



Optimal lockdown and vaccination policies to contain the spread of a mutating infectious disease

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Abstract

We develop a piecewise deterministic control model to study optimal lockdown and vaccination policies to manage a pandemic. Lockdown is modeled as an impulse control that allows the decision maker to switch from one level of restrictions to another. Vaccination policy is a continuous control. Decisions are taken under the risk of mutations of the disease, with repercussions on the transmission rate. The decision maker follows a cost minimization objective. We first characterize the optimality conditions for impulse control and show how the prospect of a mutation affects the decision maker's choice by inducing her to anticipate the net benefit of operating under a different lockdown state once a mutation occurs. The problem admits infinitely many value functions. Under some parametric conditions, we show the existence of a minimum value function that is a natural candidate solution. Focusing on this specific value function, we finally study the features of the optimal policy, especially the timing of impulse control. We prove that uncertainty surrounding future “bad” versus “good” mutation of the disease expedites versus delays the adoption of lockdown measures.

Keywords Pandemic · Lockdown · Vaccination · Mutation · Impulse control · Uncertainty

JEL Classification C61 · D81 · I18

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1 Introduction

The onset of the COVID-19 pandemic marked the renewed interest of economists for the analysis of the impacts of epidemics and the design of public policies to cope with them. This is best illustrated by the impressive number of papers that have been published on these topics over the last couple of years. Most contributions in Economics throughout the first year of the pandemic have been devoted to the analysis of lockdown, or quarantine or more generally restriction measures, as the main instrument to control the evolution of the epidemic. This was of course primarily motivated by the absence of alternative intervention (before the discovery of vaccines, and in the absence of cure). But even after the widespread use of vaccination, lockdown measures remain a credible policy tool in the eyes of policy-makers, and are considered as such in the current paper.

Specifically, our aim is to develop an original piecewise deterministic optimal control model to study optimal lockdown and vaccination policies to manage an infectious disease. Our approach is original in that it combines the following three main ingredients. First, we consider two alternatives to control the spread of the pandemic: vaccination and lockdown. Second, we take lockdown measures as impulse controls to echo the evidence that policy makers are subject to a wide set of (administrative, political, economic) constraints that prevent them from changing the level of restrictions on a daily basis. Last but not least, we account for the uncertainty that surrounds the severity of the disease and results from the frequent mutations of the virus. As perfectly outlined by Boucekkine et al. (2021), this together with policy interventions, are the fundamental drivers of the rate of transmission of the disease, and resulting economic and health impacts. In this setting, our primary concern is to study the impact of mutations—modeled as random and discrete shifts in the virus contagiousness—on health policy. We especially seek to determine whether lockdown should be used as a prevention policy in the prospect of handling better a future (potentially more severe) mutation, or as an adaptation policy to such event, once and if realized. In addition, we are interested in the effect of possible mutation on the timing of the lockdown policy: does the prospect of a mutation delay, or on the contrary expedite, the lockdown policy? The final issue we want to address is about the interplay between the vaccination and lockdown policies: Are they substitute or complement tools in the hands of the policy makers?

It goes without saying that any attempt to provide a comprehensive review of the literature on epidemics and COVID-19 would be a vain exercise given how fast it grows.¹ Rather, we more reasonably give an overview of both the economic “classics”, that is, the pre-COVID reference papers merging epidemiological and economic models, and the most recent post-COVID papers relevant to our problem.

Gersovitz and Hammer (2004) are the first to investigate the connection between the spread of an infectious disease and economic outcomes. By comparing a representative

¹ There is of course a vast literature on epidemics in mathematical biology, that is left aside here. This is because this literature, by contrast with the one in economics and operations research, does not incorporate decision problems and optimization. It generally focuses on the pandemic dynamics and on the features of endemic vs disease-free steady states.

agent problem with the optimal solution, they discuss how standard economic instruments work to internalize the externalities surrounding the epidemic propagation. In a series of excellent papers, Goenka and Liu (2012, 2020) and Goenka et al. (2014) develop full-fledged analyses of the impact of an epidemic on the macro-economy. For that purpose, they consider several versions of a model merging SIS dynamics and neoclassical growth. Goenka and Liu (2012) focus on the impact of the pandemic on endogenous labor supply, show how the pandemic dynamics can generate chaos and cycles, and discuss the type of policy intervention capable of stabilizing endogenous fluctuations. Goenka et al. (2014) go a step further by taking account of the two-way interaction between the economy and the pandemic. On the one hand, the disease negatively affects the (exogenous here) labor supply and production, while health expenditure are meant to slow down the virus transmission. Both papers adopt a social planner perspective by characterizing either the optimal growth path, or the optimal policy. Finally, in a similar framework, Goenka and Liu (2020) analyze the decentralized equilibrium when private agents also invest in human capital. They emphasize the existence of multiple balanced growth paths, with very distinct features in terms of economic performance and disease prevalence.

As to the post-COVID-19 lature, our attention was drawn to the contributions that consider lockdown measures as the single instrument to control the evolution and severity of the epidemic situation. Alvarez et al. (2021) is an excellent representative of this line of research. The authors study the optimal control of a pandemic thanks to lockdown restrictions. They consider lockdown as a continuous control, the decision maker choosing the intensity of the lockdown, or the share of population subject to a lockdown, at every instant. They capture the basic trade-off between the economic cost and health benefit of lockdown (reduction of the transmission rate). In addition, they model the pandemic dynamics with the SIR model, and adopt a short run perspective by assuming that the planning horizon is finite but unknown as it is determined by the arrival of a vaccine. They investigate the features—timing, duration and intensity—of the optimal policy both analytically and by means of a calibrated model. Another very interesting piece of work is due to Dobson et al. (2023). The authors develop an hybrid economic-epidemiological model to analyze the optimal control of a pandemic, when in its early stage, when instruments available to the policy makers are lockdown, testing and isolation. One important result is that combining testing with isolation is more effective in terms of the balance between health and economic impacts, than lockdowns. Other related papers differ from this approach along several ways.² The most relevant to our analysis are those *i/* that depart from the modeling of lockdown

² Goenka et al. (2022) study optimal lockdown by accounting for the waning immunity (thereby using a SIRS model). They keep working with a neoclassical growth model and focus on long run outcomes. Loerstcher and Miur (2021) do not take an optimal policy perspective. Rather they assume that the decision maker's objective is to make sure that the health system capacity cannot be overwhelmed because of the increase in the number of infected people requiring special care. The limited capacity constraint of the health system is also present in Jones et al. (2021), where the authors compare the representative agent equilibrium with the optimal solution. They use an aggregate transmission rate defined as a function of consumption and working decisions and analyze the reactions, in terms of social distancing and remote working, following the announcement of an outbreak of the pandemic. See Eichenbaum et al. (2021) for a similar approach but with different perspective. Caulkins et al. (2021) also consider the optimal design of lockdown policy by incorporating several novelties such as dealing with the level of economic activity and lockdown "fatigue" as additional states of the system. The analysis of the quite complex dynamics

as a continuous control, and *ii*/ that introduce uncertainty in the analysis. Aspri et al. (2021) observe that in the real world, policies such as lockdown cannot be revised instantaneously and further, cannot be revised before a certain amount time elapses. Based on this observation, they study the optimal design of lockdown policies, when available policies remain constant along some time interval, within a SEIRAD model (that is they also take care of the asymptomatic health status). In a more standard model, Caulkins et al. (2020) also choose to deal with lockdown policies as impulse rather than continuous controls. Adopting this perspective, they are able to address the issue of the optimal timing, onset and exit, of a one-shot lockdown regime. This is also the perspective adopted by Huberts and Thijssen (2023), in a deterministic SIR model. Finally, there also exist a few recent contributions that consider vaccination as an alternative to lockdown restrictions, to control a pandemic. For instance, Federico et al. (2022) develop a comprehensive analysis of the optimal vaccination policy within a SIRS model and show the conditions under which this policy succeeds in eradicating the disease eventually.

As mentioned above, uncertainty seems like an essential feature not only for the management of pandemic situations, but also and more generally for the understanding of both individuals behaviors and policy performance in many economic problems (ranging from finance, to investment and resource management problems). Yet there are very few studies that have incorporated this dimension into the analysis of optimal policy to fight against COVID-19. Exceptions are Gollier (2020b), Bandyopadhyay et al. (2021), Federico and Ferrari (2021), and La Torre et al. (2023). Gollier (2020b) studies the impact of uncertainty surrounding the transmission rate when lockdown restrictions are lifted and the role of learning about it on the optimal lockdown policy within a two-stage decision problem. He shows that introducing uncertainty tends to reduce the optimal rate of lockdown by lowering the expected cost of a less strict lockdown. Bandyopadhyay et al. (2021) conduct the same kind of analysis in terms of uncertainty and learning in a three-period problem. They consider that imposing a lockdown prevents the decision maker from learning about the actual contagiousness of the disease. Moreover, they introduce an additional cost of delaying lockdown, namely the lost opportunity of habit formation.³ On the other hand, Federico and Ferrari (2021) develop a stochastic optimal control problem where the decision maker has to choose the lockdown policy while being subject to an uncertainty not about the level but the evolution of the transmission rate. For that purpose, they model the dynamics of the transmission rate thanks to a diffusion (Wiener) process and take lockdown as a means to reduce the trend (deterministic part) of the process. In this setting, they conduct numerical experiments that help highlight the features of the lockdown policy. Focusing on treatment measures affecting the spread of a disease,

Footnote 2 continued

generated by their model notably show the existence of multiple Skiba points. It is also worth mentioning the contribution of Acemoglu et al. (2021) who develop a multi-group SIR model where the population is divided into three age classes (youth, middle age, old) to assess the performance of targeted lockdown policy. Gollier (2020a) investigates the very same topic. He does not analyze the optimal solution, though. Rather he considers two types of lockdown interventions, strong vs. softer.

³ Their argument being that habit formation should normally take place during a lockdown and give people incentives to behave more cautiously once it ends.

La Torre et al. (2023) look at the optimal policy when accounting for the occurrence of random shocks. These shocks correspond to new virus strains that affect both the growth rate and number of infected people. Interestingly, the probability of realization of these shocks is endogenous and depends on the number of infectives, which is the state of the system. In this setting, they characterize the stochastic steady state, i.e., the invariant distribution of the disease over a positive support, and notably show how the intensity of treatment measures shapes this distribution.

In sum, there is no study that combines lockdown as an impulse control, uncertainty surrounding the evolution of the transmission rate, and consider both lockdown and vaccination policies. This is exactly where the contribution of our paper lies. Precisely, we first develop a stochastic optimal control model to study optimal lockdown and vaccination policies in pandemic times. Lockdown is modeled as an impulse control that allows the decision maker to switch from one level of restrictions to another (stricter or softer). Vaccination policy, on the other hand, is a continuous control. Decisions are taken under the risk of mutations of the disease, with repercussions on the transmission rate. The decision maker follows a cost minimization objective. Considering a simplified model where the virus can mutate only once and there exist only two lockdown levels, with the possibility to go back and forth between them, allows us to draw a series of interesting results. We first characterize the optimality conditions for impulse control and show how the prospect of a mutation affects the decision maker's choice. In fact, it induces her to anticipate the relative benefit of a regime change after a mutation has occurred, which may or may not increase the incentive to set a lockdown. Our problem admits infinitely many value functions. Under some parametric conditions, we show the existence of a minimum value function that is a natural solution candidate to the solution. Focusing on this specific value function, we finally study the features of the optimal policy, especially the timing of impulse control. We prove that uncertainty about future possible "bad" vs. "good" mutation (in terms of contagiousness) of the disease tends to expedite vs. delay the adoption of lockdown measures. This conclusion closely parallels those of the two strands of literature on decision making under uncertainty. The first one analyzes the impact of the occurrence of random costly events (Crepin 2020, for a review), whereas the second one emphasizes the role of uncertainty, irreversibility, and learning (Dixit and Pindyck 1994). Finally, a numerical analysis based on Huberts and Thijssen (2023)'s calibration highlights the impact of the risk of (a bad) mutation on the optimal management of the pandemic. Lockdown can be worth before mutation only, when the share of infected population is quite low. Combined with high vaccination rates, it allows the decision maker to control the pace at which the virus spreads. However, lockdown measures, if any, are lifted eventually, as the decision maker is able to manage the pandemic by means of vaccination only.

The paper is organized as follows. Section 2 is devoted to our modeling strategy. Section 3 deals with the optimality conditions for impulse controls and emphasizes the difference of deciding before or after a mutation. Section 4 analyzes the impact of the risk of mutation on lockdown measures. Section 5 addresses the issue of the interplay between lockdown, reopening and the evolution of the pandemic. Section 6 conducts a numerical analysis based on the calibration of the model, and Sect. 7 concludes.

2 Model

We adopt the fully centralized perspective of a decision maker (DM) who has to manage a pandemic (as in Goenka and Liu 2012; Alvarez et al. 2021; Federico and Ferrari 2021, among others). It means that we do not address externality problems associated with the disease transmission. It sounds like a natural starting point for the study of the optimal management of epidemics.

2.1 Pandemic evolution and policy interventions

Most analyses of the optimal control of an epidemic make use of SIS and SIR models, and some variations of these. The main pre-COVID-19 contributions, that merge epidemiological and economic models, were mainly based on the SIS model (Goenka and Liu 2012; Goenka et al. 2014). In the SIS model, the (constant) population is split into two health states, the “susceptible” and the “infected,” and corresponding state equations describe changes in the health status. Early papers following the COVID-19 outbreak rather rely on the SIR model, whereby upon infection people get full immunity and enter the third “recovery” status, or die (Acemoglu et al. 2021; Jones et al. 2021). More recent contributions use the more general SIRS model to account for a waning immunity. In the latter model, recovered people become susceptible again after a while (Caulkins et al. 2021; Goenka et al. 2022).

Which is the best model to use is a difficult question because of the usual trade-off between realism and tractability. However, nearly three years after the onset of the pandemic, we have learnt a few lessons from COVID-19. One advantage of the SIR model, over the SIS, is that it takes account of disease induced mortality. But since we do not account for demographic aspects and the average death rate by COVID-19 is very low, the SIS alternative may seem more attractive. Indeed, it allows us to keep the state space one-dimensional. In addition, there is clear evidence that immunity acquired through an infection lasts for a short period of time only. This makes the standard SIR model inadequate to reproduce the dynamics of pandemic such as COVID-19. One may then opt for the more general SIRS model. Here again the SIS model looks like an acceptable simplification of the problem because of the tractability argument.

The last important point is how to incorporate vaccination policy. In epidemiological models, the vaccination control usually shows up in the state equation of the susceptible, either as a rate proportional to the state (Bolzoni et al. 2017; Di Giambardino and Iacoviello 2017) or as an absolute rate (Barrett and Hoel 2007). Then either vaccinated people become an additional state of the system (Choi and Shim 2020), or join the recovery state, with waning immunity (Federico et al. 2022). Now, we know that COVID-19 vaccines do not obtain long lasting immunity, especially in the event of a mutation of the virus. Moreover, if they are efficient to protect the populations at risk from severe forms of the disease, they poorly protect the general population against infection. So, in effect, and as long as one does not capture the severity dimension, the vaccine works as a treatment that allows for containing the spread of the disease.

Based on these observations, we abstract away from mortality, recovery, and develop a model whose features are alike a SIS model with treatment. Precisely, the dynamics

reduce to a single logistic differential equation in the state of infected, $I(t) \in [0, 1]$, of the following form⁴:

$$\dot{I}(t) = \theta(t)I(t) \left[1 - r(t) - I(t) - \frac{K}{\theta(t)} \right], \quad (1)$$

where $\theta(t)$ is the infection (transmission) rate, K is the quality of the health system (recovery rate), and $r(t) \geq 0$ is the (effective) vaccination rate. The DM controls the vaccination rate, and can progressively make people get vaccinated by means of a wide set of more or less coercive measures and incentives (Geoffard and Philipson 1997). Unlike the typical approach in the literature, vaccination applies to the state of infected because it is ultimately taken as a means to control the evolution of the disease. The quality of the health system encompasses both the qualitative and quantitative dimensions of hospital infrastructure and medical staff that, of course, partly determine the ease with which a country can cope with the pandemic.⁵

The key variable in Eq.(1) is the transmission rate of the pandemic, $\theta(t) \in M$, a finite or countable set. This rate changes across time thanks to the DM's decision of lockdown and virus mutation. This echoes Jones et al. (2021)'s observation that "infected people transmit the virus to susceptible people at a rate that depends on the nature of the virus and the frequency of social interactions."

The DM can decide on the lockdown level (of restriction) imposed upon the economy, L_k , with $k \in \mathcal{K}$, a countable set. This decision is modeled as an impulse control. Therefore when to make a change and to which level of restriction are controlled by the DM. The reason for this modeling option is twice. Most of the literature on lockdown defines this policy as a continuous control (Acemoglu et al. 2021; Alvarez et al. 2021; Goenka et al. 2022). This means that the DM chooses, at every instant, the intensity of the lockdown, or the share of the population subject to it.⁶ However, it is clear that policy makers cannot adjust such policy decisions on a daily basis. This is perfectly acknowledged by Aspri et al. (2021) who argue that policies are not adjusted instantaneously, and further that there exists some inertia. Considering lockdown measures as impulse controls follows this observation. This is also the avenue taken by Caulkins et al. (2020). This is finally the option chosen by Huberts and Thijssen (2023) who model lockdown controls, and related costs and benefit, exactly the same way as we do.

