),\ e_0(t),\ a_0(t),\ i_0(t),\ (p_{hc})_0(t),\ r_0(t)) = \left(\dfrac{\alpha_h(\mu-\alpha_f)}{\mu^2-\alpha_h\alpha_f},\ \dfrac{\alpha_f(\mu-\alpha_h)}{\mu^2-\alpha_h\alpha_f},\ 0,\ 0,\ 0,\ 0,\ 0\right)$.

By using this DFE, we can obtain the control reproduction number $R_c(\eta_f, \eta_h)$ of the model by using the next-generation method [37]. We compute the basic reproduction number under the assumption that the control parameters (η_f, η_h) are

[1] Substitute $(e_0(t),\ a_0(t),\ i_0(t),\ (p_{hc})_0(t),\ r_0(t)) = (0,\ 0,\ 0,\ 0,\ 0)$ into the equations (2.11) – (2.17), we can have: $\begin{cases} 0 = \alpha_h(1 - f(t)) - [\mu + (1-\eta_h)\lambda(t)]h(t) \\ 0 = \alpha_f(1 - h(t)) - [(1-\eta_f)\lambda(t) + \mu]f(t) \end{cases}$. First, we have $\lambda(t) = 0$. By solving the functions, then, we have: $\begin{cases} h_0(t) = \dfrac{\alpha_h(\mu-\alpha_f)}{\mu^2-\alpha_h\alpha_f} \\ f_0(t) = \dfrac{\alpha_f(\mu-\alpha_h)}{\mu^2-\alpha_h\alpha_f} \end{cases}$.

fixed at constant values. $R_0(\eta_f, \eta_h)$ can be obtained by introducing matrices $\mathcal{F} = \begin{pmatrix} (1-\eta_h)\lambda(t)h(t) + (1-\eta_f)\lambda(t)\not{f}(t) \\ 0 \\ 0 \\ 0 \end{pmatrix}$ and $\mathcal{V} = \begin{pmatrix} (k+\mu)e(t) \\ -kpe(t) + (\gamma+\mu+\nu)i(t) \\ -k(1-p)e(t) + (\gamma+\mu)a(t) \\ -\nu i(t) + (\mu+\gamma)p_{hc}(t) \end{pmatrix}$ corresponding to transmission and transition, respectively. In other words, $\mathcal{F}$ represents the rate of appearance of new infections in compartment i, $\mathcal{V}$ represents the rate of transfer of individuals into and out of compartment i, where:

$$X = \begin{pmatrix} e(t) \\ i(t) \\ p_{hc}(t) \\ a(t) \end{pmatrix} = \mathcal{F} - \mathcal{V} = \begin{pmatrix} \left[(1-\eta_h)h(t) + \left(1-\eta_f\right)\not{f}(t)\right]\beta_0[i(t) + ba(t)] \\ 0 \\ 0 \\ 0 \end{pmatrix} - \begin{pmatrix} (k+\mu)e(t) \\ -kpe(t) + (\gamma+\mu+\nu)i(t) \\ -k(1-p)e(t) + (\gamma+\mu)a(t) \\ -\nu i(t) + (\mu+\gamma)p_{hc}(t) \end{pmatrix}$$

Using the next generation method, we can obtain the following Jacobian matrix:

$$\mathrm{F} = \begin{pmatrix} 0 & \frac{\partial \mathcal{F}_{11}}{\partial i(t)} & \frac{\partial \mathcal{F}_{11}}{\partial a(t)} & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}, \quad \tilde{\mathrm{V}} = \begin{pmatrix} k+\mu & 0 & 0 & 0 \\ -kp & \gamma+\mu+\nu & 0 & 0 \\ -k(1-p) & 0 & \gamma+\mu & 0 \\ 0 & -\nu & 0 & \mu+\gamma \end{pmatrix}$$

Where:

$$\frac{\partial \mathcal{F}_{11}}{\partial i(t)} = \beta_0\left[(1-\eta_h)\frac{\alpha_h\left(\mu-\alpha_f\right)}{\mu^2-\alpha_h\alpha_f} + \left(1-\eta_f\right)\frac{\alpha_f(\mu-\alpha_h)}{\mu^2-\alpha_h\alpha_f}\right]$$

$$\frac{\partial \mathcal{F}_{11}}{\partial a(t)} = \beta_0 b\left[(1-\eta_h)\frac{\alpha_h\left(\mu-\alpha_f\right)}{\mu^2-\alpha_h\alpha_f} + \left(1-\eta_f\right)\frac{\alpha_f(\mu-\alpha_h)}{\mu^2-\alpha_h\alpha_f}\right]$$

By using the spectral radius of the matrix $\mathrm{F}\tilde{\mathrm{V}}^{-1}$, we can obtain the effective reproduction number $R_c(\eta_f, \eta_h, \alpha_h, \alpha_f)$ as follows:

$$R_c\left(\eta_f,\ \eta_h\right) = \Xi\left\{\mu\left[\alpha_f\left(1-\eta_f\right) + \alpha_h(1-\eta_h)\right] + \alpha_f\alpha_h\left(\eta_f+\eta_h-2\right)\right\}$$

Where $\Xi = \frac{\beta_0 k[b(1-p)(\gamma+\mu+\nu)+p(\gamma+\mu)]}{(\gamma+\mu+\nu)(k+\mu)(\mu^2-\alpha_f\alpha_h)(\gamma+\mu)}$. We can express basic reproduction number $R_0(0,0)$ as follows, if $(\eta_f, \eta_h) = (0, 0)$:

$$R_0(0,\ 0) = \Xi\left[\mu\left(\alpha_f+\alpha_h\right) - 2\alpha_f\alpha_h\right]$$

Then we have:

$$R_c = \frac{\mu\left[\alpha_f\left(1-\eta_f\right) + \alpha_h(1-\eta_h)\right] + \alpha_f\alpha_h\left(\eta_f+\eta_h-2\right)}{\mu\left(\alpha_f+\alpha_h\right) - 2\alpha_f\alpha_h}R_0$$

We use the following proposition to summarize the stability of the model. Proposition 1 has two basic implications. If $R_0 \le 1$, COVID-19 will gradually disappear. If $R_0 > 1$, COVID-19 will spread exponentially and become an epidemic.

Proposition 1:

1. there is a unique globally asymptotically stable equilibrium point $(h_0(t), \not{f}_0(t),\ e_0(t),\ a_0(t),\ i_0(t),\ (p_{hc})_0(t),\ r_0(t))$, if $\mu(\alpha_f+\alpha_h) - 2\alpha_f\alpha_h \le \frac{1}{\Xi}$
2. there is a unique globally asymptotically stable equilibrium point $(h^*(t), \not{f}^*(t),\ e^*(t),\ a^*(t),\ i^*(t),\ (p_{hc})^*(t))$, if $\mu(\alpha_f+\alpha_h) - 2\alpha_f\alpha_h > \frac{1}{\Xi}$.

Where $(h^*(t), \not{f}^*(t),\ e^*(t),\ a^*(t),\ i^*(t),\ (p_{hc})^*(t))$ is the solution of equations (2.10) – (2.16).

Proof. See Appendix A.1. ■

Game theory model

Consider a game space $\Theta = (N,\ \{S_i\}_{i\in N},\ \{u_i(\cdot)\}_{i\in N},\ \{A_i\}_{i\in N},\ T,\ \tilde{N})$, where $N = \{G,\ C\}$ represent participants set. We denote G as the government and C as individuals. $\tilde{N}$ is Nature [38]. $S_i = \{s_i^{(1)},\ s_i^{(2)},\ \ldots,\ s_i^{(n_i)}\},\ i \in N$ represents the ith player's strategy set, and $S_i \triangleq \prod_{i=1}^{n} S_i$. Define the mixed strategy σ_i: $S_i \to [0,\ 1]$ as a probability distribution on S_i, where

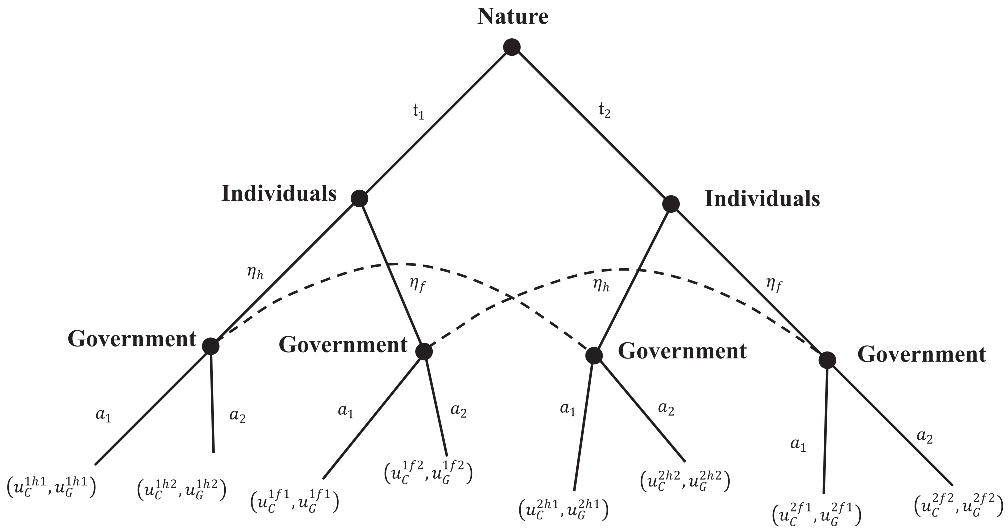

Fig. 2. Government and individuals' game tree.

$\sum_{k=1}^{n_i} \sigma_i(s_i^{(k)}) = 1$. $A_i, i \in N$ is the action set, where $A_G = \{a_1, a_2\}$ and $A_c = \{\eta_h, \eta_f\}$, a_1 represents "implement NPIs" and a_2 represents do nothing; η_i represents individuals choose to signal η_i, $i \in \mathcal{T}$. $T = \{\mathfrak{t}_1, \mathfrak{t}_2\}$ represents individuals' type set, where $\mathfrak{t}_1$ is health-centered type and $\mathfrak{t}_2$ is freedom-centered type. $(u_i(\cdot))_{i \in N}$ is the players' payoff function.

