

Research Article

Paramahansa Pramanik*

Optimal lock-down intensity: A stochastic pandemic control approach of path integral

<https://doi.org/10.1515/cmb-2023-0110>

received May 20, 2023; accepted November 30, 2023

Abstract: The aim of this article is to determine the optimal intensity of lock-down measures and vaccination rates to control the spread of coronavirus disease 2019. The study uses a stochastic susceptible-infected-recovered (SIR) model with infection dynamics. A Feynman-type path integral control approach is used to derive a forward Fokker-Plank-type equation for the system, which helps in performing a stochastic control analysis. The simulation study concludes that increasing the diffusion coefficients leads to a downward trend in the susceptible and recovery curves, while the infection curve becomes ergodic. Additionally, the study shows that the optimal lock-down intensity is stable around zero, and the vaccination rate increases over time.

Keywords: stochastic control, stochastic SIR model, Feynman-type path integral, disease spread networks

MSC 2020: 93E20, 49K45, 49L20

1 Introduction

Locking down economies and increasing vaccination rates are strategies used to reduce the spread of coronavirus disease 2019 (COVID-19), which has claimed over 1.1 million lives in the United States and around seven million worldwide. It has become clear over the past couple of years that a comprehensive solution requires better epidemiology, improved economic analysis, and advanced optimization techniques to tame this pandemic. Since all mathematical epidemic models are based on the susceptible-infected-recovered (SIR) model, we assume that COVID-19 dynamics in this article follows a stochastic SIR model. Extensive research has been conducted on the dynamic behavior of different epidemic models, as detailed in [1,9, 50,55]. Stochastic modeling of pandemics is crucial when the number of infected individuals is low or when various transmission and recovery rates influence the pandemic outcome [3,46,47,49,50].

In recent times, a considerable amount of research has been conducted regarding the COVID-19 pandemic from a control point of view. Several studies, such as [4,7,10,15,18,21,26,40,63], have discussed the medical, societal, and economic impacts of the lock-down measures. These studies assume that the government is acting as a central planner that can control the epidemic in a way that benefits the population as a whole [21]. However, it is not reasonable to assume that all individuals will follow government mandates regarding COVID-19. While some people may reduce interactions with others out of fear of getting sick, others may not [21]. Therefore, individualistic points of view have been studied, and game theory has been introduced in pandemic models, as shown by Reluga [56,57], Li et al. [33], and Elie et al. [14].

The pandemic has revealed several risk factors and comorbidities, such as obesity, pre-existing health conditions like diabetes or hypertension, and advanced age, as the primary causes of spreading the virus [2,58,65]. Additionally, environmental factors such as air pollution, temperature, and humidity have contributed to the rapid spread of COVID-19 in China [22,67]. However, the impact of particulate matter 2.5 in the air

* **Corresponding author: Paramahansa Pramanik**, Department of Mathematics and Statistics, University of South Alabama, 411 North University Boulevard, Mobile, AL, 36688, USA, e-mail: ppramanik@southalabama.edu

on the spread of COVID-19 remains largely unexplored as the pandemic continues to spread [2]. Recently, raging wildfires in the western United States have increased the concentration of $PM_{2.5}$ in the air, and the study by Albrecht et al. [2] aimed to explore whether this has any impact on the spread of COVID-19.

This article presents a control approach for a quadratic cost function that uses a variation of the Feynman-type path integral method. The approach considers a forward-looking stochastic SIR model with an infection dynamics based on an Erdos-Renyi-type random network. The aim is to obtain a forward Fokker-Plank-type equation for the COVID-19 environment [37,54,66] and determine the optimal lock-down intensity and vaccination rate. The lock-down intensity is defined as the ratio of employment due to COVID-19 to total employment during the absence of this pandemic, and it ranges between 0 and 1, where 0 represents a complete shut-down of the economy. The path integral control and dynamic programming tools facilitate the analysis of this stochastic pandemic control model. An algorithm is applied to obtain a numerical solution. The assumption made throughout this article is that all agents in the pandemic environment are risk-averse. Therefore, the simulation of optimal lock-down intensity goes up at the beginning of the time interval and then approaches zero because, with complete information, an agent refuses to go out and get infected by COVID-19.