Moreover, about three years after its outbreak, it is fair to say that many (sanitary, economic) uncertainties have surrounded and continue to surround the COVID-19 pandemic. However most contributions on the optimal design of lockdown policies get rid of uncertainty (or at best conduct sensitivity analyses of calibrated models).

⁴ This is a very convenient feature especially if one wants to derive analytical results, which is the aim of the paper.

⁵ The role of the quality and/or capacity of the health system has been analyzed in a series of contributions by Goenka et al. (2014), Caulkins et al. (2021), Jones et al. (2021) and Loerstcher and Miur (2021), among others. In the coming analysis, it is taken as given because we choose to focus on policies and shocks affecting the infection rate. So we do not consider any other type of control capable of changing K , like investment in the health system. But this is a potential interesting extension of the analysis.

⁶ This introduces an additional quadratic term in the dynamical system, which is crucial to understand the effect of such a policy on the epidemic and the economy.

Noticeable exceptions are Gollier (2020b), Bandyopadhyay et al. (2021) and Federico and Ferrari (2021). In different settings, the first two contributions account for the uncertainty in the transmission rate and analyze how uncertainty and the possibility of learning about the exact severity of the disease shape policy intervention. Federico and Ferrari (2021) model the transmission rate as a random variable whose evolution is driven by a stochastic process (where lockdown measures affect the trend part of it). Dealing with the random evolution of the disease seems to be very important, again in light of the COVID situation. We however believe that significant virus mutations, that is occurrences of new variants, are better described by jump processes, as they do not occur continuously but at certain points in time.⁷ Accordingly, let us denote as Z_j the j th mutation or virus variant, with $j \in \mathcal{J}$, another countable set. The set M thus comprises values of transmission rate, θ_{jk} , associated with mutations Z_j (alpha, delta, omicron, etc. for COVID-19) and lockdown levels L_k .⁸

In the remainder of the analysis, a regime is defined by a pair (Z_j, L_k) and is associated with a single infection rate $\theta_{jk} \in M$. We also take θ as a second state variable, whose evolution is then driven by either mutation or impulse control.⁹

2.2 Regime change by mutation and impulse control

Hereafter, we precisely explain how the θ can change as a result of random mutations and DM's actions.

Regime change by mutation We consider that the DM, when designing her policy, may be subject to two kinds of uncertainty. They have to do with both the severity and the evolution of the pandemic. Specifically, we take account of the possibility that the virus mutates, while spreading across the population, with repercussions on its infectious power. As a result, there is uncertainty surrounding not only the time when a mutation occurs, but also the nature of this mutation (contagiousness of mutated virus). We model mutations in the framework of piecewise-deterministic process, which has been set up in, e.g., Davis (1984) and Vermes (1985). An excellent exposition can be found in Chapter 8 of Dockner et al. (2000). Following Davis (1984), Section 3, let $X_t = (\theta(t), I(t))$ represent the state of the piecewise deterministic process for any time $t \geq t_0$. The state space is defined by

$$E = M \times [0, 1].$$

⁷ There exists a quite vast literature in mathematical Biology that analyzes the features of epidemiological models with mutations. There are two ways to incorporate mutations. Either they result from the time varying nature of human interactions that implies that infection rates are also varying, thus mutating (Gracy et al. 2021). Or, in multi-strain models, mutations capture switches, for infected people; from one strain of the virus to another (Dobie 2022; Martcheva 2009; Meehan et al. 2018). These are different perspectives that do not deal with mutations as random processes.

⁸ We can list the elements of M in the form of a $|\mathcal{J}| \times |\mathcal{K}|$ matrix whose j th row consists of the θ_{jk} for $k \in \mathcal{K}$ etc.

⁹ There is an analogy between our approach (see the Eq. 1) and the representation of capital accumulation in the stochastic Ramsey model. In this model, the state equation for the capital stock depends on two state variables, capital itself and the TFP, which follows an exogenous stochastic process (typically a stationary AR1 process). The resolution is based upon discretization in a finite numbers of "regimes" of values for the TFP (see, for example, Tauchen 1990).

Let \mathcal{B} denote the Borel sets of $[0, 1]$. Define \mathcal{E} as

$$\mathcal{E} = \{(\theta, A) : \theta \in M, A \in \mathcal{B}\}.$$

Then (E, \mathcal{E}) is a Borel space. The probability law of $X = (X_t)_{t \geq t_0}$ is determined by the following objects:

1. Vector fields $(\mathcal{H}_\theta, \theta \in M)$ associated with the differential equation (1). For each $\theta \in M$ the vector field \mathcal{H}_θ determines a unique integral curve $\phi_\theta(t, I_0)$ satisfying (1) and with the initial value $I(t_0) = I_0$.
2. A measurable function $\lambda : E \mapsto \mathbb{R}_+$ which defines a survival function F_θ by

$$F_\theta(t) = \exp \left[- \int_{t_0}^t \lambda(\theta, \phi_\theta(\tau, I_0)) d\tau \right].$$

3. A transition measure $Q : M \times E \mapsto [0, 1]$. For any $\theta \in M$, $Q(\theta, \cdot)$ is a measurable function and for any $X_t \in E$, $Q(\cdot, X_t)$ is a probability distribution over M .

For given lockdown level L_k , the motion of the process X proceeds as follows. Starting from the initial state $X_0 = (\theta_0, I_0) \in E$, with $\theta(t_0) = \theta_0$, the trajectory of the infected population follows $\phi_{\theta_0}(t, I_0)$ until a mutation occurs at some date t_1 , with the probability

$$\Pr [t_1 > t] = F_{\theta_0}(t).$$

At t_1 the state jumps to regime $\theta(t_1) \in M$ with the probability distribution $Q(\cdot, X_{t_1})$. The value of I at jump is unchanged, i.e.,

$$I(t_1) = \phi_{\theta_0}(t_1, I_0).$$

Starting from $X_{t_1} = (\theta(t_1), I(t_1))$ the process repeats so that next inter-jump time $t_2 - t_1$ and the post-jump location $(\theta(t_2), I(t_2))$ are selected in a similar way. This gives a piecewise deterministic trajectory $X = (X_t)_{t \geq t_0}$ with jump times t_1, t_2, \dots . In the rest of this paper, for the piecewise deterministic stochastic processes X , we use the natural filtration $\mathbf{F} := (\mathcal{F}_t^X)_{t \geq t_0}$.

Regime change by impulse control Not only mutations cause regime changes, but also the DM’s impulse action regarding the lockdown level. The two kinds of change have similar impacts on the system. Changing the restriction level, $L_m \rightarrow L_n$, with $m, n \in \mathcal{K}$, also affects the transmission rate of the disease, θ . By convention, we say that lockdown regime L_n is stricter than L_m if and only if $L_m < L_n$ and the diffusion rate θ is smaller in a stricter state. This is consistent with evidence that imposing a stricter lockdown allows health authorities to reduce the infection rate and slow down the virus diffusion. For given virus mutation, Z_j , lockdown level L_m , and initial state of the system $X_0 = (\theta_0, I_0)$, if the DM chooses to change the lockdown to another level L_n at date T_1 , then this results in a new regime $\theta(T_1) \neq \theta_0$ etc.

To sum up, the mappings from E into itself by mutations and impulse controls can be categorized as follows. A jump caused by mutation from Z_j to Z_i , $j, i \in \mathcal{J}$, $j \neq i$,

leads to the mapping $(\theta_{jk}, I) \rightarrow (\theta_{ik}, I)$ in E if the mutation occurs when the system operates under L_k . On the other hand, a jump caused by the impulse control $L_m \rightarrow L_n$ changes (θ_{jm}, I) to (θ_{jn}, I) in E .

2.3 Objective function

To capture the cost-benefit analysis underlying the choice of the vaccination and lockdown policies under a pandemic, we follow the vast majority of the literature by adopting a cost minimization perspective (see, for instance Aspri et al. 2021; Federico and Ferrari 2021; Gollier 2020b). The DM wants to minimize the total discounted cost of the pandemic. The instantaneous cost is regime dependent and typically encompasses two dimensions. First, policy interventions, through vaccination and lockdown measures, are costly. Second, the economy incurs a cost that is increasing in the number of infected people. The trade-off associated with the vaccination policy is as follows: Vaccination policy is costly because it requires to purchase vaccine doses, and develop temporary vaccination infrastructure (see Barrett 2003, for a review of vaccination costs). But it allows the DM to reduce the social cost of sickness by reducing the spread of the pandemic.¹⁰ The benefit of the lockdown policy is of the same nature, while its economic cost depends on the level of restrictions, and captures expenditure to take care of people working remotely or no longer working, the fact that the level of economic activity shrinks etc.

Following again the literature (Di Giamberardino and Iacoviello 2017; Okosun et al. 2011; Huberts and Thijssen 2023), we shall work with a separable quadratic cost function:

$$C(r, L_k, I) = \frac{r^2}{2} + h_k + \beta \frac{I^2}{2}, \quad (2)$$

where $h_k \geq 0$ captures the constant economic cost inherent in operating under lockdown level L_k . Beside the instantaneous cost of lockdown, we also introduce a lump-sum (sunk) cost incurred when switching the lockdown state from L_m to L_n . This cost actually depends on the intensity of the change, captured by the difference $L_n - L_m$, and is denoted by $\Gamma(L_n - L_m) \equiv \Gamma_n \geq 0$. It is in essence of political or social nature. Huberts and Thijssen (2023) provide justifications for considering this additional cost. Arguably, there exists asymmetry between imposing a lockdown measure and removing the very same measure that we should take into account when modeling lockdown as an impulse control. Moreover, this is in the line with the “lockdown fatigue” approach developed by Caulkins et al. (2021), or with the argument of “political backlash” put forward by Bandyopadhyay et al. (2021).

¹⁰ Infected people incur both economic (reduced income) and psychological costs. They may also impose a cost to society, especially where the health care system is publicly funded.

Within any regime θ , characterized by a pair (L_k, Z_j) , holding at instant t , the general optimization problem can be written as:

$$V(\theta, I) = \min_{r, \{(T_i, L(T_i))\}_{i \geq 1}, t \leq T_i \leq \infty} \mathbb{E}_Z \left\{ \sum_i e^{-\rho(T_{i-1}-t)} \left[\int_{T_{i-1}}^{T_i} C(r, L(T_{i-1}), I) e^{-\rho(\tau-T_{i-1})} d\tau + \Gamma_i e^{-\rho(T_i-T_{i-1})} \right] \right\}$$

where the expectation operator refers to random mutations, $T_0 = t, L(T_0) = L_k, \Gamma_i$ is the lump-sum cost associated with the change in intensity of lockdown from $L(T_{i-1})$ to $L(T_i)$, the lockdown level chosen at $T_i, \rho > 0$ is the discount rate, and $V(\cdot)$ is the value function. The optimization is subject to (1) with initial state $X_t = (\theta(t), I(t)), \theta(t) = \theta$ and $I(t) = I$, given.

At this stage, one can notice that our problem is very similar to Huberts and Thijssens (2023)’s analysis of the optimal timing of lockdown measures during a pandemic. However, our approach is original in that it combines both impulse controls (which they do consider), that is controlled regime shifts, and random shifts (which they don’t). So we study a stochastic counterpart of the problem and consider an additional continuous control.¹¹

On the mathematical side, it is clear that this model of controlled piecewise deterministic Markov process is a controlled deterministic dynamical system plus a compounded Poisson jumping process. Specifically, the evolution of the state of the system, $X_t = (\theta(t), I(t))$, between consecutive impulse controls follows

$$d \begin{pmatrix} \theta(t) \\ I(t) \end{pmatrix} = \begin{pmatrix} 0 \\ \theta(t)I(t) (1 - r(t) - I(t)) - I(t)K \end{pmatrix} dt + \begin{pmatrix} q(\theta(t), I(t)) \\ 0 \end{pmatrix} dJ_t,$$

for $T_i < t < T_{i+1}$, for some function $q(\theta, I)$, and where

$$J_t = \sum_{i=1}^{N_t} Y_i,$$

is a compound Poisson process where the jump sizes Y_i are i.i.d random variables, and N_t is a Poisson process with jump intensity λ . This is a special case of controlled stochastic processes with jump diffusion. Therefore, well-developed methods for the latter can be applied for mathematical analysis of the processes. In particular, Chapter 8 in Okensdal and Sulem (2007) provides an excellent theoretical framework and analytical techniques, such as the existence of viscosity solutions and the verification theorem.

Before moving to the analysis, one should note that this paper aims at studying the interplay between the two relevant characteristics of the optimal control of pandemics,

¹¹ These novelties comes at the expense of the description of the disease dynamics: they use a SIR(S) model, while we stick to a SIS model.

i.e., vaccination and lockdown. Given this objective, we now present a series of simplifications that will help us to gain analytical insights into the fundamentals drivers of the solution. We restrict the analysis to a limited number of pandemic and lockdown states. We assume that there exist two variants of the virus only. For ease of notation, under lockdown level L_k , we denote these variants and corresponding transmission rates as \bar{Z} , \underline{Z} , $\bar{\theta}_k$ and $\underline{\theta}_k$. Without loss of generality, whatever the lockdown level, the pandemic situation under \bar{Z} is supposed to be worst than under \underline{Z} , which implies that $\bar{\theta}_k > \underline{\theta}_k$. More importantly, we assume that mutation can occur at most once. In addition, we also consider that the DM can choose between two lockdown levels only, so $\mathcal{K} = \{m, n\}$ and the variation of intensity of lockdown is given.¹² Thus, \mathcal{M} consists of four regimes, $\{\bar{\theta}_m, \bar{\theta}_n, \underline{\theta}_m, \underline{\theta}_n\}$. This means that we switch-off the second source of uncertainty (when a mutation occurs, its nature is known).¹³ We allow the DM to go back and forth between these two restriction levels, L_m and L_n . Moreover, we take $\lambda(\theta, I)$ as a positive constant: $\lambda(\theta, I) = \lambda$. Finally, hereafter, the function $\bar{V}_k(I)$ (resp., $\underline{V}_k(I)$) denotes the value function in regime $\bar{\theta}_k$ (resp., $\underline{\theta}_k$), for $k \in \mathcal{K}$. When keeping track of the virus variant is not required, we use the generic notations θ_k , $V_k(I)$ etc. to refer to a regime with lockdown level L_k .

3 Optimality conditions

We first state the optimality conditions for both continuous and impulse control, particular attention being paid to the ones characterizing the latter. All of the proofs are gathered in the Appendices.

3.1 Continuous, impulse controls and the quasi variation inequality

For any state I , regime θ , corresponding to the pair (L_m, \bar{Z}) , either stays as it is or switches to a new one by DM's impulse control or virus mutation. If no regime changes happen, then it must be that (a) the value function satisfies the Hamilton-Jacobi-Bellman (HJB) equation, and (b) regime change is not profitable for the DM. Therefore, in a regime with (L_m, \bar{Z}) , by dynamic programming, the stationary value function $\bar{V}_m(I)$ satisfies the inequality

$$\rho \bar{V}_m(I) \leq \bar{H}_m^*(I, \bar{V}'_m(I)) \quad (3)$$

where

¹² Huberts and Thijssen (2023) actually analyze the same case in the first part of their paper. It allows to simply oppose two situations: The one in which the economy is locked-down and the one where it is "opened."

¹³ Some results of this special case can be generalized to the more general case where the nature of mutation is unknown (see "Appendix A.9").

$$\begin{aligned} \bar{H}_m^* (I, \bar{V}'_m(I)) &= I \bar{V}'_m(I) [\bar{\theta}_m (1 - I) - K] \\ &\quad - \frac{[\bar{\theta}_m I \bar{V}'_m(I)]^2}{2} + h_m + \frac{\beta}{2} I^2 \end{aligned} \tag{4}$$

is the current value minimized Hamiltonian associated with optimal vaccination rate¹⁴

$$\bar{r}_m^* = \bar{V}'_m (I) \bar{\theta}_m I. \tag{5}$$

For the solution (5) to be well-behaved, one must have $\bar{V}'_m (I) \geq 0$. Then, how exactly the vaccination rate changes with I depends on the particular shape of the value function. If it is convex, then the larger the infection rate, the larger the vaccination rate. In addition, any impulse control $L_m \rightarrow L_n$, satisfying $L_m < L_n$ (case of a stricter lockdown), will induce a drop in the vaccination rate. This is the first evidence of the substitutable nature of the two controls.

The DM does not take the impulse control $L_m \rightarrow L_n$ if doing so is not profitable. Hence, L_m continues if

$$\bar{V}_m (I) \leq \bar{V}_n (I) + \Gamma_n. \tag{6}$$

Since at least one of the inequalities (3)-(6) must hold true, we can formulate the quasi-variational inequality (QVI) associated with the decision problem in regime (L_m, \bar{Z}) :

$$\max \left\{ \rho \bar{V}_m (I) - \bar{H}_m^* (I, \bar{V}'_m (I)), \bar{V}_m (I) - \bar{V}_n (I) - \Gamma_n \right\} = 0 \quad \text{for } I \in (0, 1). \tag{7}$$

Similarly, we get a second QVI characterizing regime (L_m, \underline{Z}) :

$$\begin{aligned} \max \left\{ (\rho + \lambda) \underline{V}_m (I) - \underline{H}_m^* (I, \underline{V}'_m (I), \lambda \bar{V}_m (I)), \underline{V}_m (I) - \underline{V}_n (I) - \Gamma_n \right\} &= 0 \\ \text{for } I \in (0, 1). \end{aligned} \tag{8}$$

with $\underline{H}_m^*(.)$ the current value minimized Hamiltonian corresponding to this regime.