We make the following assumptions about individuals. According to [15], we set individuals' payoff function as follows: $\pi_h = \frac{\alpha_h(1-\eta)\lambda}{\alpha_h(1-\eta)\lambda+\mu}$ and $\pi_{\not f} = \frac{\alpha_f(1-\eta)\lambda}{\alpha_f(1-\eta)\lambda+\mu}$. We use the subscript h and $\not f$ to denote health-centered and freedom-centered individuals distancing, respectively. The probabilities of infection among health-centered and freedom-centered individuals, which are denoted as π_h and $\pi_{\not f}$, respectively.

The government's goal is to minimize the total social risk of infection and to achieve budget balance. The government's incentives to fight the epidemic come from a surplus or balance of expenditure. We assume that the government will punish individuals who do not comply with restrictions, the punishment coefficient is denoted as ζ. The punishment mechanism increases the government's anti-epidemic incentive since it can increase the government's revenue. The government expenditure dynamic equation can be represented as:

$$\dot{g}(t) = \zeta(1-\eta)s(t) - \varrho p_{hc}(t) \tag{2.17}$$

We denote the marginal cost for public health per individuals as ϱ. The government optimal programming can be expressed as follows:

$$\begin{cases} \min_{\eta} \left\{ \frac{\alpha_h(1-\eta)\lambda}{\alpha_h(1-\eta)\lambda+\mu} + \frac{\alpha_f(1-\eta)\lambda}{\alpha_f(1-\eta)\lambda+\mu} \right\} \\ s.t. \zeta(1-\eta)s(t) - \varrho p_{hc}(t) \geq 0 \end{cases} \tag{1}$$

Fig. 2 shows the dynamic game between the government and individuals. In the first stage, individuals select a reduction contact rate η. Furthermore, the government's judgment on whether to impose restrictions depends on the will of the individual. When individuals send information to the government that they want to be healthy, the government imposes restrictions. In this paper, we express individual willingness through reduction contact rate η. As we assumed earlier, when $\eta \in (\frac{1}{2}, 1]$, the individual wants to be healthy. When $\eta \in [0, \frac{1}{2})$, individuals want to be free. In the second stage, the government decides whether to implement NPIs. If the government does not implement NPIs, individuals remain free as they currently are. If the government chooses to implement NPIs in the second stage, it will choose η^* to minimize the total social infection risk. According to programming (1), we can have $\eta^* = 0$. Suppose the health-centered individuals' posterior probability is w, $1-w$ is the probability of freedom-centered. If individuals choose the reduction contact rate η_h, their short-term payoff is $\pi_h(\eta_h, \alpha_h)$ or $\pi_{\not f}(\eta_h, \alpha_h)$. In the first stage, the individuals' type is a private information. We assume that in the second stage, once the government chooses to implement NPIs, it will know the actual type of individual. Thus, the reduction of contact rate in the second stage is independent compared with that in the first stage. Under complete information, the government will choose to implement NPIs if and only if most of individuals are health-centered. We use d for the discount rate. Table 1 shows the players' payoff function. We make a further assumption for the government when it choose anti-epidemic. The government will have a fixed risk $\xi \in [0, +\infty)$ when it chooses anti-epidemic. If the government takes action, it may be at high cost but little benefit.

Table 1
Players' payoff function corresponding to Fig. 2.

Individuals	Government
$u_C^{1h1}=0$	$u_G^{1h1}=\xi$
$u_C^{1h2}=\frac{\alpha_h(1-\eta_h)\lambda(1+d)}{\alpha_h(1-\eta_h)\lambda+\mu}$	$u_G^{1h2}=\left(\frac{\alpha_h(1-\eta_h)\lambda}{\alpha_h(1-\eta_h)\lambda+\mu}+\frac{\alpha_f(1-\eta_h)\lambda}{\alpha_f(1-\eta_h)\lambda+\mu}\right)(1+d)$
$u_C^{1f1}=\frac{\alpha_h(1-\eta_f)\lambda}{\alpha_h(1-\eta_f)\lambda+\mu}$	$u_G^{1f1}=\frac{\alpha_h(1-\eta_f)\lambda}{\alpha_h(1-\eta_f)\lambda+\mu}+\xi$
$u_C^{1f2}=\frac{\alpha_h(1+d)(1-\eta_f)\lambda}{\alpha_h(1-\eta_f)\lambda+\mu}$	$u_G^{1f2}=\left(\frac{\alpha_h(1-\eta_f)\lambda}{\alpha_h(1-\eta_f)\lambda+\mu}+\frac{\alpha_f(1-\eta_f)\lambda}{\alpha_f(1-\eta_f)\lambda+\mu}\right)(1+d)$
$u_C^{2h1}=0$	$u_C^{2h1}=\xi$
$u_C^{2h2}=\frac{\alpha_f(1+d)(1-\eta_h)\lambda}{\alpha_f(1-\eta_h)\lambda+\mu}$	$u_G^{2h2}=\left(\frac{\alpha_h(1-\eta_h)\lambda}{\alpha_h(1-\eta_h)\lambda+\mu}+\frac{\alpha_f(1-\eta_h)\lambda}{\alpha_f(1-\eta_h)\lambda+\mu}\right)(1+d)$
$u_C^{2f1}=\frac{\alpha_f(1-\eta_f)\lambda}{\alpha_f(1-\eta_f)\lambda+\mu}$	$u_G^{2f1}=\frac{\alpha_f(1-\eta_f)\lambda}{\alpha_f(1-\eta_f)\lambda+\mu}+\xi$
$u_C^{2f2}=\frac{\alpha_f(1-\eta_f)(1+d)\lambda}{\alpha_f(1-\eta_f)\lambda+\mu}$	$u_G^{2f2}=\left(\frac{\alpha_h(1-\eta_f)\lambda}{\alpha_h(1-\eta_f)\lambda+\mu}+\frac{\alpha_f(1-\eta_f)\lambda}{\alpha_f(1-\eta_f)\lambda+\mu}\right)(1+d)$

Note: The superscript represents the corresponding branch in the Fig. 2. For example, u_C^{1h1} indicates the infection risk of health-centered individuals in choosing to send signals η_h when the government takes a strict NPIs.

Proposition 2: The following mixed perfect Bayesian Nash equilibria hold:

$$(1)\ \left((\eta_h,\ \eta_h),\ (a_1,\ a_1);\ w\in\left[0,\ \frac{1+d}{\xi+(1+d)}\right]\cap\left[\frac{(1+d)\left(\frac{\alpha_h(1-\eta_f)\lambda}{\alpha_h(1-\eta_f)\lambda+\mu}\right)}{(2+d)\left(\frac{\alpha_h(1-\eta_f)\lambda}{\alpha_h(1-\eta_f)\lambda+\mu}\right)+\xi},\ 1\right),\ l\in[0,1+d)\right);$$

$$(2)\ \left((\eta_f,\ \eta_f),\ (a_2,\ a_2);\ w\in\left[0,\ \frac{(1+d)\frac{\alpha_f\lambda}{\alpha_f\lambda+\mu}}{(2+d)(\frac{\alpha_f\lambda}{\alpha_f\lambda+\mu})+\xi}\right]\cap\left[\frac{(1+d)}{\xi+(1+d)},\ 1\right)\right),\ \forall l\in[0,\ 1],\text{ if }\alpha_f\le\frac{\mu(1+d)}{\lambda(1+d-2\xi)}.$$

Proof see Appendix A.2. ■

Proposition 2 answers our first research question: *Is there some equilibrium that can reach an agreement between the government and individuals with different preferences?* The action of the first-acting signaling sender transmits information to the receiver of the second-acting signal. At the same time, this makes the receiver's actions dependent on the signal chosen by the sender. When individuals do not want the government to implement NPIs in the second stage, they will protest if the government imposes restrictive policies. Assume that the freedom-centered individuals choose signaling η_f. If health-centered individuals choose η_h to insist the government implementing NPIs, their total infection risk would be 0. Thus, health-centered individuals would not choose the same signals as freedom-centered individuals only if the following condition is met: $\eta_f=0$. Similarly, on the one hand, when freedom-centered individuals choose signals η_f to prevent the government from implementing NPIs, their total infection risk would be: $\frac{\alpha_f(1-\eta_f)(1+d)\lambda}{\alpha_f(1-\eta_f)\lambda+\mu}$; On the other hand, if they choose $\eta>\eta_f$, their total payoff will not be lower than η_f, because they can always choose a η higher than η_f to induce the government to implement NPIs. Therefore, η_f is a signal sending from the freedom-centered individuals only if the following conditions are true: $\eta_f=0$.