These QVIs are equivalent to the HJB equations holding for standard optimal control without impulse. Their boundary conditions are given at the switching state. More precisely, at a value I_n^* where the impulse control $L_m \rightarrow L_n$ is taken, we have

¹⁴ The minimized vaccine rate r_k^* , in lockdown state L_k and for any virus state, satisfies

$$r_k^* = V'_k (I) \theta_k I.$$

$$V_m(I_n^*) = V_n(I_n^*) + \Gamma_n. \quad (9)$$

We now prove the following criteria for the decision to take an impulse control.

Theorem 1 *Suppose the DM takes the impulse control $L_m \rightarrow L_n$ after mutation at an interior point $\bar{I}_n^* \in (0, 1)$. If value functions $\bar{V}_m(I)$ and $\bar{V}_n(I)$ are both differentiable in $(0, 1)$, then \bar{I}_n^* satisfies the equation*

$$\rho \left[\bar{V}_n(\bar{I}_n^*) + \Gamma_n \right] = \bar{H}_m^* \left(\bar{I}_n^*, \bar{V}_n'(\bar{I}_n^*) \right). \quad (10)$$

Similarly, suppose the impulse control is taken before mutation at an interior point $\underline{I}_n^ \in (0, 1)$. If $\underline{V}_m(I)$ and $\underline{V}_n(I)$ are differentiable in $(0, 1)$, then \underline{I}_n^* is defined by*

$$(\rho + \lambda) \left[\underline{V}_n(\underline{I}_n^*) + \Gamma_n \right] = \underline{H}_m^* \left(\underline{I}_n^*, \underline{V}_n'(\underline{I}_n^*), \lambda \bar{V}_m(\underline{I}_n^*) \right). \quad (11)$$

Conditions (10) and (11) are essentially transversality conditions that govern the optimal transition between different regimes. In (deterministic) optimal control literature, they are usually stated in terms of the maximized Hamiltonians, holding before and after the regime change (see for instance Boucekine et al. 2013). In the absence of lump-sum cost, these optimality conditions impose the continuity of the Hamiltonians at the date of regime switching, when the impulse control is taken, and the continuity of the co-state variable(s) as long as the level of the state variable(s) at which the decision is taken is free. In dynamic programming, these correspond to the well-known value matching and smooth pasting conditions, expressed in terms of value functions and their first order derivative. Here, we merge both approaches because of the specificity of our problem. Indeed, the comparison between conditions (10) and (11) reveals how the prospect of a mutation affects the impulse control. We observe that both the hazard rate and the value function after a mutation, in regime (L_m, \bar{Z}) , show up in the optimality condition before any mutation.

For further discussion, let us express these conditions in the specified model. We obtain:

$$\begin{aligned} & \bar{I}_n^* \bar{V}_n'(\bar{I}_n^*) (\bar{\theta}_n - \bar{\theta}_m) (1 - \bar{I}_n^*) \\ & - \frac{1}{2} \left[\bar{I}_n^* \bar{V}_n'(\bar{I}_n^*) \right]^2 (\bar{\theta}_n^2 - \bar{\theta}_m^2) + h_n - h_m + \rho \Gamma_n = 0, \end{aligned} \quad (12)$$

and

$$\begin{aligned} & \underline{I}_n^* \underline{V}_n'(\underline{I}_n^*) (\underline{\theta}_n - \underline{\theta}_m) (1 - \underline{I}_n^*) - \frac{1}{2} \left[\underline{I}_n^* \underline{V}_n'(\underline{I}_n^*) \right]^2 (\underline{\theta}_n^2 - \underline{\theta}_m^2) \\ & + h_n - h_m + \lambda (\bar{V}_n(\underline{I}_n^*) - \bar{V}_m(\underline{I}_n^*)) + (\rho + \lambda) \Gamma_n = 0, \end{aligned} \quad (13)$$

respectively.

We immediately notice that the optimality condition before a mutation is modified in two ways, compared to the condition after. First, we get that being subject to uncertainty

induces the DM to use an augmented discount rate, that is the sum of the pure rate of time preference and the hazard rate. This is a well-known effect of considering the occurrence of stochastic (exogenous) events in optimal control problems. To stay close to the topic under scrutiny, this is actually similar to what is obtained in the “short-term analyses” of optimal lockdown policy that assume that the planning horizon is finite but uncertain (Alvarez et al. 2021; Jones et al. 2021 etc.), except that the source of uncertainty is different.¹⁵ Second, there is an extra term that involves the difference of the value functions obtained in the two lockdown states, after the mutation. This term represents the net (positive or negative) gain of operating under lockdown regime L_n , instead of L_m . It means that when contemplating the opportunity to take a lockdown measure before a mutation, the DM has to take into account the net gain to be in a different lockdown state if and once a mutation occurs. This feature is very much in line with what Long et al. (2017) have shown in different context.¹⁶

In what follows, we provide necessary conditions for lockdown measures and discuss them.

3.2 Necessary conditions for impulse control

We further investigate the parametric conditions under which the DM may want to take an impulse control $L_m \rightarrow L_n$, whatever the pandemic state. That is, we provide necessary conditions for the existence of threshold levels of the state variable for an impulse control.

Theorem 2 *If the impulse control $L_m \rightarrow L_n$ is taken for some $\bar{I}_n^* \in (0, 1)$ after mutation, then*

$$\frac{\bar{\theta}_m - \bar{\theta}_n}{2(\bar{\theta}_m + \bar{\theta}_n)} \geq h_n - h_m + \rho\Gamma_n. \tag{14}$$

If the impulse control $L_m \rightarrow L_n$ is taken for some $\underline{I}_n^ \in (0, 1)$ before mutation, then*

$$\frac{\theta_m - \theta_n}{2(\theta_m + \theta_n)} \geq h_n - h_m + (\rho + \lambda)\Gamma_n + \lambda(\bar{V}_n(\underline{I}_n^*) - \bar{V}_m(\underline{I}_n^*)) \tag{15}$$

After a mutation, the necessary condition involves the net total cost, which is the sum of the lump-sum cost and of the difference between operation costs ($h_n - h_m$), and the health net gain ($\bar{\theta}_m - \bar{\theta}_n$) of switching the lockdown state. Then, we observe that using the impulse control after a mutation has occurred can be worth only when the health gain is sufficiently large and/or the cost imposed to society is low enough.

¹⁵ These papers typically assume that the horizon is given by the arrival of a vaccine, modeled as a Poisson process, which boils down to working with a deterministic infinite horizon optimal control problem with an increased discount rate (equal to the sum of the rate of time preferences and the constant hazard rate) and salvage value function.

¹⁶ The authors analyze a two-player differential game with impulse, or regime switching, strategies and show how the prospect of a future regime switching by one player affects the regime switching strategy of the other.

Before a mutation, the necessary condition is changed since we know by Theorem 1 that the DM should take care of what comes next. Suppose first that the value functions after mutation are the same. The eventuality of a lockdown regime switching in that scenario requires the health gain be much larger. With different value functions, what happens after the mutation also matters. If the DM expects that the overall cost of the pandemic is going to be larger ($\bar{V}_n(\cdot) - \bar{V}_m(\cdot) > 0$), once the mutation occurs, in the new lockdown state L_n , then the necessary condition becomes even harder to meet. If on the contrary, the DM considers that the economy will be better prepared to handle the pandemic with the new regime ($\bar{V}_n(\cdot) - \bar{V}_m(\cdot) < 0$), then the necessary condition for impulse control is less demanding.¹⁷

The influence of stochastic mutation on impulse control raises a series of questions: how does the DM adapt her policy to the risk of virus mutation? Is there an incentive to use lockdown as a prevention to a potential mutation, or as an adaptation to an actual one? Does the prospect of a mutation hasten, or on the contrary, delay lockdown measures? What is the joint effect of lockdown measures and mutations on the dynamics of the pandemic? The next sections address these important issues.

4 Effect of mutation on lockdown and reopening

From now on, for the ease of exposition, we consider the following specific scenario (and replace general indexes m, n with 0, 1):

Assumption 1

$$h_1 > h_0, \quad \Gamma_1 > 0 = \Gamma_0, \quad \bar{\theta}_0 > \bar{\theta}_1, \quad \underline{\theta}_0 > \underline{\theta}_1.$$

All in all, this means that L_0 is the reference situation with no lockdown constraints imposed upon society, while L_1 refers to the situation in which the economy is locked-down. Accordingly, the move $L_0 \rightarrow L_1$ corresponds to a lockdown of the economy (introduction of some constraints), whereas $L_1 \rightarrow L_0$ captures a reopening (removal of these constraints). The ranking between cost and infection rate parameters follow directly from this characterization.

We need to introduce a couple of additional notations for the coming analysis. Based on Theorem 2 and conditions (14)–(15), we first define critical levels for lockdown in any regime, for $k = 0, 1$:

$$\begin{aligned} \bar{\delta}_k &= 2 \frac{\bar{\theta}_0 + \bar{\theta}_1}{\theta_0 - \theta_1} [h_1 - h_0 + \rho\Gamma_k], \\ \underline{\delta}_k &= 2 \frac{\underline{\theta}_0 + \underline{\theta}_1}{\theta_0 - \theta_1} \left[h_1 - h_0 + \rho\Gamma_k + \lambda \max_{\{0 \leq I \leq 1\}} (\bar{V}_1(I) + \Gamma_k - \bar{V}_0(I)) \right]. \end{aligned} \tag{16}$$

¹⁷ In “Appendix A.3”, we also provide sufficient conditions for an impulse control in any regime.

Next, we denote by $U_k(I)$, for $k = 0, 1$, the solution to the following HJB equation:

$$\rho U_k(I) = H_k^*(I, U'_k(I)), \quad \text{for } I \in (0, 1), \tag{17}$$

that holds before a mutation, or after a mutation for $\lambda = 0$.¹⁸

In what follows, we draw some conclusions about the features of the solution, especially the possibility of occurrence and timing of impulse controls under a risk of mutation.

4.1 Connection between lockdown and reopening

We first obtain some general results, that hold regardless of the pandemic state,¹⁹ connecting the occurrence of lockdown vs. reopening type of impulse controls (“Appendix A.4”).

Proposition 1 *Under Assumption 1,*

(2.1) *If lockdown occurs at a point $I_1^* \in (0, 1)$, then $\delta_1 < 1$. In this case, I_1^* satisfies*

$$I_1^* \leq 1 - \sqrt{\delta_1}. \tag{18}$$

Conversely, if lockdown does not occur at an interior point, then $V_0(I) = U_0(I)$ for all $I \in [0, 1]$. In this case, either $V_1(I) = V_0(I)$ for all $I \in [0, 1]$, or there is $I_0^ \in (0, 1)$ such that*

$$\begin{aligned} V_1(I) &= V_0(I) && \text{for } I \leq I_0^*, \\ V_1(I) &< V_0(I) \leq V_1(I) + \Gamma_1 && \text{for } I_0^* < I \leq 1. \end{aligned} \tag{19}$$

In the latter case reopening occurs at $I = I_0^$.*

(2.2) *If reopening occurs at a point $I_0^* \in (0, 1)$, then $\delta_0 < 1$. In this case, I_0^* satisfies*

$$I_0^* \leq 1 - \sqrt{\delta_0}. \tag{20}$$

Conversely, if reopening does not occur at an interior point, then lockdown also does not happen at an interior point. In this case

$$V_1(I) = V_0(I) = U_0(I) \quad \text{for } 0 \leq I \leq 1. \tag{21}$$

¹⁸ Note that $U_k(I)$ needs not be the value function of our problem. This is the optimal value without possibility of impulse control. Hence, by optimality of the value functions, it is necessary that

$$V_k(I) \leq U_k(I), \quad k = 0, 1.$$

¹⁹ Remember that we use no lower or upper bar for variables or functions in this generic case.

Proposition 1 has two parts, divided into two claims. The first claim in each part (conditions for lockdown and reopening at an interior point) follows directly from Theorem 2. Let us then discuss the second one. If a lockdown does not happen at an interior point where some individuals are infected, then the DM does not impose lockdown in the current regime. In this case, by definition, the value function $U_0(I)$ yields the lowest social cost ($V_0(I) = U_0(I)$) as lockdown is never beneficial. Then, there are two possibilities. Either the social cost of lockdown is higher than that without lockdown, or the social cost of lockdown is lower but adding the lump-sum start up cost the total cost is too high. In the former case naturally the DM will never impose lockdown in the current regime, and if the regime is after mutation and the state is locked down before mutation, once mutation occurs, the DM will immediately reopen. The latter case can only happen if the share of the infected population is already high ($I_0^* < I \leq 1$). In the case where lockdown is imposed before mutation when the infected population is low, i.e., at some $I \leq I_0^*$, the DM would immediately reopen when the mutation occurs. If the mutation occurs with high infected population, i.e., at some $I > I_0^*$, since the social cost with lockdown is lower, the DM will keep the state locked down until the share of the infected people drops to I_0^* , and then reopen at this moment.

Finally, if reopening does not occur at an interior point, the only possibility is that $I_0^* = 1$. This means even if the entire population is infected, lockdown is not a choice. So, certainly lockdown does not happen for any size of infected population.

4.2 Risk of mutation and timing of lockdown

The most important part of our analysis deals with the impact of the prospect of mutation on both types of impulse control decisions, before mutation.

At first, we look at the impact of the mutation risk on the critical levels defined in (16). For an impulse control $L_0 \rightarrow L_1$ to occur at an interior point, it is necessary that $\delta_1 < 1$. Since by (7)–(8)

$$\bar{V}_1(I) \leq \bar{V}_0(I) \leq \bar{V}_1(I) + \Gamma_1$$

for all $I \in [0, 1]$, it follows that δ_0 is nonincreasing in λ whereas δ_1 is nondecreasing in λ . Hence, reopening at an interior point before mutation can be impossible without mutation, but becomes possible with mutation, and lockdown can be possible without mutation, but becomes impossible with mutation. To be more precise, based on the above results, we see that if reopening at an interior point is not possible after mutation, then

$$\delta_0 = 2 \frac{\theta_0 + \theta_1}{\theta_0 - \theta_1} (h_1 - h_0)$$

is independent of λ , but

$$\delta_1 = 2 \frac{\theta_0 + \theta_1}{\theta_0 - \theta_1} [h_1 - h_0 + (\rho + \lambda) \Gamma_1]$$

is strictly increased by a multiple of λ . This means lockdown before mutation can become less possible.

Admittedly, this is not informative enough because the δ_k are just upper bound on the threshold levels I_k^* . So, we have no option but to study the impact of mutation on these levels. For that purpose, we conduct an analysis that is based on the comparison between critical thresholds for lockdown I_k^* , in the conditions before a mutation but with no risk of mutation, and after a mutation. In the case $\lambda = 0$, and when $\theta_k > K$, we can show the existence of a minimum value function and exploit its features to get to the result.

Indeed, when $\theta_k > K$ and for our specified model, the value function (17) is not unique. There are two reasons for the non-uniqueness of the value function. The first one is in line with standard optimal control problems where the HJB equations without boundary conditions generally have infinitely many solutions.²⁰ The second reason comes directly from the special setting of the current study. The value function solves

$$\rho U_k(I) = I\theta_k U'_k(I) (I_k - I) - \frac{[\theta_k I U'_k(I)]^2}{2} + \rho q_k(I),$$

$$\text{with } q_k(I) = \frac{h_k}{\rho} + \frac{\beta}{2\rho} I^2, \text{ and } I_k = 1 - \frac{K}{\theta_k}, \tag{22}$$

which has two positive solutions in $U'_k(\cdot)$, associated with two vaccination rates according to (5). The DM can use one of the roots on some intervals of I and the other root on other intervals of I . There is no constraint and each choice leads to a value function. As there are infinitely many possible partitions of the interval of I , there are infinitely many value functions. Nonetheless, it can be shown that there exists a minimum solution among all possible solutions to the HJB equation.

Lemma 1, in the ‘‘Appendix A.5’’, constructs this minimum value function under lockdown state L_k when $\lambda = 0$. Let $U_k(I; I'_k)$ be this function, it is defined by

$$U'_k(I; I'_k) = \frac{1}{\theta_k I} \left[I_k - I - \sqrt{(I_k - I)^2 + 2\rho [q_k(I) - U_k(I; I'_k)]} \right],$$

$$\text{with } I'_k = \frac{2I_k}{1 + \sqrt{1 + \frac{2\beta}{\rho} \theta_k I_k}}. \tag{23}$$

This particular value function has the advantage of generating very neat dynamics (second part of the same ‘‘Appendix’’). The system admits a unique globally stable steady state (StS), $I'_k \in (0, 1)$.²¹ Making use of this, we can show that the prospect of mutation actually affects both lockdown and reopening (‘‘Appendix A.6’’).

²⁰ To solve this issues, in the linear-quadratic optimal control case, affine optimal strategy is one possible choice. Dockner and Long (1993) provide one example of this kind, where they study both linear and nonlinear strategies.

²¹ We have $\dot{I} < 0$ if and only if $I < I'_k$. In addition, $I(t)$ reaches $I'_k \in (0, 1)$ in finite time from any point $I > 0$.

Proposition 2 (3.1) *Suppose lockdown occurs before mutation at an interior point $\underline{I}_1^*(0) \in (0, 1)$ with $\lambda = 0$ which is less than the least positive steady state. If after mutation lockdown does not occur at some $\bar{I}_1^* \leq \underline{I}_1^*(0)$, then there is $\varepsilon > 0$ such that lockdown occurs at some $\underline{I}_1^*(\lambda) > \underline{I}_1^*(0)$ for $0 < \lambda < \varepsilon$. If after mutation lockdown occurs at some $\bar{I}_1^* < \underline{I}_1^*(0)$, then there is $\varepsilon > 0$ such that lockdown occurs at some $\underline{I}_1^*(\lambda) \leq \underline{I}_1^*(0)$ for $0 < \lambda < \varepsilon$.*

(3.2) *Suppose $\underline{\theta}_1 < K$ and that reopening occurs before mutation at an interior point $\underline{I}_0^*(0) \in (0, 1)$ with $\lambda = 0$. If after mutation reopening occurs at some $\bar{I}_0^* < \underline{I}_0^*(0)$, then there is $\varepsilon > 0$ such that reopening occurs at some $\underline{I}_0^*(\lambda) < \underline{I}_0^*(0)$ for $0 < \lambda < \varepsilon$. If after mutation reopening occurs at some $\bar{I}_0^* > \underline{I}_0^*(0)$, then there is $\varepsilon > 0$ such that reopening occurs at some $\underline{I}_0^*(\lambda) \geq \underline{I}_0^*(0)$ for $0 < \lambda < \varepsilon$.*

As mentioned before, the proof is built on the comparison between threshold levels for impulse control, in the absence of uncertainty, in the pandemic conditions of before vs. after a mutation. Based on this comparison, we can then determine whether being subject to a risk of mutation makes one delay or on the contrary expedite impulse control. We further consider scenarios in which in the absence of policy intervention, the share of infected would increase in every pandemic regime. The first part of Proposition 2 deals with the impact of uncertainty about the virus on the lockdown decision. We obtain that if in the absence of uncertainty, it would be optimal for the DM to take the lockdown decision “sooner” in the worst case scenario (in terms of infectivity) than in the best case one, then uncertainty expedites lockdown. Put differently, the prospect of a “bad mutation” at some uncertain future date induces the DM to act more cautiously. In this particular context, this boils down to imposing a lockdown sooner (than in the absence of risk of mutation) in order to be better prepared to the future likely event of a mutation. Here the DM acknowledges that the economy would be better-off under lockdown following the occurrence of a bad mutation. There is an additional gain of being under lockdown that shows up in the optimality condition (11) and increases the incentives to impose such restrictions for low levels of infection in the population.

On the contrary, the risk of experiencing a good mutation delays lockdown measures. In this case, given the costs of this policy (that are known for sure) and its uncertain, yet likely low, benefit, the DM prefers to wait and see the evolution of the pandemic situation before taking this kind of decision. The second part of the Proposition provides symmetric results for reopening decision: a bad mutation delays reopening before it happens and then decision is surrounded by uncertainty, and a good one does the opposite.

We can draw a parallel between our conclusions and the two main strands of the literature on decision making under uncertainty. First and foremost, there is a long tradition of papers studying the impact of the occurrence of random events on optimal decision making dating back to Dasgupta and Heal (1974) and Cropper (1976). Many papers precisely ask how being subject to costly (sometimes catastrophic) events shapes decisions, with many applications in environmental and resources economics (see Crepin 2020, for a recent overview of the literature). One of the main messages is that the optimal response to a risk of costly event is to behave more cautiously (in

terms of resource extraction or polluting emissions for instance). Our result in the bad mutation case clearly echoes those obtained in the literature, and extends them to the class of impulse controls. Second, these results also have a connection with the real option value literature (Dixit and Pindyck 1994) that emphasizes the role of uncertainty and learning in forming decisions under irreversibility (see Bandyopadhyay et al. 2021; Gollier 2020b, for contributions on the control of epidemics).²² In our setting, because the DM can take the lockdown and reopening decisions whenever she wants, there exists (at least in the good mutation case) an incentive to wait and possibly experience the mutation before acting, as upon a mutation, the information about the disease contagiousness is revealed.

5 Evolution of the pandemic

This section discusses the link between impulse controls and the evolution of the pandemic. This discussion is conducted whatever the pandemic regime. It starts with a brief overview of the general features of the dynamics, that is of the features that hold whatever the value function.

5.1 Dynamics: general insights

The share of infected people, under lockdown state L_k , is governed by the following differential equation

$$\dot{I} = I \left[\theta_k (1 - I) - K - \theta_k^2 I V_k'(I) \right], \text{ for } k = 0, 1. \tag{24}$$

Since $V_k' \geq 0$, from (5), $I(t)$ is decreasing over time if $\theta_k (1 - I) \leq K$. Hence, if $\theta_k \leq K$ and the corresponding regime is terminal, then $I(t)$ monotonically decrease to 0, the disease-free StS, as $t \rightarrow \infty$. In this case, the efficiency of the health system is so high that the share of infected people decreases no matter what, i.e., even in the absence of vaccination policy, r . This implies that vaccination is only useful to control the speed of decrease of the contagion. This situation may arise if θ_k falls below K as a result of the lockdown measure. If on the contrary, $\theta_k > K$, the dynamic and the StS analyses are more complicated as they depend on the shape of the value function, vaccination policy, and of course are regime-dependent. The disease-free StS is always unstable. In addition, there exist an odd number of endemic StS, i.e., StS featuring positive I (see ‘‘Appendix A.7’’). The infimum and the supremum of the set of endemic StS, \hat{I}_k, \tilde{I}_k , are such that $0 < \hat{I}_k \leq \tilde{I}_k < I_k$, with I_k defined in (22),²³ and

$$\lim_{t \rightarrow \infty} I(t) = \hat{I}_k \text{ if } 0 < I(0) < \hat{I}_k, \text{ and } \lim_{t \rightarrow \infty} I(t) = \tilde{I}_k \text{ if } \tilde{I}_k < I(0) \leq 1.$$

²² Because lockdown decision involves a sunk-cost, it is at least partly irreversible.

²³ Since \tilde{I}_k is a positive root of the function on the right-hand side of (24), it follows that $\theta_k (1 - \tilde{I}_k) \geq K$, which yields an upper bound on the supremum, and on the entire set of StS.

Next we examine how lockdown controls shape the pandemic dynamics, and vice-versa.

5.2 Interplay between lockdown, reopening, and the pandemic dynamics

Under Assumption 1, by (7) and (8),

$$V_1(I) \leq V_0(I) \leq V_1(I) + \Gamma_1, \quad \text{for } I \in [0, 1],$$

for both before and after mutation. We can be a little more precise regarding the ranking of the value functions, for different states, I , and depending on whether there exist threshold levels I_k^* for switching to lockdown state L_k . Indeed, since $h_1 > h_0$, and

$$U_k(0) = \frac{h_k}{\rho} \quad \text{for } k = 0, 1,$$

it follows that

$$V_1(0) \leq V_0(0) \leq \frac{h_0}{\rho} < U_1(0).$$

Hence, $V_1(I) \neq U_1(I)$ for small I . This implies that

$$V_1(I) = V_0(I) \tag{25}$$

for I sufficiently small. Either (25) holds for all $I \in [0, 1]$ or there is $I_0^* \in (0, 1)$ such that (25) holds only for $0 \leq I \leq I_0^*$. In case I_0^* exists,

$$V_1(I) < V_0(I) < V_1(I) + \Gamma_1 \tag{26}$$

holds for $I > I_0^*$ and is near I_0^* . Either (26) holds for all $I \in (I_0^*, 1]$ or there is $I_1^* < 1$ such that (26) only holds for $I_0^* < I < I_1^*$. For $I > I_1^*$ the relation

$$V_0(I) = V_1(I) + \Gamma_1$$

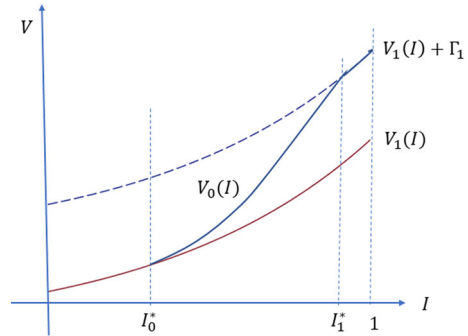
must hold. With this in mind, it is possible to assess the possible outcomes, in any pandemic regime. There are three possible scenarios whether I_0^* and/or I_1^* exist(s). Hereafter, we only discuss the most general one where both thresholds exist, the two others being postponed to the ‘‘Appendix A.10’’.²⁴

In this case, as illustrated in Fig. 1, for $I \leq I_0^*$, $V_1(I) = V_0(I)$. For $I_0^* < I < I_1^*$, (26) holds, and for $I_1^* \leq I \leq 1$, $V_0(I) = V_1(I) + \Gamma_1$.

If at $t = 0$ the state is locked down and $I(0) \leq I_0^*$, reopening immediately occurs. After that, either $I(t)$ approaches a StS and the state remains un-locked down, or $I(t)$

²⁴ In order to have a full picture of the optimal solution, one would then have to combine the properties of the solution before mutation, given that a mutation will occur eventually (i.e. before the steady state is achieved), with the ones of the solution after, when there is no more room for a mutation of the virus.

Fig. 1 Both I_0^* and I_1^* exist



approaches I_1^* , triggering lockdown. In the latter case, either $I(t)$ approached a StS while the state remains locked down, or it reaches I_0^* triggering reopening. In the latter case $I(t)$ will approach to I_1^* and trigger lockdown again. The pattern repeats until the virus mutates if one considers the situation before a mutation. So in the case, we obtain a quite sophisticated policy whereby the DM adapts to the evolution of the pandemic by switching-on and off the lockdown button. As expected, beside the vaccination policy, the DM uses the impulse control to manage the spread of the virus across the population. When the situation gets worse, the DM take the lockdown decision that is later removed when it improves. Overall, many things can happen depending on the respective locations of StS and threshold levels. To dig deeper into this issue, we need to proceed to numerical simulations.

Before that, let us conclude this discussion with an additional result regarding the asymptotic behavior of the system (“Appendix A.8”).

Proposition 3 *Under Assumption 1, if $\theta_0 > K$, then $I(t)$ does not converge to the disease-free StS. If $\theta_0, \theta_1 \leq K$, then the state is un-locked down for large t and $I(t)$ converges to zero.*

This confirms that the ranking between the θ_k s, and especially θ_0 the infection rate in the absence of lockdown, and K is crucial to characterize the asymptotic behavior of the optimal solution. When $\theta_0 > K$, the share of infected people will converge to a positive value whatever the case. The public policy proves itself worth for controlling the pandemic, but it never allows the system to erase it. By contrast, in the best case scenario where the health system is very efficient, $\theta_0, \theta_1 \leq K$, the pandemic will necessary vanish eventually, making the vaccination and lockdown policy less essential.

6 Calibration

Hereafter, we perform a numerical analysis by borrowing the following set of parameters from Huberts and Thijssen (2023) (see their table 1):

$$K = 0.1, \quad \rho = 0.1/365, \quad \beta = 4, \quad \Gamma_1 = 2, \quad h_0 = 0, \quad h_1 = 0.01884.$$

A couple of remarks are in order here. These authors run simulations based on a proper calibration of their SIR model to UK for COVID-19. Since their basic model is the same as ours, we simply take their values for the parameters we share.²⁵ The unit of time is the day and costs are all measured in Million GBP. The main difference between our approach and theirs is that they consider neither mutation, nor vaccination. So, the first thing to do is to attribute their estimates of the infection rates to the situation before or after mutation. We choose to use them for depicting the situation before mutation

$$\underline{\theta}_0 = 0.3, \quad \underline{\theta}_1 = 0.15.$$

This likely corresponds to a pandemic regime dominated by the variant Alpha. Then, the situation after mutation corresponds to what happened when the next variant Delta became prevalent. Experts' estimates point to Delta being nearly 80% more contagious than Alpha.²⁶ Thus, as for the infection rates after mutation, we suppose that

$$\bar{\theta}_0 = 0.3 \times 1.8 = 0.54, \quad \bar{\theta}_1 = 0.15 \times 1.8 = 0.27,$$

and will consider a milder increase later for robustness check. We also have to calibrate the hazard rate, or the rate of arrival of a new variant. There is no compelling evidence for this. However, proceeding to simple back-to-envelope calculations, we take $\lambda = 0.015$, which represents 5 to 6 mutations per year.²⁷ Finally, our model does not include a parameter for the quadratic cost of vaccination. We add one for the numerical analysis and normalize it to one for the benchmark scenario, as we found no estimate of the daily total cost of vaccination policy in the UK. For robustness, we will also analyze the features of the solution for a lower value of this parameter.

In "Appendix A.11", we determine the expressions of the minimum value functions after and before mutation. Their representation is shown in Fig. 2. One observes that it is never optimal for the DM to lockdown the economy since $\bar{U}_1 > \bar{U}_0$ for $I \in [0, 1]$. Therefore, after mutation the economy must converge to the StS $\bar{I}'_0 \approx 0.01425$ in finite time. One can also notice that the vaccination rate is maintained at a relatively high level in mode 0, which certainly explains why the share of infected at the StS is actually lower than the one that would be reached under lockdown ($\bar{I}'_1 \approx 0.01762$).

²⁵ In their SIR model, their recovery rate is the rate at which people move from the infected to the recovery state. We keep this value and interpret it literally. As to the daily cost of infected people, β , we have to make a conversion because they use a linear cost in I , whereas we use a quadratic one. In addition we normalize the population size to 1, while they use a value of 500. We compare the cost if the entire population is infected. In Huberts and Thijssen (2023), each person costs 0.004 million pounds per day and there are 500 people. So if the entire population is infected, the cost is 2 million per day. In our model, if the entire population is infected, $I = 1$ and the cost is $\beta/2$ million per day. So $\beta=2*2=4$. Finally note that we cannot directly compare our results to theirs as they use a SIR model and make sure the system converges to the disease-free StS.

²⁶ See <https://www.yalemedicine.org/news/covid-19-variants-of-concern-omicron>. Admittedly, different variants also feature different severity, which we do not capture here.

²⁷ To get this figure, divide the number of variants of concerns, in World Health Organization's words, that have occurred between Nov. 2021 and Nov. 2022, by the number of days (Alpha, Beta, Gamma, Delta, Omicron).

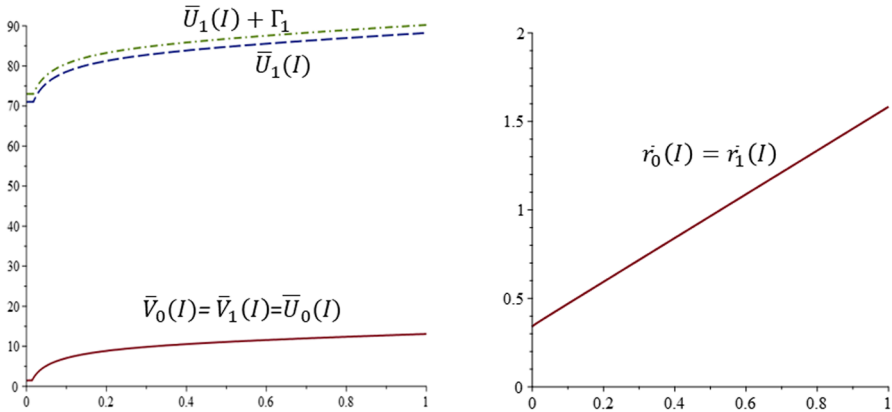


Fig. 2 Value functions (left) and vaccination rates (right) after mutation

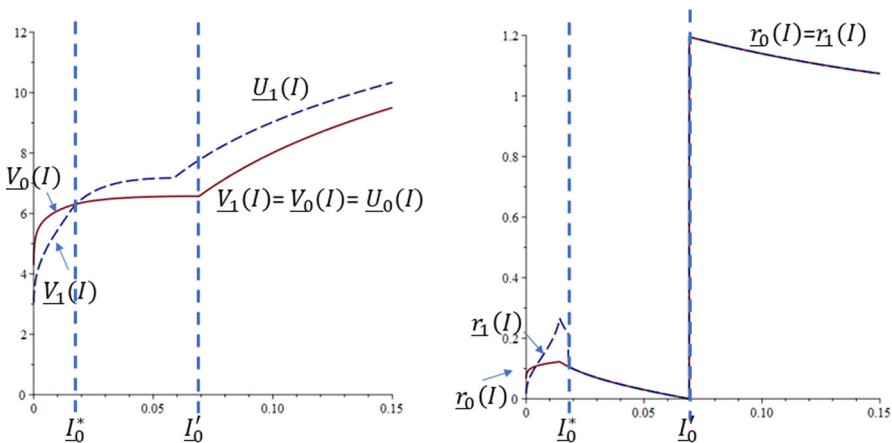


Fig. 3 Value functions (left) and vaccination rates (right) before mutation

Moreover, if at the moment of mutation the state were locked down, the DM would reopen immediately while if it is already open, it remains so.