Our hypothesis satisfies Spence-Mirrlees condition [39]. It shows that reduction contact rates have different effects on utility for different types of individuals; In particular, health-centered individuals are more likely to choose higher η than freedom-centered individuals. Fig. 3 shows the individual signaling and screening mechanism. The condition for the existence of pooling equilibrium is:

$$lu_G^{1i1}+(1-l)u_G^{2j1}>0\Rightarrow\frac{\alpha_h(1-\eta_i)\lambda}{\alpha_h(1-\eta_i)\lambda+\mu}+\frac{\alpha_f(1-\eta_i)\lambda}{\alpha_f(1-\eta_i)\lambda+\mu}<0,\ i,\ j\in\{h,\ f\}\text{ and }i\ne j \tag{2.18}$$

If the government does not receive new information, it chooses not to implement NPIs. If the condition (2.18) is not true, under the pooling equilibrium, the government will choose to implement NPIs; Because pooling equilibrium cannot prevent the implementation of NPIs, the optimal choice of health-centered individuals is $\eta_{max}=1$, and the optimal choice of freedom-centered individuals is: 0. Therefore, as long as condition (2.18) is not true, there is no pooling equilibrium. Suppose the condition (2.18) is true. There is a reduction contact rate η that prevents governments from implementing NPIs. It should be satisfied that neither health-centered nor freedom-centered individuals are willing to deviate from the η_i. If not, it will induce the government to implement NPIs in the worst-case scenario. Both types of individuals choose the lower (higher) reduction contact rate (health-centered (freedom-centered) individuals choose the lower (higher) reduction contact rate to

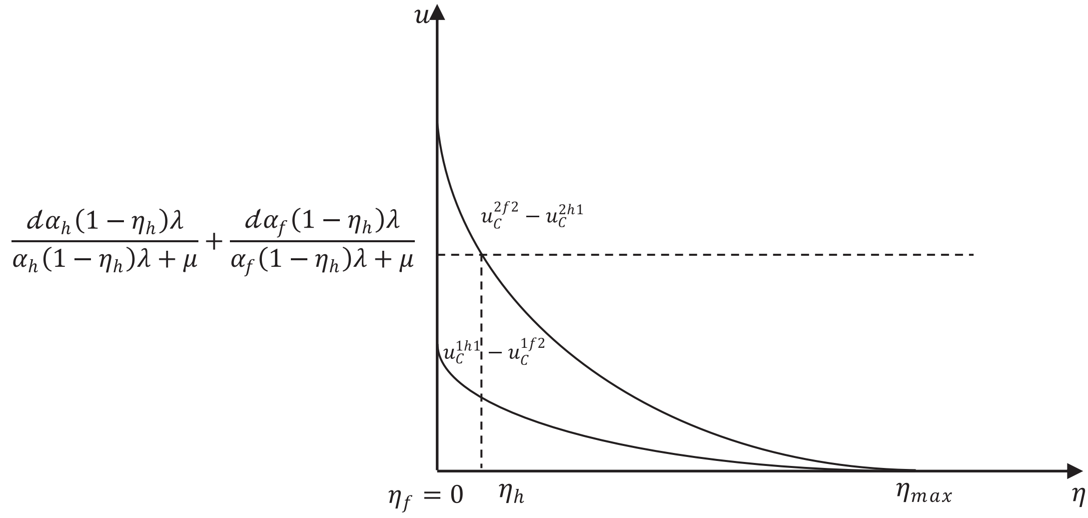

Fig. 3. Individuals' signaling and screening.

prevent (support) the government from implementing NPIs). We re-analysis the equilibrium: $((\eta_h, \eta_h), (a_1, a_1))$. From the signaling perspective, $((\eta_h, \eta_h), (a_1, a_1))$ can be hold. Combined with the government's budget constraints, if $\eta_h \in (0, \frac{1}{2})$, we find that only if $\zeta(1-\eta_h)s(t) - \varrho p_{hc}(t) \geq 0$, namely, $\eta_h \leq \frac{\zeta s(t) - \varrho p_{hc}(t)}{\zeta s(t)}$, the government would choose to implement NPIs. It means that increasing of η_h can result the government spending increasing. When $R_0 \leq 1$, we can have $\eta_h < 1$.

Corollary 1: Under pooling equilibrium $((\boldsymbol{\eta_f}, \boldsymbol{\eta_f}), (\boldsymbol{a}_2, \boldsymbol{a}_2))$, $\forall \boldsymbol{w},\ \boldsymbol{l} \in [0,\ 1]$, we have:

1. $\boldsymbol{\beta}_0 \in \left[0,\ \frac{(\gamma+\mu+v)(k+\mu)(\mu^2-\alpha_f\alpha_h)(\gamma+\mu)\lambda(1+d-2\xi)}{k[b(1-p)(\gamma+\mu+v)+p(\gamma+\mu)]\mu^2(1+d)}\right]$, only if $\boldsymbol{R}_0 \leq 1$;
2. $\boldsymbol{\beta}_0 \in \left(\frac{(\gamma+\mu+v)(k+\mu)(\mu^2-\alpha_f\alpha_h)(\gamma+\mu)\lambda(1+d-2\xi)}{k[b(1-p)(\gamma+\mu+v)+p(\gamma+\mu)]\mu^2(1+d)},\ +\infty\right)$, only if $\boldsymbol{R}_0 > 1$.

Under pooling equilibrium $((\boldsymbol{\eta_h}, \boldsymbol{\eta_h}), (\boldsymbol{a}_1, \boldsymbol{a}_1))$, $\forall \boldsymbol{w},\ \boldsymbol{l} \in [0,\ 1]$, we have:

1. $\boldsymbol{R_c} \in [0,\ 1-\boldsymbol{\eta_h}]$, if $\boldsymbol{R}_0 \leq 1$;
2. $\boldsymbol{R_c} \in (1-\boldsymbol{\eta_h},\ +\infty)$, if $\boldsymbol{R}_0 > 1$

Proof. See Appendix A.3. ■

Corollary 1 appears similar to the results of proposition 1, but the two findings are distinct. The results of the proposition are produced without proving the pooling equilibrium. In other words, we infer the optimal proportion of different types of individuals within which the disease can be controlled without knowing whether the government will intervene. Corollary 1 has a different logical starting point. It describes how disease can be controlled by a parameter when government is not involved and everyone is a freedom-centered individual. Corollary 1 has a clear description of the proportion of individual types and a clear description of whether the government chooses to implement NPIs. By corollary 1, we find that the control of the epidemic depends on the infection transmission rate $\boldsymbol{\beta}_0$ in the pooling equilibrium. $\boldsymbol{\beta}_0$ is a property of a disease that is not interfered by any non-drug factors. The findings of proposition 2 and Corollary 1 have interesting implications. We first show that when there are two different preferences (types) of individuals, the final equilibrium is that all individuals would choose freedom and the government does not impose strict NPIs. This will lead to two outcomes for an epidemic: if the virus has high infectiousness, namely, $\boldsymbol{\beta}_0 \in \left(\frac{(\gamma+\mu+v)(k+\mu)(\mu^2-\alpha_f\alpha_h)(\gamma+\mu)\lambda(1+d-2\xi)}{k[b(1-p)(\gamma+\mu+v)+p(\gamma+\mu)]\mu^2(1+d)},\ +\infty\right)$, then the Pareto optimal outcome is that everyone chooses to live with the virus together. If the virus has low infectiousness, namely, $\boldsymbol{\beta}_0 \in \left[0,\ \frac{(\gamma+\mu+v)(k+\mu)(\mu^2-\alpha_f\alpha_h)(\gamma+\mu)\lambda(1+d-2\xi)}{k[b(1-p)(\gamma+\mu+v)+p(\gamma+\mu)]\mu^2(1+d)}\right]$ then the Pareto optimal outcome is that the virus will eventually disappear.

From proposition 2, we know the optimal decision of government and individual. However, we do not know in what epidemic context individuals will make "health-centered" decisions. Choi and Shim [15] further explains why individuals send a particular signal. The economic intuition is also obvious. If we fixed the other parameters, an increase in $\boldsymbol{R}_0$ would represent a greater severity of the epidemic, and a rational individual would signal a response to the epidemic while ensuring that the risk of infection was minimized. In contrast, a decrease in $\boldsymbol{R}_0$ represents a reduction in epidemic size. Individuals find that the risks associated with freedom are consistent with the health risks associated with being confined. Therefore, rational individuals will choose freedom.

Corollaries 1 and the findings from Choi and Shim [15] answer our second research question: *What impact will government action have on the epidemic?* We summarize the analysis results of Corollary 1 the results from Choi and Shim

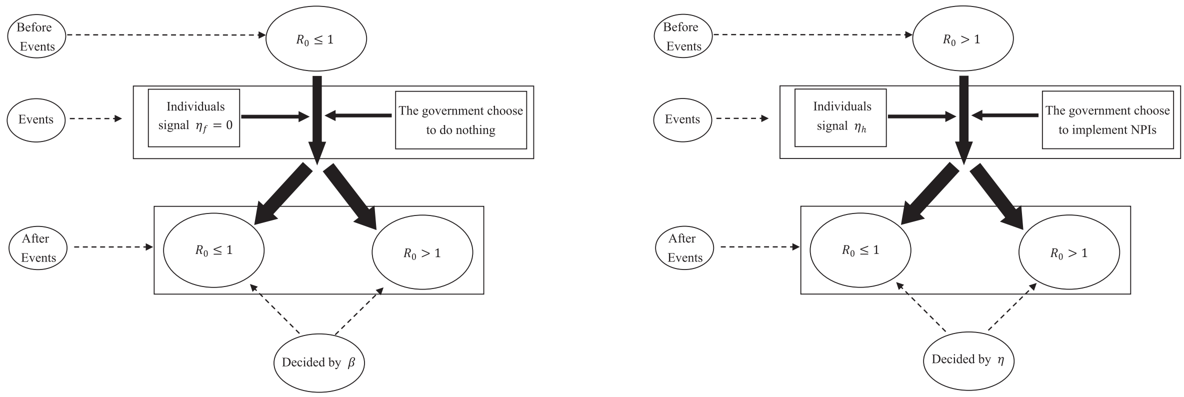

Fig. 4. (a) The impact of government and individual games on the epidemic when individual types are mixed to η_f. (b) The impact of government and individual games on the epidemic when individual types are mixed to η_h.

[15] through Fig. 4. Fig. 4(a) shows that at the beginning when the epidemic attribute is given: it could disappear naturally, individuals send η_f to indicate that they want freedom, and the government decides not to implement the restrictive policy. According to our analysis in corollary 1, there are two different effects of the individual-government game. Whether an epidemic can be contained is affected by the transmission contact rate β_0. Fig. 4(b) shows that at the beginning when the epidemic attribute is given: it could not disappear naturally, individuals send η_h to indicate that they want to be healthy, and the government decides to implement restrictive policies. When the government decides to intervene, whether or not the epidemic is contained depends on how strictly the government's restriction policy is applied. The more restrictive the policy, the better the epidemic can be contained.