Let us now have a look at the situation before mutation. Based on the comparison between minimum value functions, we get $\underline{U}_0(I) < \underline{U}_1(I)$ for some $I \in (0, 1)$. So, there exists a reopening point, at $I_0^* \approx 0.01788$. We have $\underline{V}_1(I) = \underline{V}_0(I) = \underline{U}_0(I)$, and $\underline{r}_1(I) = \underline{r}_0(I)$ for $I > I_0^*$. In addition, one can check that there is no lockdown point, I_1^* (as $\underline{V}_1 + \Gamma_1 > \underline{U}_0$ for all I). This means that $\underline{V}_0(I) = \underline{U}_0(I)$ for all $I \in (0, 1)$. The value functions and the vaccination rates before mutation are shown in Fig. 3 (see also the ‘‘Appendix’’).

Next the question is: how does this reopening point compare to the potential StS of the system? For the given parameter values, we find

$$I_0' \approx 0.06915, \quad I_1' \approx 0.05861.$$

The StS before mutation are both much lower than after. In addition, the StS under lockdown is smaller than under an open economy. This is certainly driven by the lower infection rate.

More importantly, these numbers imply that, depending on the initial state I_0 , three situations may occur before mutation. Suppose that no mutation takes place. First, if at the initial time the state is not locked down, it will remain unlocked all the time. In this case, for any value of $I_0 \in (0, 1)$, $I(t)$ reaches \underline{I}'_0 in finite time. Next, if at the initial time the state is locked down, there are two cases depending on whether $I_0 \geq \underline{I}_0^*$ or $I_0 < \underline{I}_0^*$. When $I_0 \geq \underline{I}_0^*$, the DM reopens the economy immediately, and $I(t)$ moves toward \underline{I}'_0 . When $I_0 < \underline{I}_0^*$, the DM does not immediately reopen and $I(t)$ increases. The DM lifts the lockdown when $I(t)$ achieves \underline{I}_0^* . After that, $I(t)$ continues to increase until it reaches \underline{I}'_0 . Of course, whenever mutation occurs, the DM immediately reopens and $I(t)$ goes to \bar{I}'_0 in finite time.

The most interesting case is the one where there is a lockdown initially and $I_0 < \underline{I}_0^*$. In this case, lockdown is a means to slow down the evolution of the disease. Quite noteworthy, lockdown measures are combined with pretty high vaccination rates (at least higher as those of an open economy). This further reinforces the capacity of the DM to keep the pandemic under control. It also highlights that the two instruments display some complementarity. But this is not sufficient to stop the pandemic progression. As time goes by, the share of infected increases, which triggers reopening. This may sound quite counterintuitive. This is not, though. Indeed, once the lockdown is lifted, the DM can rely on vaccination, at pretty low rates, to control the spread of the disease, to put the system onto the path of the StS with a positive yet low share of infected population. This points to vaccination being the preferable policy to deal with the pandemic eventually. Overall, the numerical analysis emphasizes the qualitatively different features of the optimal policy, depending on whether the DM takes action before or after mutation.

Finally note that for robustness check, we have conducted two additional analyses. First, we have considered the case where the virus after mutation is only 20% more contagious than before and obtained the same general pattern.²⁸ Second, we have run the simulations by using a parameter value of 0.4 (10% of β) for the cost of vaccination. Of course, considering a lower cost of vaccination makes this control even more attractive, compared to lockdown. This could ultimately lead to a full management of the pandemic thanks to vaccination only, even before mutation. This is indeed what we obtain in this scenario.²⁹

²⁸ The DM keeps the state unlocked all the time after mutation, and $I(t)$ reaches $\bar{I}'_0 \approx 0.01639$. Before mutation, there is a reopening point at $\underline{I}_0^* \approx 0.01830$, but no lockdown point. The StS before mutation is $\underline{I}'_0 \approx 0.04129$, which is reached in finite time regardless whether initially the state is open or locked down. Compared with the original case, the StS and the reopening point before mutation are both slightly lower, and the StS after mutation is slightly higher.

²⁹ Here the reopening point is $\underline{I}_0^* = 1$, i.e., the DM immediately opens the economy if locked and keeps it opened otherwise.

7 Conclusion

This is the first paper that combines lockdown as an impulse control, vaccination and uncertainty surrounding the evolution of the transmission rate to analyze the optimal control of a pandemic. The aim of the paper is to analyze the impact of random mutations of the disease on policy interventions, especially lockdown decisions. Lockdown is modeled as an impulse control that allows the system to switch from one level of restrictions to another (stricter or softer). This can be a valuable option, together with vaccination, to control the spread of the disease. More fundamentally, in our setting, lockdown can serve as a way to anticipate a mutation or to respond to it. Indeed, decisions are taken under the risk of mutations of the disease, with repercussions on the transmission rate. The decision maker follows a cost minimization objective. In a simplified model where the virus can mutate only once and there exist only two lockdown levels, we first characterize the optimality conditions for impulse control and show how the prospect of a mutation affects the decision maker's choice. In fact, it induces her to anticipate the relative benefit of a regime change after a mutation has occurred, which may or may not increase the incentive to set a lockdown. Our problem admits infinitely many value functions. Under some parametric conditions, we show the existence of a minimum value function that is a natural candidate to the solution. We then study the features of the optimal policy and notably prove that uncertainty surrounding future mutation of the disease expedites lockdown intervention whenever mutation increases contagiousness. This conclusion strikingly echoes those of the literature dealing with the impact of the occurrence of (random) costly events on decision making, and extends them to the class of impulse control. We finally conduct a numerical analysis that shows how the management of the pandemic is influenced by the prospect of a bad mutation. Lockdown control can be valuable only before mutation (even if the infection rate is lower) and should be combined with pretty high rates of vaccination to control the pace of diffusion of the pandemic. However, lockdown measures should be removed in finite time and the pandemic controlled by means of vaccination eventually. Several avenues can be taken for future works. Modeling an endogenous hazard, a larger number of mutations and lockdown levels, and regime-dependent costs and recovery rates are promising future lines of research, among others.

A Appendix

A.1 Proof of Theorem 1

Suppose DM takes the impulse control $L_m \rightarrow L_n$ after mutation at $I^* \in (0, 1)$. Then the value function $\bar{V}_m(I)$ satisfies the HJB equation

$$\rho \bar{V}_m(I) = \bar{H}_m^*(I, \bar{V}_m'(I)) \quad (27)$$

with

$$\overline{H}_m^*(I, p) = Ip [\overline{\theta}_m (1 - I) - K] - \frac{[\overline{\theta}_m Ip]^2}{2} + h_m + \frac{\beta}{2} I^2.$$

The differential equation is supplemented by the boundary condition (9). The HJB equation is quadratic in $\overline{V}'_m(I)$. Let $\overline{Q}_m(I, v)$ be the minimum positive root, v , of the equation

$$\rho v = H_m^*(I, p).$$

If solution exists, we can write the equation in the form

$$\overline{V}'_m(I) = \overline{Q}_m(I, \overline{V}_m(I))$$

Thus $\overline{V}_m(I)$ satisfies the integral equation

$$\overline{V}_m(I) = \overline{V}_n(I^*) + \Gamma_n + \int_{I^*}^I \overline{Q}_m(u, \overline{V}_m(u)) du$$

for I on the side of I^* on which the HJB equation holds equal. Since the DM chooses I^* to maximize $\overline{V}_m(I)$, the derivative of the right-hand side with respect to I^* vanishes. Hence

$$\overline{V}'_n(I^*) - \overline{Q}_m(I^*, \overline{V}_m(I^*)) = 0.$$

By the terminal condition (9), we also have

$$\overline{V}'_n(I^*) - \overline{Q}_m(I^*, \overline{V}_n(I^*) + \Gamma_n) = 0$$

which is equivalent to (10). This proves the first part of the theorem.

Suppose the impulse control $L_m \rightarrow L_n$ is taken before mutation at $I^* \in (0, 1)$. Then for I on the side of I^* the value function $\underline{V}_m(I)$ satisfies the HJB equation

$$(\rho + \lambda) \underline{V}_m(I) = \underline{H}_m^*(I, \underline{V}'_m(I), \overline{V}_m(I)) \tag{28}$$

for I on the side of I^* before the impulse control is taken, with

$$\underline{H}_m^*(I, p, W) = Ip [\underline{\theta}_m (1 - I) - K] - \frac{[\underline{\theta}_m Ip]^2}{2} + h_m + \frac{\beta}{2} I^2 + W, \tag{29}$$

and \underline{V}_m satisfying the boundary condition:

$$\underline{V}_m(I_n^*) = \underline{V}_n(I_n^*) + \Gamma_n.$$

Let $\underline{Q}_m(I, v, W)$ be the minimum positive root, v , of the equation

$$(\rho + \lambda) v = \underline{H}_m^*(I, p, W),$$

if the root exists. Then

$$\underline{V}_m(I) = \underline{V}_n(I^*) + \Gamma_n + \int_{I^*}^I \underline{Q}_m(u, \underline{V}_m(u), \bar{V}_m(u)) du.$$

Differentiate the right-hand side with respect to I^* . It follows from the terminal condition that

$$\underline{V}'_n(I^*) - \underline{Q}_m(I^*, \underline{V}_n(I^*) + \Gamma_n, \bar{V}_m(I^*)) = 0.$$

This leads to (11). The proof is complete.

A.2 Proof of Theorem 2

To prove (14), we note that equation (12) is quadratic in $\bar{I}_n^* \bar{V}'_n(\bar{I}_n^*)$. For this equation to be solvable, it is necessary that

$$(1 - \bar{I}_n^*)^2 + 2 \frac{\bar{\theta}_m + \bar{\theta}_n}{\bar{\theta}_n - \bar{\theta}_m} [h_n - h_m + \rho \Gamma_n] \geq 0.$$

Or equivalently,

$$(1 - \bar{I}_n^*)^2 \geq 2 \frac{\bar{\theta}_m + \bar{\theta}_n}{\bar{\theta}_m - \bar{\theta}_n} [h_n - h_m + \rho \Gamma_n].$$

As $\bar{I}_n^* \in (0, 1)$, we further need to impose that the RHS is not greater than 1, which leads to (14).

Similarly, equation (13) is quadratic in $\underline{I}_n^* \underline{V}'_n(\underline{I}_n^*)$. To have a real root, it is necessary that

$$(1 - \underline{I}_n^*)^2 + 2 \frac{\underline{\theta}_m + \underline{\theta}_n}{\underline{\theta}_n - \underline{\theta}_m} [h_n - h_m + (\rho + \lambda) \Gamma_n + \lambda (\bar{V}_n(\underline{I}_n^*) - \bar{V}_m(\underline{I}_n^*))] \geq 0.$$

Again, because $\underline{I}_n^* \in (0, 1)$, the second term must be no greater than 1, so we get (15).

A.3 Sufficient condition for impulse control

For the sake of completeness, we can also provide sufficient conditions for an impulse control in any regime. For that purpose, we need to introduce a couple of additional

concepts. Let $\bar{U}_k(I)$ and $\underline{U}_k(I)$, for $k = m, n$, be the solutions to the following HJB equations:

$$\rho \bar{U}_k(I) = \bar{H}_k^*(I, \bar{U}'_k(I)), \quad \text{for } I \in (0, 1), \quad (30)$$

and

$$(\rho + \lambda) \underline{U}_k(I) = \underline{H}_k^*(I, \underline{U}'_k(I), \lambda \bar{V}_k(I)) \quad \text{for } I \in (0, 1), \quad (31)$$

where \bar{H}_k^* and \underline{H}_k^* are defined in (4) and (29), and \bar{V}_k is the value function in mode k after mutation. We remark that $\bar{U}_k(I)$ and $\underline{U}_k(I)$ need not be the value functions. These are optimal values without possibility of impulse control. Hence, by optimality of the value functions, it is necessary that

$$\bar{V}_k(I) \leq \bar{U}_k(I), \quad \underline{V}_k(I) \leq \underline{U}_k(I) \quad \text{for } I \in (0, 1), \quad k = m, n.$$

Then, we can establish that³⁰:

Theorem 3 *Suppose $\Gamma_m + \Gamma_n > 0$. Let U_k be either \bar{U}_k after mutation, or \underline{U}_k before mutation, for $k = m, n$. The following claims are true.*

1. *If*

$$h_n - h_m + \rho\Gamma_n < 0 \quad (32)$$

and

$$U_m(1) < U_n(1) + \Gamma_n. \quad (33)$$

Then impulse control $L_m \rightarrow L_n$ must occur at some $I^ \in (0, 1)$.*

1. 2. *If*

$$h_n - h_m + \rho\Gamma_n > 0 \quad (34)$$

and

$$U_m(1) > U_n(1) + \Gamma_n \quad (35)$$

Then $L_m \rightarrow L_n$ must occur at some $I_n^ \in (0, 1)$.*

These sufficient conditions all sound pretty natural. For an interpretation, it is enough to focus on part 2. of Theorem 3. Consider that the impulse control $L_m \rightarrow L_n$ corresponds a tightening of the lockdown policy. Then, logically the total net cost of the measure should be positive ($h_n - h_m + \rho\Gamma_n > 0$). Given this, condition (35) simply states that the DM has to find it worth to place the economy under the most

³⁰ Note that the results are presented in the most general way, i.e., hold for any pandemic regime.

restrictive lockdown regime in the worst case scenario where a hundred percent of the population gets infected.

Proof Part 1. Suppose (32) and (33) both hold but $L_m \rightarrow L_n$ does not occur at an interior point $I^* \in (0, 1)$. We first show that $V_m(0) \neq U_m(0)$. From HJB equations (30) and (31) we find

$$\bar{U}_m(0) = \frac{h_m}{\rho}, \quad \underline{U}_m(0) = \frac{h_m + \lambda \bar{V}_m(0)}{\rho + \lambda} \quad \text{for } m = 0, 1. \tag{36}$$

In the case after mutation, by (32) $\bar{V}_m(0) \neq \bar{U}_m(0)$ since otherwise

$$\bar{V}_m(0) = \frac{h_m}{\rho} > \frac{h_n}{\rho} + \Gamma_n \geq \bar{V}_n(0) + \Gamma_n,$$

violating (7) at $I = 0$. In the case before mutation, since

$$\bar{V}_m(0) = \frac{h_n}{\rho} + \Gamma_n, \quad \bar{V}_n(0) = \frac{h_n}{\rho},$$

by (36)

$$\underline{U}_m(0) = \frac{h_m + \bar{V}_m(0)}{\rho + \lambda} > \frac{h_n}{\rho} + \Gamma_n, \quad \underline{U}_n(0) = \frac{h_n + \lambda h_n / \rho}{\rho + \lambda} = \frac{h_n}{\rho}.$$

Hence, we again have $\underline{V}_m(0) \neq \underline{U}_m(0)$. Since the regime change does not occur, we must have $V_m(I) = V_n(I) + \Gamma_n$ for $I \in [0, 1]$. This equivalent to

$$V_n(I) = V_m(I) - \Gamma_n < V_m(I) + \Gamma_m.$$

By (7) or (8) with n and m interchanged, we have

$$\rho \bar{V}_n(I) = \bar{H}_n^*(I, V'_n(I)) \quad \text{or} \quad \rho \underline{V}_n(I) = \underline{H}_n(I, \underline{V}'_n(I), \lambda \bar{V}_n(I)) \tag{37}$$

for $I \in (0, 1)$. Therefore, $V_n(I) = U_n(I)$ for $I \in (0, 1)$. However, by (33),

$$V_m(1) \leq U_m(1) < U_n(1) + \Gamma_n = V_n(1) + \Gamma_n.$$

This is a contradiction.

Part 2. Suppose (34) and (35) both hold but $L_m \rightarrow L_n$ does not occur at an interior point. We show that $V_m(0) \neq V_n(0) + \Gamma_n$. If $V_m(0) = V_n(0) + \Gamma_n$ holds, then

$$V_n(0) = V_m(0) - \Gamma_n < V_m(0) + \Gamma_m.$$

In the case after mutation, $\bar{V}_n(I)$ satisfies the first equation in (37). Therefore $\bar{V}_n(0) = h_n/\rho$. However, by (34)

$$\bar{V}_m(0) \leq \bar{U}_m(0) = \frac{h_m}{\rho} < \frac{h_n}{\rho} + \Gamma_n = \bar{V}_n(0) + \Gamma_n.$$

This is a contradiction. Hence $\bar{V}_m(0) < \bar{V}_n(0) + \Gamma_n$. In the case before mutation, Since $\bar{V}_m(0) = h_m/\rho$ and $\bar{V}_n(0) = h_n/\rho$, by (36)

$$\underline{U}_m(0) = \frac{h_m + \lambda \bar{V}_m(0)}{\rho + \lambda} = \frac{h_m}{\rho}, \quad \underline{U}_n(0) = \frac{h_n + \lambda \bar{V}_n(0)}{\rho + \lambda} = \frac{h_n}{\rho}.$$

Since $\underline{V}_n(I)$ satisfies the second equation in (37), it follows that $\underline{V}_n(0) = \underline{U}_n(0) = h_n/\rho$. This again leads to

$$\underline{V}_m(0) \leq \underline{U}_m(0) = \frac{h_m}{\rho} < \frac{h_n}{\rho} + \Gamma_n = \underline{V}_n(0) + \Gamma_n,$$

contradicting the assumption. Hence, in any case $V_m(0) \neq V_n(0) + \Gamma_n$.

By (7) and (8), $V_m(I)$ satisfies either

$$\rho \bar{V}_m(I) = \bar{H}_m^*(I, \bar{V}'_m(I)) \text{ or } (\rho + \lambda) \underline{V}_m(I) = \underline{H}_m^*(I, \underline{V}'_m(I), \lambda \bar{V}_m(I)) \tag{38}$$

for I near 0. Since $L_m \rightarrow L_n$ does not occur, it follows that $V_m(I) \leq V_n(I) + \Gamma_n$. In particular, $V_m(1) \leq V_n(1) + \Gamma_n$. Furthermore, $V_m(I)$ satisfies (38) for all $I \in (0, 1)$. Thus $V_m(I) = U_m(I)$ for all $I \in (0, 1)$. Therefore, by (35),

$$V_m(1) = U_m(1) > U_n(1) + \Gamma_n \geq V_n(1) + \Gamma_n.$$

This is a contradiction. Thus $L_m \rightarrow L_n$ must occur at an interior point.

The proof is complete. □

A.4 Proof of Proposition 1

Relations (18) and (20) directly come from the definitions of the δ s and conditions stated in Theorem 2.

Suppose $L_0 \rightarrow L_1$ does not occur at a finite time. Then $V_0(I)$ satisfies the HJB equation (27) after mutation or (28) before mutation for all $I \in (0, 1)$. Hence, $V_0(I) = U_0(I)$ for $I \in [0, 1]$. Moreover, by (7) or (8)

$$V_1(I) \leq V_0(I) \leq V_1(I) + \Gamma_1 \quad \text{for all } I \in [0, 1].$$

From HJB equations (30) and (31) we find

$$\bar{U}_m(0) = \frac{h_m}{\rho}, \quad \underline{U}_m(0) = \frac{h_m + \lambda \bar{V}_m(0)}{\rho + \lambda} \quad \text{for } m = 0, 1.$$

Since $h_1 > h_0$, it follows from $V_1 \leq V_0$ that

$$\bar{V}_1(0) = \bar{V}_0(0) = \frac{h_0}{\rho} < \bar{U}_1(0).$$

This leads to

$$\underline{U}_0(0) = \frac{h_0 + \lambda h_0 / \rho}{\rho + \lambda} = \frac{h_0}{\rho}, \quad \underline{U}_1(0) = \frac{h_0 + \lambda h_1 / \rho}{\rho + \lambda} > \frac{h_0}{\rho}.$$

Hence,

$$\underline{V}_1(0) \leq \underline{V}_0(0) = \frac{h_0}{\rho} < \underline{U}_1(0).$$

Therefore, in any case, $V_1(I) \neq U_1(I)$ for small I . Hence $V_1(I) = V_0(I)$ for small I . If there is $I \in [0, 1]$ such that $V_1(I) < V_0(I)$, then the infimum of such I is the transition point between locked down and unlockdown. That is, it is the point of reopening, I_0^* . So, (19) holds. If there is no such point $I \in [0, 1]$, then $V_1(I) = V_0(I)$ for all $I \in [0, 1]$. This proves Part 1.

Suppose reopening occurs at a finite time. Furthermore, by (14),

$$\delta_0 \equiv 2 \frac{\theta_0 + \theta_1}{\theta_0 - \theta_1} [h_1 - h_0] \leq (1 - I_0^*)^2 < 1.$$

The above inequality also implies (20).

Suppose $L_1 \rightarrow L_0$ does not occur at a finite time. Since $h_1 > h_0$, $V_1(I)$ does not satisfy the HJB equation. Hence $V_1(I) = V_0(I)$ for small I . It is not possible that $V_1(I) < V_0(I)$ for some $I \in (0, 1)$, because, otherwise, at the infimum of such I is $L_1 \rightarrow L_0$ takes place. Hence, $V_1(I) = V_0(I)$ for all $I \in [0, 1]$. This implies that $V_0(I) < V_1(I) + \Gamma_1$ for all $I \in (0, 1)$. Hence, lockdown does not happen at an interior point. Hence, by (7), $V_0(I)$ satisfies the HJB equation for all I . Therefore $V_0(I) = U_0(I)$ for $I \in [0, 1]$. This proves Part 2.

A.5 Minimum value function

A.5.1 Existence

Lemma 1 *Suppose $\theta_m > K$ and either the regime is after mutation or before mutation with $\lambda = 0$. Let*

$$I'_m = \frac{2(\theta_m - K)}{\theta_m \left[1 + \sqrt{1 + \frac{2\beta}{\rho}(\theta_m - K)} \right]}.$$

Then, there is $I''_m > I'_m$ such that for any $I_0 \in (I'_m, I''_m)$ the HJB equation

$$\rho U_m(I) = IU'_m(I) [\theta_m(1 - I) - K] - \frac{[\theta_m IU'_m(I)]^2}{2} + \rho q_m(I) \quad (39)$$

has a solution $U_m(I; I_0)$ that satisfies

$$U_m(I_0; I_0) = q_m(I_0) \quad (40)$$

and

$$U'_m(I; I_0) = \frac{1}{\theta_m I} \left[I_m - I - \sqrt{(I_m - I)^2 + 2\rho [q_m(I) - U_m(I; I_0)]} \right] \quad (41)$$

for $0 < I < I_0$.

Furthermore, $U_m(I; I_0)$ is increasing in I_0 .

To prove this, let

$$M = \min_{0 \leq I \leq 1} \left\{ \frac{(I_m - I)^2}{2\rho} + q_m(I) \right\}$$

with $I_m = 1 - K/\theta_m$, and let I''_m satisfies

$$q_m(I''_m) = M.$$

It can be shown that $I'_m < I''_m < I_m$. Let $U_m(I; I_0)$ be the solution to the HJB equation (39) with the initial condition (40), where I_0 satisfies $I'_m \leq I_0 \leq I''_m$.

We first show that $U_m(I; I_0)$ exists and is positive for all $I \in (0, I_0)$. To see that $U_m(I; I_0)$ exists for all $I \in (0, I_0)$, it suffices to show that the right-hand side of (41) is real for such I . If not, then there is some $I_1 \in (0, I_0)$ such that the quantity in the square root is positive for $I_1 < I < I_0$ and it becomes zero at $I = I_1$. This implies that

$$U_m(I_1; I_0) = \frac{(I_m - I_1)^2}{2\rho} + q_m(I_1) < U_m(I_0; I_0) = q_m(I_0).$$

However, since $q_m(I)$ is increasing and $I_0 \leq I_m''$, it follows that

$$q_m(I_0) \leq q_m(I_m'') = \min_{0 \leq I \leq 1} \left\{ \frac{(I_m - I)^2}{2\rho} + q_m(I) \right\} \leq \frac{(I_m - I_1)^2}{2\rho} + q_m(I_1).$$

This is impossible. Therefore, the right-hand side of (41) is real for all $0 < I \leq I_0$.

We next show that $U_m(I; I_0) > q_m(I)$ for $0 < I < I_0$. If this is not true, then there is $I_2 \in (0, I_0)$ such that $U_m(I; I_0) > q_m(I)$ for $I_2 < I < I_0$ and $U_m(I_2; I_0) = q_m(I_2)$. Therefore,

$$\begin{aligned} U_m'(I_2; I_0) &= \lim_{h \rightarrow 0} \frac{U_m(I_2 + h; I_0) - U_m(I_2; I_0)}{h} \\ &\geq \lim_{h \rightarrow 0} \frac{q_m(I_2 + h) - q_m(I_2)}{h} = q_m'(I_2) = \frac{\beta}{\rho} I_2 > 0. \end{aligned}$$

However, by (41), $U_m'(I_2; I_0) = 0$, contradicting the above inequalities.

This proves the existence and positivity of $U_m(I; I_0)$ for $0 < I < I_0$.

At $I = I_0$, the right-hand side of (41) vanishes, while $q_m'(I_0) > 0$. Hence, if U_m continue to satisfy (41), one would have $U_m(I; I_0) < q_m(I)$ for $I > I_0$ and is near I_0 . However, this would lead to $U_m'(I; I_0) < 0$. Hence it is necessary that

$$U_m'(I; I_0) = \frac{1}{\theta_m I} \left[I_m - I + \sqrt{(I_m - I)^2 + 2\rho [q_m(I) - U_m(I; I_0)]} \right] \tag{42}$$

for $I > I_0$.

It follows that

$$\lim_{I \rightarrow I_0^+} U_m'(I; I_0) = \frac{2(I_m - I_0)}{\theta_m I_0}.$$

To ensure $U_m(I; I_0) \leq q_m(I)$ for $I > I_0$, it is necessary that the above slope is less than that of q_m at I_0 . I.e.,

$$\frac{2(I_m - I_0)}{\theta_m I_0} \leq q_m'(I_0) = \frac{\beta}{\rho} I_0.$$

Hence

$$\beta \theta_m I_0^2 \geq 2\rho (I_m - I_0).$$

This inequality leads to

$$I_0 \geq \frac{2(\theta_m - K)}{\theta_m \left[1 + \sqrt{1 + \frac{2\beta}{\rho} (\theta_m - K)} \right]} = I_m'.$$

We next show the solution of (42) with initial condition (40) exists for all $I_0 \leq I \leq 1$. We first show that $U_m(I; I_0) < q_m(I)$ for $I > I_0$. If it is not true, then there is $I_3 > I_0$ such that $U_m(I; I_0) < q_m(I)$ for $I_0 < I < I_3$ and $U_m(I_3; I_0) = q_m(I_3)$. Hence

$$\begin{aligned} U'_m(I_3; I_0) &= \lim_{h \rightarrow 0} \frac{q_m(I_3) - U_m(I_3 - h; I_0)}{h} \\ &\geq \lim_{h \rightarrow 0} \frac{q_m(I_3) - q_m(I_3 - h)}{h} = q'_m(I_3) = \frac{\beta}{\rho} I_3. \end{aligned}$$

On the other hand, by (42)

$$U'_m(I_3; I_0) = \frac{2(I_m - I_3)}{\theta_m I_3} \text{ if } I_3 < I_m \text{ or } U'_m(I_3; I_0) = 0 \text{ if } I_3 \geq I_m.$$

The latter case is obviously impossible. The former case leads to

$$\theta_m \beta I_3^2 \leq 2\rho(I_m - I_3)$$

and so $I_3 \leq I'_m \leq I_0$. It is also impossible. So, no such I_3 exists.

Since $U_m(I; I_0) < q_m(I)$ for all $I > I_0$, the right-hand side of (42) exists and is positive for such I . This proves that $U_m(I; I_0)$ exists and is increasing for $I > I_0$.

It remains to show that $U_m(I; I_0)$ is increasing in I_0 . Suppose $I'_m \leq I'_0 < I''_0 \leq I''_m$. By definition,

$$U_m(I'_0; I'_0) = q_m(I'_0) < U_m(I'_0; I''_0).$$

Suppose there is an $I_4 \in (0, 1)$ such that $U_m(I_4; I'_0) = U_m(I_4; I''_0)$. If $I_4 < I'_0$, then both $U_m(I; I'_0)$ and $U_m(I_4; I''_0)$ are solutions to the initial value problem

$$\begin{aligned} Y'(I) &= \frac{1}{\theta_m I} \left[I_m - I - \sqrt{(I_m - I)^2 + 2\rho[q_m(I) - Y(I)]} \right] \quad \text{for } I < I_4, \\ Y(I_4) &= U_m(I_4; I'_0) = U_m(I_4; I''_0). \end{aligned}$$

This contradicts the uniqueness of solution. (Note that the right-hand side of the differential equation satisfies the Lipschitz condition.) If $I > I''_0$, then both $U_m(I; I'_0)$ and $U_m(I_4; I''_0)$ are solutions to the initial value problem

$$\begin{aligned} Z'(I) &= \frac{1}{\theta_m I} \left[I_m - I + \sqrt{(I_m - I)^2 + 2\rho[q_m(I) - Z(I)]} \right] \quad \text{for } I < I_4, \\ Z(I_4) &= U_m(I_4; I'_0) = U_m(I_4; I''_0), \end{aligned}$$

again violating the uniqueness of solution. Finally, for any $I'_0 < I < I''_0$ we have

$$U_m(I; I'_0) < q_m(I) < U_m(I; I''_0).$$

So no such I_4 exists. This proves the monotonicity of $U_m(I; I_0)$ with respect to I_0 .

The proof of the lemma is complete.

Based on the above lemma, $U_m(I; I'_m)$ is the minimum value function among all solutions to the HJB equation with least nonnegative vaccination rate $r_m(I)$ at all I .

A.5.2 Dynamics and steady state

We first show that $\dot{I} < 0$ for $I > I'_m$. By (42) with $I_0 = I'_m$,

$$\theta_m IU'_m(I; I'_m) = I_m - I + \sqrt{(I_m - I)^2 + 2\rho [q_m(I) - U_m(I; I'_m)]}.$$

Hence,

$$\theta_m IU'_m(I; I'_m) \begin{cases} > 2(I_m - I) & \text{if } I'_m < I \leq I_m \\ > 0 & \text{if } I > I_m. \end{cases}$$

In view of (24),

$$\dot{I} = \theta_m(1 - I) - K - \theta_m^2 IU'_m(I) < -\theta_m[I_m - I] \leq 0$$

if $I'_m < I \leq I_m$, and

$$\dot{I} \leq I_m - I < 0$$

if $I > I_m$. Furthermore,

$$\lim_{I \rightarrow I'_m+} \theta_m IU'_m(I) = 2(I_m - I'_m).$$

Hence,

$$\begin{aligned} \lim_{I \rightarrow I'_m+} \dot{I} &= \theta_m(1 - I'_m) - K - 2\theta_m(I_m I'_m) \\ &= -(\theta_m - K) + \frac{2(\theta_m - K)}{1 + \sqrt{1 + \frac{2\beta}{\rho}(\theta_m - K)}} < 0. \end{aligned}$$

Hence, \dot{I} has negative upper bound for $I > I'_m$. Consequently, $I(t)$ decreases to I'_m in finite time from any initial value $I(t_0) > I'_m$.

We next show that $\dot{I} > 0$ if $I < I'_m$. For such I ,

$$\begin{aligned} \theta_m IU'_m(I) &= I_m - I - \sqrt{(I_m - I)^2 + 2\rho [q_m(I) - U_m(I)]} \\ &< I_m - I \end{aligned}$$

if the regime is after mutation or before mutation with $\lambda = 0$. Hence,

$$\dot{I} = \theta_m (1 - I) - K - \theta_m^2 I U'_m (I) > 0 \quad \text{for } I < I'_m.$$

In addition, as $I \rightarrow I'_m$ from left, $\theta_m I U'_m (I) \rightarrow 0$. Hence

$$\dot{I} \rightarrow \theta_m (I_m - I'_m) = \theta_m - K - \frac{2 (\theta_m - K)}{1 + \sqrt{1 + \frac{2\beta}{\rho} (\theta_m - K)}} > 0.$$

Hence, \dot{I} has a positive lower bound for $I < I'_m$. Therefore, $I (t)$ increases to I'_m in finite time.

A.6 Proof of Proposition 2

Part 1

We define a function F_1 by

$$F_1 (I) = I \underline{V}'_1 (I; \lambda) (1 - I) (\underline{\theta}_1 - \underline{\theta}_0) - \frac{1}{2} [I \underline{V}'_1 (I; \lambda)]^2 (\underline{\theta}_1^2 - \underline{\theta}_0^2) + \alpha (h_1 - h_0) + \rho \Gamma_1$$

where $\underline{V}_1 (I; \lambda)$ satisfies

$$(\rho + \lambda) \underline{V}_1 (I; \lambda) = \underline{H}_1^* (I, \underline{V}'_1 (I; \lambda), \lambda \bar{V} (I)). \tag{43}$$

Then (13) with $m = 0$ and $n = 1$ can be written as

$$F_1 (\underline{I}_1^* (\lambda)) + \lambda [\bar{V}_1 (\underline{I}_1^* (\lambda); \lambda) - \bar{V}_0 (\underline{I}_1^* (\lambda); \lambda) + \Gamma_1] = 0. \tag{44}$$

Suppose after mutation lockdown does not occur at $\underline{I}_1^* (0)$, by (7),

$$\bar{V}_1 (I) - \bar{V}_0 (I) + \Gamma_1 > 0 \quad \text{for } I \text{ greater than and is near } \underline{I}_1^* (0).$$

It follows that the second term on the left-hand side of (44) is positive for λ positive and small. Hence

$$F_1 (\underline{I}_1^* (\lambda)) < 0 \tag{45}$$

for such λ . We show that $F_1 (I) > 0$ for $I < \underline{I}_1^* (0)$ and is near $\underline{I}_1^* (0)$. Once proven, it would imply $\underline{I}_1^* (\lambda) > \underline{I}_1^* (0)$ for λ positive and small.