Discussion

First, we get a value range of health-centered individual proportion through proposition 1. The number of health-centered individuals within a given range can lead a viral outbreak to die out. On the other hand, when the health-centered proportion is in that specific range, an epidemic can be expected. This finding is obtained by applying two key lemmas. By introducing different preferences (types) of individuals, we get the equilibrium from the individual versus the government game. We reach the first significant finding from proposition 2: pooling equilibria exist, but no separating equilibrium. This has important theoretical implications. The absence of a separating equilibrium implies that the two players in the game cannot distinguish between types by sending signals. Health-centered individuals could not reduce their own infection risk by expressing their opinions, and freedom-centered individuals could not express their opinions to decrease the likelihood of restrictive policies. Separating equilibrium is impossible in reality because government must implement one set of policies for the entire population and thus cannot fully satisfy both types completely.

Our second important finding is that there are two pooling equilibria: 1. Both types of individuals send η_h, and the government implements strict restrictive policies; 2. If both types of individuals send η_f, the government lifts the restrictive policy. These two situations are the most common in reality. The first pooling equilibrium appears in China in 2020–2022. China has adopted a zero-COVID-19 policy since 2020. The Chinese government conducted large-scale nucleic acid testing from 2020 to December 2022, quarantined close contacts, and imposed a strict lockdown that could span the entire city. During this period, China's strict restrictions were effective. According to Johns Hopkins University, only 16,000 people have died of COVID-19 in China during the three-year period. In the beginning, the public supported the government's approach, and many people were proud of the country's ability to control the pandemic [40–42]. However, as the virus has become more infectious, cases increased and restrictions became more onerous. Although initially effective, restrictive policies had a significant negative impact on people's mental health and income, and by 2022 there was a swing in public opinion against restrictive policies. This could be seen in an increase in protests and discontent expressed online [43–45]. The second pooling equilibrium is the current situation of all countries (China also announced lifting its coronavirus restrictions at the end of 2022). Since 2020, most countries have gone through a process from strict restrictive policies to relaxed NPIs. Due to the restrictive policies at the beginning, some countries even broke out anti-blockade demonstrations [12,13] . These anti-blockade demonstrations prove a conflict of preferences between two different types of individuals. It also shows that the government cannot simultaneously satisfy the preferences of two different types of people.

From a theoretical perspective, the main reason for the generation of pooling equilibrium is that the cost of signal transmission is too high, whether for health-centered or freedom-centered individuals. If the cost of sending health-centered (freedom-centered) signals is too high, health-centered individuals will not choose to send high η_h (low η_f). That is because the government set a standard to identify individuals as centered in health (freedom): Only if they send high η_h (low η_f) could they be considered health-centered (freedom-centered). As a result, high costs discourage signals from health-centered (freedom-centered) individuals, who think they would be better off sending lower (higher) η. The question worth considering is why the cost of sending signals needs to be lowered. The government's choice of policies is governed by the majority rule. A straightforward explanation is preference falsification, where individuals face incentives to hide their true preferences (i.e. not reveal their type) due to social pressure [46]. This type of public pressure is essentially a kind of cost, which can constrain the objective function of the actor. If most individuals are freedom-centered, health-centered individuals would find it costly to express their sincere preferences if it led to their neighbors labeling them as health-obsessed authoritarians. In addition to these social costs, protesting government policies is also costly in terms of time and effort. Since individuals can only expect to capture a small fraction of the expected benefit of such collective action, they are likely to remain silent when they perceive themselves to be in the minority. The same logic applies to the incentives of freedom-centered individuals when most of the population is health-centered.

Another possible explanation for preference falsification and pooling equilibrium can be found in social choice theory. Implemented social choice rules have three important properties: unanimity, Maskin monotonicity and no veto power [47,48]. They are defined as follows:

Definition 1 (unanimity): $\forall(\boldsymbol{a},\ \boldsymbol{x}) \in \boldsymbol{Z} \times \boldsymbol{X}$, if $\boldsymbol{a} \succcurlyeq_{\boldsymbol{i}}^{\boldsymbol{x}} \boldsymbol{b}$, $\forall \boldsymbol{i} \in \boldsymbol{N}$, $\forall \boldsymbol{b} \in \boldsymbol{Z}$, we have $\boldsymbol{a} \in \mathfrak{F}(\boldsymbol{x})$.

Definition 2 (Maskin monotonicity): Let $\boldsymbol{L_i}(\boldsymbol{a}, \boldsymbol{x}) = \{\boldsymbol{b} \in \boldsymbol{Z}\text{:}\ \boldsymbol{a} \succcurlyeq_{\boldsymbol{i}} \boldsymbol{b}\}$. If for any given two state $x,\ x' \in X$ and $\forall a \in \Im(x)$, $a \geq_i b$, so that $a \succcurlyeq_i b$, $\forall \boldsymbol{b} \in \boldsymbol{Z}$, we have: $\boldsymbol{a} \in \mathfrak{F}(\boldsymbol{x}')$.

Definition 3 (no veto power): $\forall(\boldsymbol{a},\ \boldsymbol{j},\ \boldsymbol{x}) \in \boldsymbol{Z} \times \boldsymbol{N} \times \boldsymbol{X}$, if $\boldsymbol{a} \succcurlyeq_{\boldsymbol{i}} \boldsymbol{b}$, $\forall \boldsymbol{i} \neq \boldsymbol{j}$, $\forall \boldsymbol{b} \in \boldsymbol{Z}$, we have $\boldsymbol{a} \in \mathfrak{F}(\boldsymbol{x})$.

Where $\boldsymbol{a},\ \boldsymbol{b} \in \boldsymbol{Z}$ represents the result space. This can be interpreted as the space formed by the result of the mechanism design. $\mathfrak{F}$ is the social choice function, which can be interpreted as the goal the designer hopes to achieve. Definition 1 means that if a social choice function is considered by everyone to be the best, it must be selected for implementation and become the socially optimal solution. Definition 2 means that if a scheme is selected by society at the beginning, and the change of state can only lead to the preference of individuals for this scheme, then this scheme must be selected by society again in the new state $\boldsymbol{x}'$. Definition 3 means that if an scheme is preferred by all but one individual, it must be selected. We can deduce the order: definition 3 ⇒ definition 1 ⇒ definition 2. These three definitions explain why people choose preference falsification. Because whether a few individuals like it or not, it will be implemented if the majority of individuals like it (definition 3). These few individuals know that their resistance is ineffective, and to reduce the cost of resistance, they choose preference falsification. When preference falsification occurs, definition 1 appears: When all individuals like the scheme, it must be implemented and is socially optimal. When it is executed, definition 2 tells us that if a scheme is considered the best in the original state, it must be selected again in the new state if it is still considered the best. That is why restriction policy cannot be re-implemented (the government will be reviled by its citizens if it reintroduces restrictions).

Mechanism design theory can also explain why individuals misreport their preferences. Under incomplete information, according to the Myerson-Satterthwaite theorem [43], there must be no effective decision, and satisfy the *ex ante* budget balance as well as the interim individual rationality constraints of Bayesian incentive compatible social selection rules. Under complete information, according to Gibbard-Satterthwaite theorem [44,45], as long as the social selection mechanism is not a dictatorship, then the mechanism must not be a surjective and truthfully implementable in dominant strategies. In other words, there is no mechanism for individuals to report the truth.

Pooling equilibrium cannot express individual preferences clearly. Thus, why not separating equilibrium? Previously we briefly analyzed the impossibility of separating equilibrium in reality. The condition of separating equilibrium is that the government can carry out different policies for two preferences in the same situation. For example, state governments can vary in their policies. New York has strict restrictions, and Florida has relaxed ones. However, this does not achieve a separating equilibrium except to the extent to which people are willing to move states on the basis of policy. Since citizen mobility will always be less than complete, a perfect separating equilibrium will not exist.

In a system of competitive federalism with free movement of individuals across state borders, however, the benefits of a separating equilibrium can be partially realized. The political advantage of a federal state is that states can pursue different policies rather than the same policies of the central government. If different states and local governments take different policy approaches and citizens base their location decisions partially on the policy mix of each locale, we will see sorting by individual type into different jurisdictions and greater alignment between individual preferences and government policy. This is known in the public finance and political economy literature as "Tiebout sorting" [49–51].

Such jurisdictional sorting will be far from perfect, however. In general, people are reluctant to leave their homes, and policy mix will be only one factor among many considered by people when they "vote with their feet." In the context of pandemic policy, there are additional complicating factors. For one, the success of public health policies in one location depends on similar efforts across jurisdictional borders or on preventing mobility across borders. Free movement of people across borders during a pandemic would thus undermine the objectives of health-centered individuals even when their state or local government enacted restrictive policies internally. Tiebout sorting cannot go very far towards a separating equilibrium in pandemic policy while an outbreak is occurring, but if citizens have already jurisdictionally sorted prior to an outbreak it will have some effect.

The above discussion speaks to research question 1. Corollaries 1 and findings from Choi and Shim [15] jointly answer the question of the impact of the game between government and individuals on the epidemic. When individuals are focused on freedom, government will not implement restrictive policies. The impact of this equilibrium on the epidemic does not depend on the strategies of either side but on the virus itself. If the virus is highly contagious, the epidemic cannot ultimately be contained; If the virus is weakly contagious, the epidemic will eventually die out naturally. In the context of

COVID-19, our conclusions can explain why COVID-19 eventually became a global epidemic. The game between the government and individuals under COVID-19 satisfies the pooling equilibrium we analyzed. From Fig. 4(a), we know that when the government does nothing and citizens demand freedom, the virus' infectivity decides whether the epidemic disappears. The infectivity of COVID-19 is the highest among known human viruses, and its $\boldsymbol{R}_0$ value reached 18.6 [52]. As a result, the epidemic caused by COVID-19 will continue.

On the contrary, the situation in China for the December 2020–2022 period fits into another pooling equilibrium we obtained: individuals are focused on health rather than freedom. As a result, the government chose to implement strict restrictions for three years, which resulted in a low death rate and a low number of confirmed cases of COVID-19 in China during the three years. China brought COVID-19 under control for three years. It closely supports our finding that when individuals send health signals, and governments implement NPIs, the containment of epidemics depends on the severity of restrictive policies.

We also find a strange phenomenon. The original COVID-19 virus' $\boldsymbol{R}_0 = 3.76$ [53]. According to Choi and Shim [15], when $\boldsymbol{R}_0 > 1$, individuals should send "health-centered" signals rather than "freedom-centered" signals. However, most countries worldwide have already begun to loosen restrictions by the end of 2020. The fundamental reason here is imperfect information. Our results are based on the assumption that individuals accurately assess the properties of the virus and respond appropriately (See Fig. 4(a) and (b)). This does not work in the real world. Although the relevant experts have a good understanding of the virus, most individuals will be ignorant and can be manipulated by misinformation and disinformation. It appears that many people globally underestimated the risks of the virus or held false beliefs about public health measures such as vaccination and masks [54].

For individuals to make sound and rational decisions, government information about the virus needs to be comprehensive, transparent, and trusted. Based on our budget-constraint assumptions for the government's assumptions, we can understand why the government might end the restrictions early (the main reason being that maintaining a high level of restrictions would lead to a budget deficit, which would worsen the recession). They become contradictory: If the government remains transparent about the outbreak, it can cause panic among individuals. Individuals will send "health-centered" signals. The government maintains a strict policy of restrictions. Nevertheless, the fiscal deficit results from the long period of restraint. How solve this contradiction (or finding an equilibrium between these two points) is a significant question for future research. A new research issue comes from [29,30,55], which proposes that SED is a critical way to solve social efficiency (welfare). In the case of public health emergencies, how to maximize social welfare through government decision-making is an important research direction in the future. SED is one of the potential solutions.

Conclusion

This paper analyzes whether the government should implement restrictive policies under different preferences. We have the following significant findings: There are two pooling equilibria. When all types of individuals send anti-epidemic signals, the government will adopt strict restrictions; Containment of the epidemic depends on the transmission infection rate and contact rate. When all individuals send signals that they want freedom, the government will abandon restrictive policies. if the virus transmission infection rate is too high, the epidemic will not be controlled and will continue to develop globally. If the virus transmission infection rate is low, the epidemic will eventually be contained. When all individuals send signals that they want health, the government can reduce the spread of the virus by limiting individual contact rates through strict policies. Thus, whether the epidemic is over depends on the severity of government restriction policies.

Our study has two important innovations: Theoretically, we extend the epidemiological theory by combining political economy theory with epidemiological theory. We combine the basic regeneration number of epidemiological theory with the results of game theory to find more realistic results. In practical terms, our research can provide guidance for policy during the ongoing COVID-19 pandemic and possible new epidemics in the future. Our findings could facilitate the development of public epidemiological management.

Appendix A

A.1 proof of proposition 1

Before we proof proposition 1, we first introduce 2 lemmas:

Lemma 1. *If $\boldsymbol{R}_0 \le 1$, then the DFE point is the unique solution of equations (2.11) – (2.17), and it is unique globally asymptotically stable equilibrium point [37]*

Lemma 2. *If $\boldsymbol{R}_0 > 1$, then the Disease perpetuation point is the unique solution of equations (2.11) – (2.17), and it is unique globally asymptotically stable equilibrium point [37].*

When $\boldsymbol{\eta_i} = 0$, $\forall \boldsymbol{i} \in \mathcal{T}$:

$$\boldsymbol{R}_0(0,\ 0) = \boldsymbol{\Xi}\left[\boldsymbol{\mu}\left(\boldsymbol{\alpha_f} + \boldsymbol{\alpha_h}\right) - 2\boldsymbol{\alpha_f}\boldsymbol{\alpha_h}\right] \le 1 \Rightarrow \boldsymbol{\mu}\left(\boldsymbol{\alpha_f} + \boldsymbol{\alpha_h}\right) - 2\boldsymbol{\alpha_f}\boldsymbol{\alpha_h} \le \frac{1}{\boldsymbol{\Xi}}$$

Similarly, when $\boldsymbol{R}_0(0,0) > 1$, we can have $\boldsymbol{\mu}(\boldsymbol{\alpha_f}+\boldsymbol{\alpha_h}) - 2\boldsymbol{\alpha_f}\boldsymbol{\alpha_h} > \frac{1}{\Xi}$. Using Lemma 1 and 2, we complete the proof of proposition 1.■

A.2 Proof of proposition 2

First, we proof the separating equilibrium (We have omitted the proof that contradicts our basic assumptions. readers can verify other contradictory situations if interesting). *If* t_i *chooses* $\boldsymbol{\eta_i}$*, namely, if Nature chooses the type* t_2 *for individuals, they would choose* $\boldsymbol{\eta_h}$*; if Nature chooses the type* t_2 *for individuals, they would choose* $\boldsymbol{\eta_f}$. if $\boldsymbol{u}_G^{1h1} \le \boldsymbol{u}_G^{1h2}$ and $\boldsymbol{u}_G^{2f1} \ge \boldsymbol{u}_G^{2f2}$, namely, $\begin{cases} \xi \le \left(\frac{\alpha_h(1-\eta_h)\lambda}{\alpha_h(1-\eta_h)\lambda+\mu} + \frac{\alpha_f(1-\eta_h)\lambda}{\alpha_f(1-\eta_h)\lambda+\mu}\right)(1+\boldsymbol{d}) \\ \frac{\alpha_f(1-\eta_f)\lambda}{\alpha_f(1-\eta_f)\lambda+\mu} + \xi \ge \left(\frac{\alpha_h(1-\eta_f)\lambda}{\alpha_h(1-\eta_f)\lambda+\mu} + \frac{\alpha_f(1-\eta_f)\lambda}{\alpha_f(1-\eta_f)\lambda+\mu}\right)(1+\boldsymbol{d}) \end{cases}$; $\boldsymbol{u}_C^{1h1} \le \boldsymbol{u}_C^{1f2}$ and $\boldsymbol{u}_C^{2f2} \le \boldsymbol{u}_C^{2h1}$, namely, $\begin{cases} 0 \le \frac{1-\eta_f}{\alpha_h(1-\eta_f)\lambda+\mu} \\ \frac{1-\eta_f}{\alpha_f(1-\eta_f)\lambda+\mu} \le 0 \end{cases} \Rightarrow \boldsymbol{\eta_f} = 1$. If the situation holds, it means $\boldsymbol{\eta_f} = 1$. It is impossible that all individuals signaling $\boldsymbol{\eta} = 1$ in the separating equilibrium. Thus, there is no separating equilibrium in situation 1.

Then we proof the mixed equilibrium. Suppose the strategy is mixed to $\boldsymbol{\eta_h}$, namely, if Nature chooses the type t_1 for individuals, they would choose $\boldsymbol{\eta_h}$; if Nature chooses the type t_2 for individuals, they also would choose $\boldsymbol{\eta_f}$. Thus, we have:

$$\begin{aligned}
\boldsymbol{a}^*(\boldsymbol{\eta_h}) &= \underset{\boldsymbol{a}}{\boldsymbol{argmin}}\{\boldsymbol{w}\boldsymbol{u_G}(\boldsymbol{\eta_h},\ \boldsymbol{a},\ t_1) + (1-\boldsymbol{w})\boldsymbol{u_G}(\boldsymbol{\eta_h},\ \boldsymbol{a},\ t_2)\} \\
&= \min_{\boldsymbol{a}}\left\{\left[\boldsymbol{w}\left(\boldsymbol{u}_G^{1h1}+\boldsymbol{u}_G^{2h1}\right)\right]_{\boldsymbol{a}_1}, \left[(1-\boldsymbol{w})\left(\boldsymbol{u}_G^{1h2}+\boldsymbol{u}_G^{2h2}\right)\right]_{\boldsymbol{a}_2}\right\} \\
&= \min_{\boldsymbol{a}}\left\{\left[2\boldsymbol{w}\boldsymbol{\xi}\right]_{\boldsymbol{a}_1}, \left[2(1-\boldsymbol{w})(1+\boldsymbol{d})\left(\frac{\boldsymbol{\alpha_h}(1-\boldsymbol{\eta_h})\boldsymbol{\lambda}}{\boldsymbol{\alpha_h}(1-\boldsymbol{\eta_h})\boldsymbol{\lambda}+\boldsymbol{\mu}} + \frac{\boldsymbol{\alpha_f}(1-\boldsymbol{\eta_h})\boldsymbol{\lambda}}{\boldsymbol{\alpha_f}(1-\boldsymbol{\eta_h})\boldsymbol{\lambda}+\boldsymbol{\mu}}\right)\right]_{\boldsymbol{a}_2}\right\}
\end{aligned}$$