In terms of \underline{H}_1^* and \underline{H}_0^* defined in (29), (44) with $\lambda = 0$ is equivalent to

$$\begin{aligned} \underline{H}_1^* (\underline{I}_1^* (0), \underline{V}'_1 (\underline{I}_1^* (0); 0), 0) &= \rho \underline{V}_1 (\underline{I}_1^* (0); 0) \\ \underline{H}_0^* (\underline{I}_1^* (0), \underline{V}'_1 (\underline{I}_1^* (0); 0), 0) &= \rho [\underline{V}_1 (\underline{I}_1^* (0); 0) + \Gamma_1]. \end{aligned}$$

The equations are quadratic in $I_1^*(0)$ $V_1'(I_1^*(0); 0)$. We write the second equation as

$$V_1'(I_1^*(0); 0) = Q_0(I_1^*(0), V_1(I_1^*(0); 0) + \Gamma_1, 0).$$

Since $I_1^*(0)$ is before any steady state, by ‘‘Appendix A.5.2’’, $I_1^*(0) < I_0'$ which is the intersection of $V_0(I)$ and

$$q_0(I) \equiv \frac{\alpha h_0}{\rho} + \frac{\beta}{2\rho} I^2.$$

Hence, at

$$Q_0(I_1^*(0), V_1(I_1^*(0); 0) + \Gamma_1, 0) = \frac{1}{\theta I_1^*(0)} \left[I_0 - I - \sqrt{(I_0 - I)^2 + 2\alpha h_0 + \beta I^2 - 2\rho [V_1(I_1^*(0); 0) + \Gamma_1]} \right].$$

Since lockdown with $\lambda = 0$ occurs at $I_1^*(0)$, it follows that

$$Q_0(I, V_1(I; 0) + \Gamma_1, 0) > V_1'(I; 0) \quad \text{for } I < I_1^*(0).$$

This inequality is equivalent to

$$H_0^*(I, V_1'(I; 0), 0) < \rho [V_1(I; 0) + \Gamma] \quad \text{for } I < I_1^*(0).$$

So

$$H_1^*(I, V_1'(I; 0), 0) = \rho V_1(I; 0) \quad \text{for all } I \in (0, 1).$$

Hence,

$$H_1^*(I, V_1'(I; 0), 0) - H_0^*(I, V_1'(I; 0), 0) > -\rho\Gamma_1 \quad \text{for } I < I_1^*(0).$$

By continuity of solutions with respect to parameters, we have

$$H_1^*(I, V_1'(I; \lambda), 0) - H_0^*(I, V_1'(I; \lambda), 0) > -\rho\Gamma_1 \quad \text{for } I < I_1^*(0)$$

if λ is close to 0. This is equivalent to $F_1(I) > 0$ for $I < I_1^*(0)$ and is near $I_1^*(0)$. Hence, (45) follows.

Suppose after mutation lockdown occurs at some $\bar{I}_1^* < I_1^*(0)$. Then, by continuity, $\bar{I}_1^* < I_1^*(\lambda)$ for λ near 0. Also by continuity, we have $I_1^*(\lambda)$ less than the least steady state before mutation. By (11),

$$(\rho + \lambda) [V_1(I_1^*(\lambda); \lambda) + \Gamma_1] = H_0^*(I_1^*(\lambda), V_1'(I_1^*(\lambda); \lambda), \lambda \bar{V}_0(I_1^*(\lambda))) \tag{46}$$

holds for any $\lambda \geq 0$. Since lockdown occurs at $I = \underline{I}_1^*(\lambda)$, it follows that

$$\underline{V}_1(I; \lambda) + \Gamma_1 > \underline{V}_0(I; \lambda), \quad \underline{V}'_0(I; \lambda) > \underline{V}'_1(I; \lambda) \quad \text{for } I < \underline{I}_1^*(\lambda).$$

Since $\underline{I}_1^*(\lambda)$ is less than the least steady state before mutation, we get

$$\begin{aligned} & \underline{Q}_0(I, V, \lambda \bar{V}_0) \\ &= \frac{1}{\theta_0 I} \left[\underline{I}_0 - I - \sqrt{(\underline{I}_0 - I)^2 + 2\alpha h_0 + \beta I^2 + 2\lambda \bar{V}_0 - 2(\rho + \lambda) V} \right] \end{aligned}$$

which is increasing in V . Hence,

$$\begin{aligned} & \underline{Q}_0(I, \underline{V}_1(I; \lambda) + \Gamma_1, \lambda \bar{V}_0(I)) > \underline{Q}_0(I, \underline{V}_0(I; \lambda), \lambda \bar{V}_0(I)) \\ &= \underline{V}'_0(I; \lambda) > \underline{V}'_1(I; \lambda) \end{aligned}$$

for $I < \underline{I}_1^*(\lambda)$. This inequality is equivalent to

$$\underline{H}_0^*(I, \underline{V}'_1(I; \lambda), \lambda \bar{V}_0(I)) < (\rho + \lambda) [\underline{V}_1(I; \lambda) + \Gamma_1].$$

It can be written as

$$\begin{aligned} & \underline{H}_0^*(I, \underline{V}'_1(I; \lambda), 0) < \rho [\underline{V}_1(I; \lambda) + \Gamma_1] - \lambda [\bar{V}_0(I) - \underline{V}_1(I; \lambda) - \Gamma_1] \\ & \text{for } I < \underline{I}_1^*(\lambda). \end{aligned} \quad (47)$$

Furthermore, $\underline{V}_1(I; \lambda)$ satisfies the HJB equation

$$(\rho + \lambda) \underline{V}_1(I; \lambda) = \underline{H}_1^*(I, \underline{V}_1(I; \lambda), \lambda \bar{V}_1(I)) \quad \text{for any } I \in (0, 1)$$

which is equivalent to

$$\underline{H}_1^*(I, \underline{V}_1(I; \lambda), 0) = \rho \underline{V}_1(I; \lambda) - \lambda [\bar{V}_1(I) - \underline{V}_1(I; \lambda)] \quad (48)$$

Note that by (7),

$$\bar{V}_0(I) = \bar{V}_1(I) + \Gamma_1 \quad \text{for } I \geq \bar{I}_1^*.$$

Subtracting the corresponding sides of (47) and (48) yields

$$\underline{H}_1^*(I, \underline{V}'_1(I; \lambda), 0) - \underline{H}_0^*(I, \underline{V}'_1(I; \lambda), 0) > -\rho \Gamma_1$$

for any I that satisfies $\bar{I}_1^* < I < \underline{I}_1^*(\lambda)$ and for any λ near 0. However, $\underline{I}_1^*(0)$ satisfies the equations (46) and (48) with $\lambda = 0$ and $I = \underline{I}_1^*(0)$. It follows that

$$\underline{H}_1^*(\underline{I}_1^*(0), \underline{V}'_1(\underline{I}_1^*(0); 0)) - \underline{H}_0^*(\underline{I}_1^*(0), \underline{V}'_1(\underline{I}_1^*(0); 0), 0) = -\rho \Gamma_1.$$

Therefore, it is necessary that $\underline{I}_1^*(0) \geq \underline{I}_1^*(\lambda)$.

Part 2

We define a function F_0 by

$$F_0(I) = I \underline{V}'_0(I; \lambda) (1 - I) (\underline{\theta}_0 - \underline{\theta}_1) - \frac{1}{2} [I \underline{V}'_0(I; \lambda)]^2 (\underline{\theta}_0^2 - \underline{\theta}_1^2) + \alpha (h_0 - h_1)$$

where $\underline{V}_0(I; \lambda)$ satisfies

$$(\rho + \lambda) \underline{V}_0(I; \lambda) = \underline{H}_0^*(I, \underline{V}'_0(I; \lambda), \lambda \bar{V}_0(I)).$$

Then (13) with $m = 1$ and $n = 0$ can be written as

$$F_0(\underline{I}_0^*(\lambda)) + \lambda [\bar{V}_0(\underline{I}_0^*(\lambda); \lambda) - \bar{V}_1(\underline{I}_0^*(\lambda); \lambda)] = 0. \tag{49}$$

In the case where after mutation reopening occurs either immediately or at some $\bar{I}_0^* \geq \underline{I}_0^*(0)$, then

$$\bar{V}_1(I) = \bar{V}_0(I) \quad \text{for } I \leq \underline{I}_0^*(0).$$

In the case where after mutation reopening occurs at $\bar{I}_0^* < \underline{I}_0^*(0)$, by (7),

$$\bar{V}_0(I) - \bar{V}_1(I) > 0 \quad \text{for } I \text{ near } \underline{I}_0^*(0),$$

it follows that the second term on the left-hand side of (49) is positive. Hence

$$F_0(\underline{I}_0^*(\lambda)) < 0. \tag{50}$$

We show that $F_0(I) > 0$ for $I > \underline{I}_0^*(0)$. Once proven, it would imply $\underline{I}_0^*(\lambda) < \underline{I}_0^*(0)$ for λ positive and small.

In terms of \underline{H}_1^* and \underline{H}_0^* defined in (29), (44) with $\lambda = 0$ is equivalent to

$$\begin{aligned} \underline{H}_0^*(\underline{I}_0^*(0), \underline{V}'_0(\underline{I}_0^*(0)), 0) &= \rho \underline{V}_0(\underline{I}_0^*(0); 0) \\ \underline{H}_1^*(\underline{I}_0^*(0), \underline{V}'_0(\underline{I}_1^*(0)), 0) &= \rho \underline{V}_0(\underline{I}_0^*(0); 0). \end{aligned}$$

The equations are quadratic in $\underline{I}_0^*(0) \underline{V}'_0(\underline{I}_0^*(0); 0)$. We write the second equation as

$$\begin{aligned} \underline{V}'_0(\underline{I}_0^*(0); 0) &= \underline{Q}_1(\underline{I}_0^*(0), \underline{V}_0(\underline{I}_1^*(0); 0), 0) \\ &\equiv \frac{1}{\underline{\theta}_1 \underline{I}_0^*(0)} \left[- (I + \underline{\varepsilon}_1) + \sqrt{(I + \underline{\varepsilon}_1)^2 + 2\alpha h_1 + \beta I^2 - 2\rho \underline{V}_0(\underline{I}_0^*(0); 0)} \right] \end{aligned}$$

where $\underline{\varepsilon}_1 = K/\theta_1 - 1$. Since reopening occurs at $\underline{I}_0^*(0)$, it follows that

$$\underline{Q}_1(I, \underline{V}_0(I; 0), 0) < \underline{V}'_0(I; 0) \quad \text{for } I > \underline{I}_0^*(0)$$

This inequality is equivalent to

$$\underline{H}_1^* (I, \underline{V}'_0 (I; 0), 0) < \rho \underline{V}_0 (I; 0) \quad \text{for } I > \underline{I}_0^* (0).$$

Also,

$$\underline{H}_0^* (I, \underline{V}'_0 (I; 0), 0) = \rho \underline{V}_0 (I; 0) \quad \text{for all } I.$$

Hence

$$\underline{H}_0^* (I, \underline{V}'_0 (I; 0), 0) - \underline{H}_1^* (I, \underline{V}'_0 (I; 0), 0) > 0$$

for $I > \underline{I}_0^* (0)$. By the continuity of solutions with respect to λ , we also have

$$\underline{H}_0^* (I, \underline{V}'_0 (I; \lambda), 0) - \underline{H}_1^* (I, \underline{V}'_0 (I; \lambda), 0) > 0$$

This is equivalent to

$$F_0 (I) > 0 \quad \text{for } I > \underline{I}_0^* (0).$$

This proves (50).

Suppose after mutation reopening occurs at some point \bar{I}_0^* such that $\underline{I}_0^* (0) < \bar{I}_0^* \leq 1$. Then

$$\bar{V}_1 (I) = \bar{V}_0 (I) \quad \text{for } I \leq \bar{I}_0^*. \tag{51}$$

By continuity, we may suppose that λ is so small such that $\underline{I}_0^* (\lambda) < \bar{I}_0^*$. By (11),

$$(\rho + \lambda) \underline{V}_0 (\underline{I}_0^* (\lambda); \lambda) = \underline{H}_1^* (\underline{I}_0^* (\lambda), \underline{V}'_0 (\underline{I}_0^* (\lambda); \lambda), \lambda \bar{V}_1 (\underline{I}_0^* (\lambda))) \tag{52}$$

holds for any $\lambda \geq 0$. Since reopening occurs at $I = \underline{I}_0^* (\lambda)$, it follows that

$$\underline{V}_0 (I; \lambda) > \underline{V}_1 (I; \lambda), \quad \underline{V}'_0 (I; \lambda) > \underline{V}'_1 (I; \lambda) \quad \text{for } I > \underline{I}_0^* (\lambda).$$

Note that $\underline{\theta}_1 < K$, it follows that

$$\begin{aligned} &\underline{Q}_1 (I, V, \lambda W) \\ &\equiv \frac{1}{\underline{\theta}_1 I} \left[- (I + \underline{\varepsilon}_1) + \sqrt{(I + \underline{\varepsilon}_1)^2 + 2\alpha h_1 + \beta I^2 + 2\lambda W - 2(\rho + \lambda) V} \right] \end{aligned}$$

is decreasing in V . Hence,

$$\underline{Q}_1 (I, \underline{V}_0 (I; \lambda), \lambda \bar{V}_1 (I)) < \underline{Q}_1 (I, \underline{V}_1 (I; \lambda), \lambda \bar{V}_1 (I)) < \underline{V}'_1 (I; \lambda) < \underline{V}'_0 (I; \lambda)$$

for $I > \underline{I}_0^*(\lambda)$. This inequality is equivalent to

$$(\rho + \lambda) \underline{V}_0(I; \lambda) > \underline{H}_1^*(I, \underline{V}'_0(I; \lambda), \lambda \bar{V}_1(I)) \quad \text{for } I > \underline{I}_0^*(\lambda),$$

which is the same as

$$\rho \underline{V}_0(I; \lambda) > \underline{H}_1^*(I, \underline{V}'_0(I; \lambda), 0) + \lambda [\bar{V}_1(I) - \underline{V}_0(I; \lambda)] \quad \text{for } I > \underline{I}_0^*(\lambda). \tag{53}$$

In addition, for any $I \in (0, 1)$, we also have

$$(\rho + \lambda) \underline{V}_0(I; \lambda) = \underline{H}_0^*(I, \underline{V}'_0(I; \lambda), \lambda \bar{V}_0(I))$$

which can be written as

$$\rho \underline{V}_0(I; \lambda) = \underline{H}_0^*(I, \underline{V}'_0(I; \lambda), 0) + \lambda [\bar{V}_0(I) - \underline{V}_0(I; \lambda)]. \tag{54}$$

Subtracting the respective sides of (53) and (54) and using (51), we find

$$\underline{H}_0^*(I; \underline{V}'_0(I; \lambda), 0) - \underline{H}_1^*(I, \underline{V}'_0(I; \lambda), 0) > 0 \quad \text{if } \underline{I}_0^*(\lambda) < I < \bar{I}_0^*$$

for λ sufficiently close to 0. However, $\underline{I}_0^*(0)$ satisfies (52) and (54) with $\lambda = 0$. That is,

$$\underline{H}_0^*(\underline{I}_0^*(0), \underline{V}'_0(\underline{I}_0^*(0); 0), 0) - \underline{H}_1^*(\underline{I}_0^*(0), \underline{V}'_0(\underline{I}_0^*(0); 0), 0) = 0.$$

This implies that $\underline{I}_0^*(0) \leq \underline{I}_0^*(\lambda)$ for λ sufficiently close to 0.

The proof is complete.

A.7 Steady states: general value functions

The other possible steady states solve

$$\theta_k(1 - I) = K + \theta_k^2 I V'_k(I).$$

Denote the left hand side as $f(I) = \theta_k(1 - I)$ and right hand side as $g(I) = K + \theta_k^2 I V'_k(I)$. At $I = 0$, we have $g(0) = K < f(0) = \theta_k$, while at $I = 1$, $g(1) = K + \theta_k^2 V'_k(1) > K > f(1) = 0$. So there exists at least one endemic StS ($I \neq 0$) as well. Furthermore $f'(I) = -\theta_k < 0$, and

$$g'(I) = \theta_k^2 V'_k(I) (1 - \epsilon(I)) \geq 0 \Leftrightarrow 1 \geq \epsilon(I)$$

where $\epsilon(I) = -\frac{IV''_k(I)}{V'_k(I)} (> 0)$ is the elasticity of marginal value function with respect to s . We observe that if this elasticity is sufficiently low for all $I \in [0, 1]$, which means that the DM is not too sensitive to a change in the number of infected, then $g(I)$ is

monotonically increasing and thus the StS is unique, $\widehat{I}_k \in (0, 1)$, and is at least locally asymptotically stable. In general however, there exist an odd number of StS featuring $I > 0$.

A.8 Proof of Proposition 3

Suppose $\theta_0 > K$. At $t = 0$, either the state is locked down or un-locked down. In the former case, if I_0^* does not exist (Case 1), then reopen occurs immediately. After reopening, the state remains open forever. Since $\theta_0 > K$, as discussed above,

$$\liminf_{t \rightarrow \infty} I(t) \geq \widehat{I}_0 > 0.$$

If I_0^* exists, then either $I(t)$ converges to a StS while the state remains locked down, or $I(t)$ approaches to I_0^* , triggering reopening. In the former case,

$$\liminf_{t \rightarrow \infty} I(t) \geq I_0^* > 0,$$

In the latter case, as discussed above, $I(t)$ does not converge to zero. Thus, in any case $I(t)$ does not converge to zero.

Suppose $\theta_0, \theta_1 \leq K$. Then either locked down or un-locked down, $I(t)$ is decreasing. So, eventually the state will be un-locked down, and $I(t)$ decreases to zero.

A.9 Extension to unknown mutations

Some of the results for the above simple model of one possible mutation can be extended to a more general case where the nature of the mutants is unknown. To be specific, we denote the set of possible mutations by $\{Z_j : j \in \mathcal{J}\}$ with \mathcal{J} a finite or countable set, and denote the transmission rate of the j th mutation under L_m by θ_{jm} . We still assume that the regimes are either not locked-down or locked-down, and use $m = 0, 1$ to represent the two types, respectively.