$$\begin{aligned}
\boldsymbol{a}^*\left(\boldsymbol{\eta_f}\right) &= \underset{\boldsymbol{a}}{\boldsymbol{argmin}}\left\{\boldsymbol{w}\boldsymbol{u_G}\left(\boldsymbol{\eta_f},\ \boldsymbol{a},\ \boldsymbol{t}_1\right) + (1-\boldsymbol{w})\boldsymbol{u_G}\left(\boldsymbol{\eta_f},\ \boldsymbol{a},\ \boldsymbol{t}_2\right)\right\} \\
&= \min_{\boldsymbol{a}}\left\{\left[\boldsymbol{w}\left(\boldsymbol{u}_G^{1f1}+\boldsymbol{u}_G^{2f1}\right)\right]_{\boldsymbol{a}_1}, \left[(1-\boldsymbol{w})\left(\boldsymbol{u}_G^{1f2}+\boldsymbol{u}_G^{2f2}\right)\right]_{\boldsymbol{a}_2}\right\} \\
&= \min_{\boldsymbol{a}}\left\{\left[\boldsymbol{w}\left(\frac{\boldsymbol{\alpha_f}\left(1-\boldsymbol{\eta_f}\right)\boldsymbol{\lambda}}{\boldsymbol{\alpha_f}\left(1-\boldsymbol{\eta_f}\right)\boldsymbol{\lambda}+\boldsymbol{\mu}} + \frac{\boldsymbol{\alpha_h}\left(1-\boldsymbol{\eta_f}\right)\boldsymbol{\lambda}}{\boldsymbol{\alpha_h}\left(1-\boldsymbol{\eta_f}\right)\boldsymbol{\lambda}+\boldsymbol{\mu}} + 2\boldsymbol{\xi}\right)\right]_{\boldsymbol{a}_1},\right. \\
&\qquad \left.\left[2(1-\boldsymbol{w})(1+\boldsymbol{d})\left(\frac{\boldsymbol{\alpha_h}\left(1-\boldsymbol{\eta_f}\right)\boldsymbol{\lambda}}{\boldsymbol{\alpha_h}\left(1-\boldsymbol{\eta_f}\right)\boldsymbol{\lambda}+\boldsymbol{\mu}} + \frac{\boldsymbol{\alpha_f}\left(1-\boldsymbol{\eta_f}\right)\boldsymbol{\lambda}}{\boldsymbol{\alpha_f}\left(1-\boldsymbol{\eta_f}\right)\boldsymbol{\lambda}+\boldsymbol{\mu}}\right)\right]_{\boldsymbol{a}_2}\right\}
\end{aligned}$$

Under the situation, the above equations should comply with the following conditions:

$$\begin{cases} (1-\boldsymbol{w})(1+\boldsymbol{d})\left(\frac{\alpha_h(1-\eta_h)\lambda}{\alpha_h(1-\eta_h)\lambda+\mu} + \frac{\alpha_f(1-\eta_h)\lambda}{\alpha_f(1-\eta_h)\lambda+\mu}\right) \le w\xi \\ w\left(\frac{\alpha_f(1-\eta_f)\lambda}{\alpha_f(1-\eta_f)\lambda+\mu} + \frac{\alpha_h(1-\eta_f)\lambda}{\alpha_h(1-\eta_f)\lambda+\mu} + 2\boldsymbol{\xi}\right) \le 2(1-\boldsymbol{w})(1+\boldsymbol{d})\left(\frac{\alpha_h(1-\eta_f)\lambda}{\alpha_h(1-\eta_f)\lambda+\mu} + \frac{\alpha_f(1-\eta_f)\lambda}{\alpha_f(1-\eta_f)\lambda+\mu}\right) \end{cases}$$

We have $\boldsymbol{w} \in \left[0,\ \frac{(1+\boldsymbol{d})\left(\frac{\alpha_h(1-\eta_h)\lambda}{\alpha_h(1-\eta_h)\lambda+\mu} + \frac{\alpha_f(1-\eta_h)\lambda}{\alpha_f(1-\eta_h)\lambda+\mu}\right)}{\xi+(1+\boldsymbol{d})\left(\frac{\alpha_h(1-\eta_h)\lambda}{\alpha_h(1-\eta_h)\lambda+\mu} + \frac{\alpha_f(1-\eta_h)\lambda}{\alpha_f(1-\eta_h)\lambda+\mu}\right)}\right] \cap \left[\frac{(1+\boldsymbol{d})\left(\frac{\alpha_h(1-\eta_f)\lambda}{\alpha_h(1-\eta_f)\lambda+\mu} + \frac{\alpha_f(1-\eta_f)\lambda}{\alpha_f(1-\eta_f)\lambda+\mu}\right)}{(2+\boldsymbol{d})\left(\frac{\alpha_f(1-\eta_f)\lambda}{\alpha_f(1-\eta_f)\lambda+\mu} + \frac{\alpha_h(1-\eta_f)\lambda}{\alpha_h(1-\eta_f)\lambda+\mu}\right)+\xi},\ 1\right)$.

Situation 1: if $\boldsymbol{u}_C^{1h1} \le \boldsymbol{u}_C^{1f1}$ and $\boldsymbol{u}_C^{2f1} \ge \boldsymbol{u}_C^{2h1}$, namely, $\begin{cases} \frac{1-\eta_f}{\alpha_h(1-\eta_f)\lambda+\mu} \ge 0 \\ \frac{1-\eta_f}{\alpha_f(1-\eta_f)\lambda+\mu} \ge 0 \end{cases}$, we have: $\begin{cases} \boldsymbol{a}^*(\boldsymbol{\eta_h}) = \boldsymbol{a}_1 \\ \boldsymbol{a}^*(\boldsymbol{\eta_f}) = \boldsymbol{a}_1 \end{cases}$. We can obtain:

$$\begin{cases} \boldsymbol{u_C}(\boldsymbol{\eta_h},\ \boldsymbol{a}^*(\boldsymbol{\eta}),\ \boldsymbol{t}_1) = \boldsymbol{u}_C^{1f1} \ge \boldsymbol{u_C}\left(\boldsymbol{\eta_f},\ \boldsymbol{a}^*(\boldsymbol{\eta}),\ \boldsymbol{t}_1\right) = \boldsymbol{u}_C^{1h1} \\ \boldsymbol{u_C}(\boldsymbol{\eta_h},\ \boldsymbol{a}^*(\boldsymbol{\eta}),\ \boldsymbol{t}_2) = \boldsymbol{u}_C^{2f1} \ge \boldsymbol{u_C}\left(\boldsymbol{\eta_f},\ \boldsymbol{a}^*(\boldsymbol{\eta}),\ \boldsymbol{t}_2\right) = \boldsymbol{u}_C^{2h1} \end{cases}$$

We verify the posterior probability of the government in the $\boldsymbol{\eta_f}$ information set, suppose, the posterior probability is $\boldsymbol{l}$:

$$\boldsymbol{l} \times \boldsymbol{u}_G^{1f1} + (1-\boldsymbol{l}) \times \boldsymbol{u}_G^{2f1} \le \boldsymbol{u}_G^{1f2} \times \boldsymbol{l} + (1-\boldsymbol{l}) \times \boldsymbol{u}_G^{2f2} \Rightarrow \boldsymbol{l} \le \frac{\frac{\alpha_h(1-\eta_f)\lambda}{\alpha_h(1-\eta_f)\lambda+\mu} + \frac{\alpha_f(1-\eta_f)\lambda}{\alpha_f(1-\eta_f)\lambda+\mu}}{\frac{\alpha_h(1-\eta_f)\lambda}{\alpha_h(1-\eta_f)\lambda+\mu} - \frac{\alpha_f(1-\eta_f)\lambda}{\alpha_f(1-\eta_f)\lambda+\mu}}(1+\boldsymbol{d})$$

$$-\frac{\frac{2\alpha_f(1-\eta_f)\lambda+\mu}{\alpha_f(1-\eta_f)\lambda+\mu}}{\frac{\alpha_h(1-\eta_f)\lambda}{\alpha_h(1-\eta_f)\lambda+\mu} - \frac{\alpha_f(1-\eta_f)\lambda}{\alpha_f(1-\eta_f)\lambda+\mu}}$$

Under the situation, $\boldsymbol{\alpha_f} = 0$. Combined with the government budget constraint, only if $\boldsymbol{\eta_h} \in (\frac{1}{2},\ 1)$, there is a pooling perfect Bayesian Nash equilibrium $((\boldsymbol{\eta_h},\ \boldsymbol{\eta_h}),\ (\boldsymbol{a}_1,\ \boldsymbol{a}_1);\ \boldsymbol{w} \in \left[0,\ \frac{1+\boldsymbol{d}}{\boldsymbol{\xi}+(1+\boldsymbol{d})}\right] \cap \left[\frac{(1+\boldsymbol{d})\left(\frac{\boldsymbol{\alpha_h}(1-\boldsymbol{\eta_f})\boldsymbol{\lambda}}{\boldsymbol{\alpha_h}(1-\boldsymbol{\eta_f})\boldsymbol{\lambda}+\boldsymbol{\mu}}\right)}{(2+\boldsymbol{d})\left(\frac{\boldsymbol{\alpha_h}(1-\boldsymbol{\eta_f})\boldsymbol{\lambda}}{\boldsymbol{\alpha_h}(1-\boldsymbol{\eta_f})\boldsymbol{\lambda}+\boldsymbol{\mu}}\right)+\boldsymbol{\xi}},\ 1\right),\ \boldsymbol{l} \in [0, 1+\boldsymbol{d})\ )$.

Situation 2: if $\boldsymbol{u_C^{1h1}} \geq \boldsymbol{u_C^{1f1}}$ and $\boldsymbol{u_C^{2f1}} \leq \boldsymbol{u_C^{2h1}}$, namely, $\begin{cases} \frac{\boldsymbol{\alpha_h}(1-\boldsymbol{\eta_f})\boldsymbol{\lambda}}{\boldsymbol{\alpha_h}(1-\boldsymbol{\eta_f})\boldsymbol{\lambda}+\boldsymbol{\mu}} \leq 0 \\ \frac{\boldsymbol{\alpha_f}(1-\boldsymbol{\eta_f})\boldsymbol{\lambda}}{\boldsymbol{\alpha_f}(1-\boldsymbol{\eta_f})\boldsymbol{\lambda}+\boldsymbol{\mu}} \leq 0 \end{cases} \Rightarrow \boldsymbol{\eta_f} = 1$, we have: $\begin{cases} \boldsymbol{a}^*(\boldsymbol{\eta_h}) = \boldsymbol{a}_1 \\ \boldsymbol{a}^*(\boldsymbol{\eta_f}) = \boldsymbol{a}_1 \end{cases}$. We can obtain:

$$\begin{cases} \boldsymbol{u_C}(\boldsymbol{\eta_h},\ \boldsymbol{a}^*(\boldsymbol{\eta}),\ \boldsymbol{t}_1) = \boldsymbol{u_C^{1h1}} \geq \boldsymbol{u_C}\left(\boldsymbol{\eta_f},\ \boldsymbol{a}^*(\boldsymbol{\eta}),\ \boldsymbol{t}_1\right) = \boldsymbol{u_C^{1f1}} \\ \boldsymbol{u_C}(\boldsymbol{\eta_h},\ \boldsymbol{a}^*(\boldsymbol{\eta}),\ \boldsymbol{t}_2) = \boldsymbol{u_C^{2h1}} \geq \boldsymbol{u_C}\left(\boldsymbol{\eta_f},\ \boldsymbol{a}^*(\boldsymbol{\eta}),\ \boldsymbol{t}_2\right) = \boldsymbol{u_C^{2f1}} \end{cases}$$

However, if $\boldsymbol{\eta_f} = 1$, the government budget constraint is less than 0. Thus, we can conclude that there is no mixed perfect Bayesian Nash equilibrium in situation 2.

Suppose the strategy is mixed to $\boldsymbol{\eta_f}$, namely, if Nature chooses the type t_1 for individuals, they would choose $\boldsymbol{\eta_f}$; if Nature chooses the type t_2 for individuals, they also would choose $\boldsymbol{\eta_f}$. Thus, we have:

$$\begin{aligned} \boldsymbol{a}^*(\boldsymbol{\eta_h}) &= \underset{\boldsymbol{a}}{\boldsymbol{argmin}}\{\boldsymbol{w}\boldsymbol{u_G}(\boldsymbol{\eta_h},\ \boldsymbol{a},\ t_1) + (1-\boldsymbol{w})\boldsymbol{u_G}(\boldsymbol{\eta_h},\ \boldsymbol{a},\ t_2)\} = \min_{\boldsymbol{a}}\left\{\left[\boldsymbol{w}\left(\boldsymbol{u_G^{1h1}} + \boldsymbol{u_G^{2h1}}\right)\right]_{\boldsymbol{a}_1}, \left[(1-\boldsymbol{w})\left(\boldsymbol{u_G^{1h2}} + \boldsymbol{u_G^{2h2}}\right)\right]_{\boldsymbol{a}_2}\right\} \\ &= \min_{\boldsymbol{a}}\left\{\left[2\boldsymbol{w}\boldsymbol{\xi}\right]_{\boldsymbol{a}_1}, \left[2(1-\boldsymbol{w})(1+\boldsymbol{d})\left(\frac{\boldsymbol{\alpha_h}(1-\boldsymbol{\eta_h})\boldsymbol{\lambda}}{\boldsymbol{\alpha_h}(1-\boldsymbol{\eta_h})\boldsymbol{\lambda}+\boldsymbol{\mu}} + \frac{\boldsymbol{\alpha_f}(1-\boldsymbol{\eta_h})\boldsymbol{\lambda}}{\boldsymbol{\alpha_f}(1-\boldsymbol{\eta_h})\boldsymbol{\lambda}+\boldsymbol{\mu}}\right)\right]_{\boldsymbol{a}_2}\right\} \end{aligned}$$

$$\begin{aligned} \boldsymbol{a}^*\left(\boldsymbol{\eta_f}\right) &= \underset{\boldsymbol{a}}{\boldsymbol{argmin}}\left\{\boldsymbol{w}\boldsymbol{u_G}\left(\boldsymbol{\eta_f},\ \boldsymbol{a},\ \boldsymbol{t}_1\right) + (1-\boldsymbol{w})\boldsymbol{u_G}\left(\boldsymbol{\eta_f},\ \boldsymbol{a},\ \boldsymbol{t}_2\right)\right\} \\ &= \min_{\boldsymbol{a}}\left\{\left[\boldsymbol{w}\left(\boldsymbol{u_G^{1f1}} + \boldsymbol{u_G^{2f1}}\right)\right]_{\boldsymbol{a}_1}, \left[(1-\boldsymbol{w})\left(\boldsymbol{u_G^{1f2}} + \boldsymbol{u_G^{2f2}}\right)\right]_{\boldsymbol{a}_2}\right\} \\ &= \min_{\boldsymbol{a}}\left\{\left[\boldsymbol{w}\left(\frac{\boldsymbol{\alpha_f}\left(1-\boldsymbol{\eta_f}\right)\boldsymbol{\lambda}}{\boldsymbol{\alpha_f}\left(1-\boldsymbol{\eta_f}\right)\boldsymbol{\lambda}+\boldsymbol{\mu}} + \frac{\boldsymbol{\alpha_h}\left(1-\boldsymbol{\eta_f}\right)\boldsymbol{\lambda}}{\boldsymbol{\alpha_h}\left(1-\boldsymbol{\eta_f}\right)\boldsymbol{\lambda}+\boldsymbol{\mu}} + 2\boldsymbol{\xi}\right)\right]_{\boldsymbol{a}_1},\right. \\ &\qquad \left.\left[2(1-\boldsymbol{w})(1+\boldsymbol{d})\left(\frac{\boldsymbol{\alpha_h}\left(1-\boldsymbol{\eta_f}\right)\boldsymbol{\lambda}}{\boldsymbol{\alpha_h}\left(1-\boldsymbol{\eta_f}\right)\boldsymbol{\lambda}+\boldsymbol{\mu}} + \frac{\boldsymbol{\alpha_f}\left(1-\boldsymbol{\eta_f}\right)\boldsymbol{\lambda}}{\boldsymbol{\alpha_f}\left(1-\boldsymbol{\eta_f}\right)\boldsymbol{\lambda}+\boldsymbol{\mu}}\right)\right]_{\boldsymbol{a}_2}\right\} \end{aligned}$$

Under the situation, the above equations should comply with the following conditions:

$$\begin{cases} (1-\boldsymbol{w})(1+\boldsymbol{d})\left(\frac{\boldsymbol{\alpha_h}(1-\boldsymbol{\eta_h})\boldsymbol{\lambda}}{\boldsymbol{\alpha_h}(1-\boldsymbol{\eta_h})\boldsymbol{\lambda}+\boldsymbol{\mu}} + \frac{\boldsymbol{\alpha_f}(1-\boldsymbol{\eta_h})\boldsymbol{\lambda}}{\boldsymbol{\alpha_f}(1-\boldsymbol{\eta_h})\boldsymbol{\lambda}+\boldsymbol{\mu}}\right) \geq w\xi \\ w\left(\frac{\boldsymbol{\alpha_f}(1-\boldsymbol{\eta_f})\boldsymbol{\lambda}}{\boldsymbol{\alpha_f}(1-\boldsymbol{\eta_f})\boldsymbol{\lambda}+\boldsymbol{\mu}} + \frac{\boldsymbol{\alpha_h}(1-\boldsymbol{\eta_f})\boldsymbol{\lambda}}{\boldsymbol{\alpha_h}(1-\boldsymbol{\eta_f})\boldsymbol{\lambda}+\boldsymbol{\mu}} + 2\boldsymbol{\xi}\right) \geq 2(1-\boldsymbol{w})(1+\boldsymbol{d})\left(\frac{\boldsymbol{\alpha_h}(1-\boldsymbol{\eta_f})\boldsymbol{\lambda}}{\boldsymbol{\alpha_h}(1-\boldsymbol{\eta_f})\boldsymbol{\lambda}+\boldsymbol{\mu}} + \frac{\boldsymbol{\alpha_f}(1-\boldsymbol{\eta_f})\boldsymbol{\lambda}}{\boldsymbol{\alpha_f}(1-\boldsymbol{\eta_f})\boldsymbol{\lambda}+\boldsymbol{\mu}}\right) \end{cases}$$

We have $\boldsymbol{w} \in \left[0,\ \frac{(1+\boldsymbol{d})\left(\frac{\boldsymbol{\alpha_h}(1-\boldsymbol{\eta_f})\boldsymbol{\lambda}}{\boldsymbol{\alpha_h}(1-\boldsymbol{\eta_f})\boldsymbol{\lambda}+\boldsymbol{\mu}} + \frac{\boldsymbol{\alpha_f}(1-\boldsymbol{\eta_f})\boldsymbol{\lambda}}{\boldsymbol{\alpha_f}(1-\boldsymbol{\eta_f})\boldsymbol{\lambda}+\boldsymbol{\mu}}\right)}{(2+\boldsymbol{d})\left(\frac{\boldsymbol{\alpha_f}(1-\boldsymbol{\eta_f})\boldsymbol{\lambda}}{\boldsymbol{\alpha_f}(1-\boldsymbol{\eta_f})\boldsymbol{\lambda}+\boldsymbol{\mu}} + \frac{\boldsymbol{\alpha_h}(1-\boldsymbol{\eta_f})\boldsymbol{\lambda}}{\boldsymbol{\alpha_h}(1-\boldsymbol{\eta_f})\boldsymbol{\lambda}+\boldsymbol{\mu}}\right)+\boldsymbol{\xi}}\right] \cap \left[\frac{(1+\boldsymbol{d})\left(\frac{\boldsymbol{\alpha_h}(1-\boldsymbol{\eta_h})\boldsymbol{\lambda}}{\boldsymbol{\alpha_h}(1-\boldsymbol{\eta_h})\boldsymbol{\lambda}+\boldsymbol{\mu}} + \frac{\boldsymbol{\alpha_f}(1-\boldsymbol{\eta_h})\boldsymbol{\lambda}}{\boldsymbol{\alpha_f}(1-\boldsymbol{\eta_h})\boldsymbol{\lambda}+\boldsymbol{\mu}}\right)}{\boldsymbol{\xi}+(1+\boldsymbol{d})\left(\frac{\boldsymbol{\alpha_h}(1-\boldsymbol{\eta_h})\boldsymbol{\lambda}}{\boldsymbol{\alpha_h}(1-\boldsymbol{\eta_h})\boldsymbol{\lambda}+\boldsymbol{\mu}} + \frac{\boldsymbol{\alpha_f}(1-\boldsymbol{\eta_h})\boldsymbol{\lambda}}{\boldsymbol{\alpha_f}(1-\boldsymbol{\eta_h})\boldsymbol{\lambda}+\boldsymbol{\mu}}\right)},\ 1\right)$.