The transition measure $Q : M \times E \mapsto [0, 1]$ is the probability distribution

$$Q(\theta_{jm}, (\theta_{lm}, I)) = \Pr \{ \theta(t_i) = \theta_{lm} | \theta(t_i^-) = \theta_{jm} \}$$

where t_i is a jump time when a mutation occurs and $\theta(t_i^-)$ is the transmission rate right before the mutation occurs. Let $V_m(\theta_{jm}, I)$ be the value function with the lockdown level L_m . By dynamic programming, we derive

$$\begin{aligned} &(\rho + \lambda) V_m(\theta_{jm}, I) \\ &\leq I \frac{\partial V_m}{\partial I}(\theta_{jm}, I) [\theta_{jm}(1 - I) - K] - \frac{1}{2} \left[\theta_{jm} I \frac{\partial V_m}{\partial I}(\theta_{jm}, I) \right]^2 \\ &\quad + \alpha h_m + \frac{\beta}{2} I^2 + \lambda \mathbb{E}[V_m](\theta_{jm}, I), \end{aligned} \tag{55}$$

where

$$\mathbb{E} [V_m] (\theta_{jm}, I) = \sum_{l \in \mathcal{J}} V_m (\theta_{lm}, I) Q (\theta_{jm}, (\theta_{lm}, I)).$$

In addition, the DM does not take the impulse control $L_m \rightarrow L_n$ if doing so is not profitable. Hence, mode m continues if

$$V_m (\theta_{jm}, I) \leq V_n (\theta_{jn}, I) + \Gamma_n.$$

Let

$$H_m (I, p, W; \theta_{jm}) = Ip [\theta_{jm} (1 - I) - K] - \frac{1}{2} [\theta_{jm} Ip]^2 + \alpha h_m + \frac{\beta}{2} I^2 + \lambda W. \tag{56}$$

The quasi-variational inequalities take the form

$$\max \left\{ (\rho + \lambda) V_m (\theta_{jm}, I) - H_m \left(I, \frac{\partial V_m}{\partial I} (\theta_{jm}, I), \mathbb{E} [V_m] (\theta_{jm}, I); \theta_{jm} \right), V_m (\theta_{jm}, I) - V_n (\theta_{jn}, I) - \Gamma_n \right\} = 0. \tag{57}$$

The following necessary condition for the DM to take the impulse control $L_m \rightarrow L_n$ at an interior I^* , where $m, n \in \{0, 1\}$, $m \neq n$, is a counterpart of Theorem 1.

Theorem 4 *Suppose that V_n is differentiable in I . If the DM takes the impulse control $L_m \rightarrow L_n$ at an interior point I^* , then*

$$(\rho + \lambda) V_n (\theta_{jn}, I^*) = H_m \left(I^*, \frac{\partial V_n}{\partial I} (\theta_{jn}, I^*), \mathbb{E} [V_m] (\theta_{jm}, I^*); \theta_{jm} \right). \tag{58}$$

Proof Note (57) is quadratic in $\partial V_m / \partial I$. Using the least positive root, we can write

$$\frac{\partial V_m}{\partial I} (\theta_{jm}, I) = K_m (I, V_m (\theta_{jm}, I), \mathbb{E} [V_m] (\theta_{jm}, I); \theta_{jm}) \tag{59}$$

for some function K_m . By the continuity of the value functions we also have

$$V_n (\theta_{jn}, I^*) = V_m (\theta_{jm}, I^*). \tag{60}$$

Hence,

$$V_m (\theta_{jm}, I) = V_n (\theta_{jn}, I^*) + \int_{I^*}^I K_m (u, V_m (\theta_{jm}, u), \mathbb{E} [V_m] (\theta_{jm}, u); \theta_{jm}) du.$$

By optimality and the assumption that I^* is interior, we find

$$0 = \frac{\partial V_n}{\partial I} (\theta_{jn}, I^*) - K_m (I^*, V_m (\theta_{jm}, I^*), \mathbb{E} [V_m] (\theta_{jm}, I^*); \theta_{jm}). \tag{61}$$

By (60), the second term on the right-hand side is the same as

$$K_m (I^*, V_n (\theta_{jn}, I^*), \mathbb{E} [V_m] (\theta_{jm}, I^*); \theta_{jm}).$$

Hence, (61) is equivalent to (58). This completes the proof of Theorem 4.

Observe that by differentiability, $V_n (\theta_{jn}, I^*)$ also satisfies Eq. (58) with m changed to n and I changed to I^* . Subtracting the respective sides, there follows that

$$\begin{aligned} &H_n \left(I^*, \frac{\partial V_n}{\partial I} (\theta_{jn}, I^*), \mathbb{E} [V_n] (\theta_{jn}, I^*); \theta_{jn} \right) \\ &- H_m \left(I^*, \frac{\partial V_n}{\partial I} (\theta_{jn}, I^*), \mathbb{E} [V_m] (\theta_{jm}, I^*); \theta_{jm} \right) = 0. \end{aligned}$$

In terms of the specific form of the Hamiltonian H_m in (56), we have

$$\begin{aligned} &I^* \frac{\partial V_n}{\partial I} (\theta_{jn}, I^*) (\theta_{jn} - \theta_{jm}) (1 - I^*) - \frac{1}{2} \left[I^* \frac{\partial V_n}{\partial I} (\theta_{jn}, I^*) \right]^2 (\theta_{jn}^2 - \theta_{jm}^2) \\ &+ \alpha (h_n - h_m) + \lambda \{ \mathbb{E} [V_n] (\theta_{jn}, I^*) - \mathbb{E} [V_m] (\theta_{jm}, I^*) \} = 0, \end{aligned} \tag{62}$$

Based on this criterion, one can derive conditions for the DM to take an impulse control of lockdown or un-lockdown. For example, one necessary condition for the impulse control $L_m \rightarrow L_n$ under the condition $\Gamma_m + \Gamma_n > 0$ is

$$\begin{aligned} &\alpha (h_n - h_m) + \rho \Gamma_n < \frac{\beta}{2} \\ &- \frac{\beta \rho}{(\rho + \lambda) \theta_{jm}^2} \left(\frac{\theta_{jm}}{K} \right)^{\frac{\rho+\lambda}{\theta_{jm}-K}} \int_0^K \left(\frac{K-v}{\theta_{jm}-v} \right)^{\frac{\rho+\lambda}{\theta_{jm}-K}} (\theta_{jm} - v) dv \end{aligned} \tag{63}$$

if $\theta_{jm} > K$ and

$$\begin{aligned} &\alpha (h_n - h_m) + \rho \Gamma_n < \frac{\beta}{2} \\ &- \frac{\beta \rho}{(\rho + \lambda) \theta_{jm}^2} \left(\frac{K}{\theta_{jm}} \right)^{\frac{\rho+\lambda}{K-\theta_{jm}}} \int_0^{\theta_m} \left(\frac{\theta_{jm}-v}{K-v} \right)^{\frac{\rho+\lambda}{K-\theta_{jm}}} (\theta_{jm} - v) dv \end{aligned} \tag{64}$$

if $\theta_{jm} < K$. The proof is too long, thus omitted. But it is available upon request.

A.10 Other cases

A.10.1 Case 1: neither I_0^* nor I_1^* exists

In this case, $V_0 (I) = V_1 (I)$ for all $I \in [0, 1]$. If at $t = 0$ the economy is locked down, then it is optimal for the DM to reopen immediately. This also means that there is no

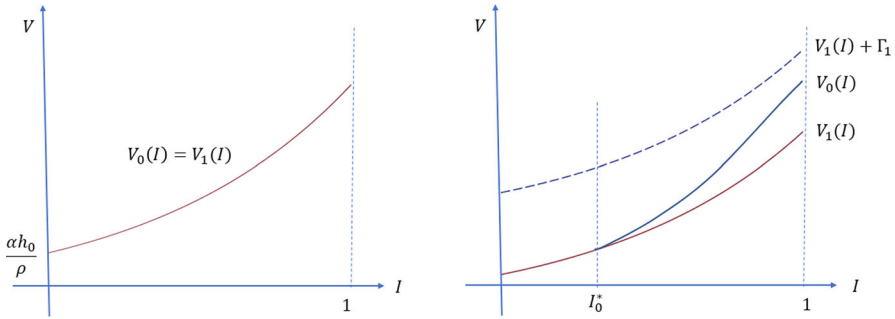


Fig. 4 Left: neither I_0^* nor I_1^* exists. Right: only I_0^* exists

room for reopening at an interior point, $t_0 \in (0, \infty)$. In the un-locked down regime, lockdown will never happen. If $\theta_0 \leq K$, the $I(t)$ decreases to zero as $t \rightarrow \infty$. If $\theta_0 > K$, then it converges to either the infimum \hat{I}_0 (if $I(0) < \hat{I}_0$), or to the supremum \tilde{I}_0 (if $I(0) > \tilde{I}_0$). This is the situation in which the impact of the pandemic is not so severe and/or the economic and social cost of a lockdown is too large for the DM to find it optimal to place the economy under a lockdown for a non-degenerated period of time (the reverse conditions of those imposed in Theorem 2 are sufficient to get this case). See figure 4, left.

A.10.2 Case 2: only I_0^* exists

Now, for $I \leq I_0^*$, we have $V_1(I) = V_0(I)$, whereas for $I_0^* < I \leq 1$, (26) holds. Then depending on the initial state of the pandemic, the impulse control decision can be the following.

If at $t = 0$ the state is locked down and $I(0) \leq I_0^*$, reopening immediately occurs. The state of the pandemic is so low that it is optimal to remove the lockdown as soon as possible. Then $I(t) \rightarrow \hat{I}_0$ if $I(0) < \hat{I}_0$ and $I(t) \rightarrow \tilde{I}_0$ if $I(0) > \tilde{I}_0$, for $\theta_0 > K$. On the other hand, if $I(0) > I_0^*$ while the state is locked down, $I(t)$ converges to a StS or decreases to I_0^* . In the latter case reopening occurs as $I(t)$ reaches I_0^* , and then the state remains open and $I(t)$ converges to a StS. So, when the initial share of infected people is sufficiently high, it is optimal for the DM to keep the lockdown in operation until the pandemic situation is under control again. Otherwise, a permanent lockdown is possible otherwise only if $\theta_1 > K$. As in Case 1, if at $t = 0$ the state is not locked-down, lockdown will not occur, and $I(t)$ will converge to a StS, positive or nil depending on $\theta_0 \gtrless K$. The corresponding value functions are depicted by Fig. 4, right.

A.11 Minimum value functions

A.11.1 After mutation

We first construct the minimum value functions \bar{V}_0 and \bar{V}_1 . Equation (27) with $\bar{\theta}_m > K$ takes the form

$$\rho \bar{V}_m(I) = I \bar{\theta}_m \bar{V}'_m(I) [\bar{I}_m - I] - \frac{[I \bar{\theta}_m \bar{V}'_m(I)]^2}{2} + h_m + \frac{\beta}{2} I^2. \tag{65}$$

We differentiate the both sides with respect to I and change the dependent variable to $\bar{r}_m(I) = I \bar{\theta}_m \bar{V}'_m(I)$. The equation is

$$\bar{r}'_m = \frac{(\rho + \bar{\theta}_m I) \bar{r}_m - \beta \bar{\theta}_m I^2}{\bar{\theta}_m I [\bar{I}_m - I - \bar{r}_m]}. \tag{66}$$

An initial condition for the minimum value function is

$$\bar{I}'_m = \frac{2 \bar{I}_m}{1 + \sqrt{1 + \frac{2\beta}{\rho} \theta_m \bar{I}_m}}, \quad \bar{r}_m(\bar{I}'_m) = \bar{I}_m - \bar{I}'_m. \tag{67}$$

After solving $\bar{r}_m(I)$ from (66) and (67), we define

$$\bar{V}_m(I) = \frac{1}{\rho} \left[\bar{r}_m(I) (\bar{I}_m - I) - \frac{[\bar{r}_m(I)]^2}{2} + h_m + \frac{\beta}{2} I^2 \right].$$

Note that \bar{I}'_m is the stable positive StS that is reached by $I(t)$ in finite time in mode m .

A.11.2 Before mutation

Equation (28) with $\underline{\theta}_m > K$ takes the form

$$(\rho + \lambda) \underline{V}_m(I) = I \underline{\theta}_m \underline{V}'_m(I) [\underline{I}_m - I] - \frac{[\underline{\theta}_m I \underline{V}'_m(I)]^2}{2} + h_m + \frac{\beta}{2} I^2 + \lambda \bar{V}_m(I). \tag{68}$$

In terms of $\underline{r}_m(I) = I \underline{\theta}_m \underline{V}'_m(I)$ in the form

$$(\rho + \lambda) \underline{V}_m(I) = \underline{r}_m(I) [\underline{I}_m - I] - \frac{[\underline{r}_m(I)]^2}{2} + h_m + \frac{\beta}{2} I^2 + \lambda \bar{V}_m(I). \tag{69}$$

By differentiation, we obtain

$$\underline{r}'_m = \frac{[\rho + \lambda + \underline{\theta}_m I] \underline{r}_m - \beta \underline{\theta}_m I^2 + \lambda \bar{r}_m \underline{\theta}_m / \bar{\theta}_m}{\underline{\theta}_m I [\underline{I}_m - I - \underline{r}_m]}. \tag{70}$$

The initial condition should be imposed at a point I'_m that satisfies

$$\begin{aligned}
 (\rho + \lambda) \underline{V}_m (I'_m) &= h_m + \frac{\beta}{2} (I'_m)^2 + \lambda \bar{V}_m (I'_m), \\
 (\rho + \lambda) \underline{V}'_m (I'_m) &= \beta I'_m + \lambda \bar{V}'_m (I'_m).
 \end{aligned}$$

By (68), the first equation is equivalent to

$$r_m (I'_m) = 2 [L_m - I'_m] \tag{71}$$

and the second equation can be written as

$$(\rho + \lambda) \frac{r_m (I'_m)}{\underline{\theta}_m} = \beta (I'_m)^2 + \lambda \frac{\bar{r}_m (I'_m)}{\bar{\theta}_m}.$$

Hence, I'_m satisfies

$$2 (\rho + \lambda) [L_m - I'_m] = \beta \underline{\theta}_m (I'_m)^2 + \lambda \frac{\underline{\theta}_m}{\bar{\theta}_m} \bar{r}_m (I'_m) \tag{72}$$

This determines I'_m .

To solve $r_m (I)$ and $\underline{V}_m (I)$, we solve Eq. (70) with two initial-value problems, one for $I > I'_m$ and the other for $I < I'_m$. For the former, we find $r_m (I'_m)$ by (71), and for the latter, we set $r_m (I'_m) = 0$. This determines $r_m (I)$. Finally, \underline{U}_m is determined by

$$\underline{U}_m (I) = \frac{1}{\rho + \lambda} \left[r_m (I) [L_m - I] - \frac{[r_m (I)]^2}{2} + h_m + \frac{\beta}{2} I^2 + \lambda \bar{V}_m (I) \right].$$

The graphs of \underline{U}_0 and \underline{U}_1 are shown in Fig. 3. Since

$$\underline{U}_0 (I) < \underline{U}_1 (I)$$

for some $I \in (0, 1)$, there is a reopening point, I_0^* . To find I_0^* , we use (13) with $n = 0$ and $m = 1$, which takes the form

$$\begin{aligned}
 r_0 (I_0^*) \left[1 - \frac{\underline{\theta}_1}{\underline{\theta}_0} \right] (1 - I_0^*) \\
 - \frac{[r_0 (I_0^*)]^2}{2} \left[1 - \left(\frac{\underline{\theta}_1}{\underline{\theta}_0} \right)^2 \right] - h_1 + \lambda [\bar{V}_0 (I_0^*) - \bar{V}_1 (I_0^*)] = 0.
 \end{aligned}$$

By computation, $I_0^* \approx 0.01788$. We next find $r_1 (I_0^*)$ by solving (69) with $m = 1$, $I = I_0^*$, and solve $r_1 (I)$ from (70) with this initial condition for $I < I_0^*$. From (69)

we find

$$\underline{V}_1(I) = \frac{1}{\rho + \lambda} \left[r_1(I) (I_1 - I) - \frac{[r_1(I)]^2}{2} + h_1 + \frac{\beta}{2} I^2 + \lambda \bar{V}_1(I) \right]$$

for $I < \underline{I}_0^*$. For $I > \underline{I}_0^*$,

$$\underline{V}_1(I) = \underline{V}_0(I) = \underline{U}_0(I),$$

and hence $r_1(I) = r_0(I)$ for $I > \underline{I}_0^*$. This determines \underline{V}_1 and r_1 for all $I \in (0, 1)$ and \underline{V}_0 and r_0 for $I > \underline{I}_0^*$. It remains to determine \underline{V}_0 and r_0 for $I < \underline{I}_0^*$. For this purpose, we need first find the lockdown point, \underline{I}_1^* .

Lockdown point The lockdown point \underline{I}_1^* exists if

$$\underline{U}_0(I) > \underline{V}_1(I) + \Gamma_1$$

for some $I \in (0, 1)$. By computation, the above inequality never holds. As a result, $\underline{V}_0(I) = \underline{U}_0(I)$ for all $I \in (0, 1)$.

The resulting value functions and vaccination rates before mutation are shown in Fig. 3 in the main text.

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