Situation 1: if: $\boldsymbol{u_C^{1h1}} \geq \boldsymbol{u_C^{1f1}}$ and $\boldsymbol{u_C^{2f1}} \leq \boldsymbol{u_C^{2h1}}$, namely, $\begin{cases} 0 \geq \frac{1-\boldsymbol{\eta_f}}{\boldsymbol{\alpha_h}(1-\boldsymbol{\eta_f})\boldsymbol{\lambda}+\boldsymbol{\mu}} \\ \frac{1-\boldsymbol{\eta_f}}{\boldsymbol{\alpha_f}(1-\boldsymbol{\eta_f})\boldsymbol{\lambda}+\boldsymbol{\mu}} \leq 0 \end{cases}$. It is impossible that $\boldsymbol{\eta_f} = 1$ can be hold, since $\boldsymbol{\eta_f} \in [0, \frac{1}{2})$. Thus, we can conclude that there is no mixed perfect Bayesian Nash equilibrium in situation 2

Situation 2: if: $\boldsymbol{u_C^{1h2}} \geq \boldsymbol{u_C^{1f2}}$ and $\boldsymbol{u_C^{2f2}} \leq \boldsymbol{u_C^{2h2}}$, namely, $\begin{cases} \frac{1-\boldsymbol{\eta_h}}{\boldsymbol{\alpha_h}(1-\boldsymbol{\eta_h})\boldsymbol{\lambda}+\boldsymbol{\mu}} \geq \frac{1-\boldsymbol{\eta_f}}{\boldsymbol{\alpha_h}(1-\boldsymbol{\eta_f})\boldsymbol{\lambda}+\boldsymbol{\mu}} \\ \frac{1-\boldsymbol{\eta_h}}{\boldsymbol{\alpha_f}(1-\boldsymbol{\eta_h})\boldsymbol{\lambda}+\boldsymbol{\mu}} \leq \frac{1-\boldsymbol{\eta_f}}{\boldsymbol{\alpha_f}(1-\boldsymbol{\eta_f})\boldsymbol{\lambda}+\boldsymbol{\mu}} \end{cases} \Rightarrow \boldsymbol{\eta_f} = \boldsymbol{\eta_h} = 0$. We have: $\begin{cases} \boldsymbol{a}^*(\boldsymbol{\eta_h}) = \boldsymbol{a}_2 \\ \boldsymbol{a}^*(\boldsymbol{\eta_f}) = \boldsymbol{a}_2 \end{cases}$. We can obtain:

$$\begin{cases} \boldsymbol{u_C}\left(\boldsymbol{\eta_f},\ \boldsymbol{a}^*(\boldsymbol{\eta}),\ t_1\right) = \boldsymbol{u_C^{1h2}} \geq \boldsymbol{u_C}(\boldsymbol{\eta_h},\ \boldsymbol{a}^*(\boldsymbol{\eta}),\ t_1) = \boldsymbol{u_C^{1f2}} \\ \boldsymbol{u_C}\left(\boldsymbol{\eta_f},\ \boldsymbol{a}^*(\boldsymbol{\eta}),\ t_2\right) = \boldsymbol{u_C^{2h2}} \geq \boldsymbol{u_C}(\boldsymbol{\eta_h},\ \boldsymbol{a}^*(\boldsymbol{\eta}),\ t_2) = \boldsymbol{u_C^{2f2}} \end{cases}$$

We verify the posterior probability of the government in the $\boldsymbol{m}_1$ information set, suppose, the posterior probability is $\boldsymbol{l}$, we can deduce that there exists an equilibrium, if the following inequation can be hold

$$\boldsymbol{u_G^{1h2}} \times \boldsymbol{l} + (1-\boldsymbol{l}) \times \boldsymbol{u_G^{2h2}} \leq \boldsymbol{l} \times \boldsymbol{u_G^{1h1}} + (1-\boldsymbol{l}) \times \boldsymbol{u_G^{2h1}} \Rightarrow \left(\frac{\boldsymbol{\alpha_h}(1-\boldsymbol{\eta_h})\boldsymbol{\lambda}}{\boldsymbol{\alpha_h}(1-\boldsymbol{\eta_h})\boldsymbol{\lambda}+\boldsymbol{\mu}} + \frac{\boldsymbol{\alpha_f}(1-\boldsymbol{\eta_h})\boldsymbol{\lambda}}{\boldsymbol{\alpha_f}(1-\boldsymbol{\eta_h})\boldsymbol{\lambda}+\boldsymbol{\mu}}\right)(1+\boldsymbol{d}) \leq 2\boldsymbol{\xi}$$

Under the situation, $\alpha_h = 0$. Thus, from the above inequation, we can also have: $\alpha_f \le \frac{\mu(1+d)}{\lambda(1+d-2\xi)}$. Only if $\alpha_f \le \frac{\mu(1+d)}{\lambda(1+d-2\xi)}$, the equilibrium can exist. There is a mixed perfect Bayesian Nash equilibrium $\left((\eta_f,\ \eta_f),\ (a_2,\ a_2);\ w \in \left[0,\ \frac{(1+d)\frac{\alpha_f\lambda}{\alpha_f\lambda+\mu}}{(2+d)(\frac{\alpha_f\lambda}{\alpha_f\lambda+\mu})+\xi}\right] \cap \left[\frac{(1+d)}{\xi+(1+d)},\ 1\right)\right)$, $\forall l \in [0,\ 1]$. ■

A.3 Proof of Corollary 1

Using proposition 2, we have only two pooling equilibria.

$$R_c = \frac{\mu\left[\alpha_f\left(1-\eta_f\right)+\alpha_h(1-\eta_h)\right]+\alpha_f\alpha_h\left(\eta_f+\eta_h-2\right)}{\mu\left[\alpha_f+\alpha_h\right]-2\alpha_f\alpha_h}R_0$$

When the pooling equilibrium $((\eta_f,\eta_f),(a_2,a_2))$ exists, we can obtain the condition: $\eta_h = \eta_f = 0$. Then, we have $R_c = R_0$ and $R_0(0,0) = \Xi\{\mu[\alpha_f + \alpha_h] - 2\alpha_f\alpha_h\}$. Since under the pooling equilibrium, $\alpha_h = 0$ and $\alpha_f = \frac{\mu(1+d)}{\lambda(1+d-2\xi)}$; $R_0(0,\ 0) = \Xi\,\frac{\mu^2(1+d)}{\lambda(1+d-2\xi)}$. If $R_0 \le 1$, we have $\Xi\,\frac{\xi\mu^2}{\lambda(1-\xi)} \le 1 \Rightarrow \beta_0 \in \left[0,\ \frac{(\gamma+\mu+\nu)(k+\mu)(\mu^2-\alpha_f\alpha_h)(\gamma+\mu)\lambda(1+d-2\xi)}{k[b(1-p)(\gamma+\mu+\nu)+p(\gamma+\mu)]\mu^2(1+d)}\right]$; if $R_0 > 1$, we have $\beta_0 \in \left(\frac{(\gamma+\mu+\nu)(k+\mu)(\mu^2-\alpha_f\alpha_h)(\gamma+\mu)\lambda(1+d-2\xi)}{k[b(1-p)(\gamma+\mu+\nu)+p(\gamma+\mu)]\mu^2(1+d)},\ +\infty\right)$.

When the pooling equilibrium $((\eta_h,\eta_h),(a_1,a_1))$ exists, we can have the following inequations: if $R_0 \le 1$, we have $R_c \in [0,\ 1-\eta_h]$; if $R_0 > 1$, we have $R_c \in (1-\eta_h,\ +\infty)$. ■

A.4 Proof of Corollary 2

We need to show in what situation $q = \frac{1}{1+\mu}$ can be hold. Notice that according to proposition 1, when $R_0 \le 1$, $h_0(t) = \frac{1}{1+\mu}$, and $h(t) = \frac{H(t)}{N(t)}$. It represents the share of health-centered individuals in the general population. Thus, we get $q = h_0(t)$. It says health-centered individuals share the same share of sus individuals as they do in the general population.■

Appendix B

Table B1.

Table B1
Parameter and variable description.

Parameters and variables	Description
Ω	Epidemiology state space
$S(t) \subset \mathbb{R}^+$	Susceptible
$H(t) \subset \mathbb{R}^+$	Health-centered
$F(t) \subset \mathbb{R}^+$	Freedom-centered
$E(t) \subset \mathbb{R}^+$	Exposed
$I(t) \subset \mathbb{R}^+$	Symptomatic
$A(t) \subset \mathbb{R}^+$	Asymptomatic
$R(t) \subset \mathbb{R}^+$	Recovered
$N(t) \subset \mathbb{R}^+$	Total population
$\{\eta_i\}_{i\in\mathcal{T}} \in [0,\ 1]$	Reduction contact rate
$\gamma \in [0,\ 1]$	Recover rate
$\mu \in \mathbb{R}^+$	Natural death rate
$\nu \in [0,\ 1]$	Hospitalization rate
$\{\alpha_i\}_{i\in\mathcal{T}} \in [0,\ 1]$	The number of ith type individuals who express their type per unit of time
$\beta_0 \in \mathbb{R}^+$	Transmission infection rate
$b \in \mathbb{R}^+$	The rate of relative infectiousness of asymptomatic cases compared to symptomatic cases
T	Individuals' type set
$\varrho \in \mathbb{R}^+$	Marginal cost for public health per individuals

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