Dynamic programming is a powerful method for studying stochastic control in various fields, such as economics, where it helps to understand the decisions of economic agents and to design optimal policies for governments, particularly during a pandemic. However, it has taken a long time for the potential of dynamic programming to be realized in designing government policies to reduce the spread of infectious diseases. Since the late 1950s, dynamic programming has been applied in economics to design government decision rules that control an economy with transition laws that take into account the decisions of agents [35]. There are two main approaches to solving a dynamic programming problem: the dynamic programming principle (DPP) and the partial differential equation (PDE) approach. Let us start by discussing the DPP approach. The oldest method under DPP is the Hamiltonian method. In this method, a Hamiltonian is constructed based on the objective function, a co-state variable, and a functional form of a stochastic differential equation. A system of equations is obtained based on the first-order conditions for state, co-state, and control variables. Further solving this system yields the steady-state level of control and state variables [8]. The second method is the Lagrangian approach, which is similar to the Hamiltonian approach but uses a Lagrangian multiplier instead of the co-state variable [11]. This method avoids the need to solve the Bellman equation for the value function, making it a more convenient analytical tool. The third method is the Feynman-Kac lemma, which links parabolic PDEs with stochastic processes [16,24]. This method involves simulating random paths of a stochastic process to solve a certain type of PDE. The Hamiltonian-Jacobi-Bellman (HJB) method of the PDE approach is the most widely used technique for solving controlled processes. This method provides necessary and sufficient conditions for optimality of a control with respect to a loss function, and its solution is the value function of the optimal control problem. The mean-field game theory, introduced by Jean-Michel Lasry and Fields Medalist Pierre-Louis Lions, has generated a lot of interest in the past decade [31]. This method incorporates an infinite number of firms and constructs a forward Fokker-Plank and a backward HJB equation. Finally, this mean-field method game method solves this system of equations to obtain the optimal values of the state and control variables.

The Feynman path integral and Schrödinger quantization are two methods of quantization used in quantum physics [17,52]. While Schrödinger quantization uses a Hamiltonian function, Feynman path integral uses a quantum Lagrangian function [5,17,20,27,64]. The path integral approach provides a different viewpoint from Schrödinger's quantization, making it a useful tool not only in quantum physics but also in engineering, biophysics, economics, and finance. However, the mathematical equivalence of these two approaches is not fully proved due to the complexity of the Feynman path integral. The mathematical difficulties arise because the Feynman path integral is not an integral by means of a countably additive measure [17,23,48]. Grid-based PDE solvers become impractical in high dimensions because of their exponential memory requirements and complexity [64]. Monte Carlo schemes provide an alternative to these solvers, and this is the main idea of path integral control. Path integral control solves a class of stochastic control problems with a Monte Carlo method for the HJB equation. This approach avoids the need for a global grid of the domain of the HJB equation [27,38,60,61]. If the objective function is quadratic and the differential equations are linear, then the solution is given in terms of a number of Riccati equations. These equations can be solved efficiently [28,44,51,53].

Incorporating randomness into the HJB equation is a straightforward process. However, when a numerical solution is calculated for both deterministic and stochastic HJB equations, difficulties arise due to dimensionality [28]. The general stochastic control problem is intractable to solve computationally because it requires an exponential amount of memory and computational time since the state space needs to be discretized. As a result, it becomes exponentially large in the number of dimensions [60,61,64]. Calculating the expected values requires visiting all states, which leads to inefficient summations of exponentially large sums [28,44,64]. Therefore, path integral control is implemented to address this issue.

The structure of this article is as follows. In Section 2.1, we explain the main problem formulation and discuss the properties of stochastic SIR and the quadratic cost function. We also demonstrate that our SIR model has a unique solution under perfect and complete information. In Section 2.2, we discuss the transmission of COVID-19 in a community with Erdos-Renyi random interaction based on five different immune groups. We also consider fine particulate matter to observe the effect of air pollution on an individual infected by the pandemic in this section. In Section 2.3, we construct the system of stochastic constraints, including infection dynamics and their properties. Section 2.4 describes the main theoretical results of this study. We conducted some simulation studies for SIR and controls in Section 3 based on the results obtained from Section 2.4. Finally, Section 4 concludes this article. All the proofs are in the appendix.

2 Framework

2.1 Model

In this section, we will create a dynamic framework to minimize a social planner's cost while subject to a stochastic SIR model with pandemic spread dynamics. This article focuses on solving a stochastic optimization problem of a single agent. To simplify our model, we assume that all agents have identical objectives.

It is a strong assumption to assume that all agents have identical preferences and locations in this model. In reality, agents are likely to have varying preferences and locations, which would make our structure more complicated. One way to deal with this heterogeneity is to construct different objective functions for different agents and assign different weights based on their location. For example, if an agent lives in a densely populated area, they are more likely to be affected by the pandemic and less likely to engage in social interaction. To be more specific, we can assign weights based on each person instead of their region. We can then assume that these weights follow a normal distribution. We construct a stochastic control problem via a conditional dynamic optimization over continuous time $[0, t]$ similar to equation (1).

Since, we are considering a homogenous agent, any subscript is ignored. Following [32], an agent's objective is to minimize a cost function:

$$u^* = \min_{v, e \in \mathcal{U}} \mathbb{E}_0 \left[\int_0^t \exp(-rs) \left[S(s) \left(\frac{1}{2} \alpha_{11} v^2(s) + \alpha_{12} v(s) + \alpha_{13} \right) + I(s) \left(\frac{1}{2} \alpha_{21} e^2(s) + \alpha_{22} e(s) + \alpha_{23} \right) \right] \right. \\ \left. + \beta(e(s), v(s)) S(s) I(s) \right] ds \Bigg| \mathcal{F}_0, \quad (1)$$

subject to

$$dS(s) = \left\{ \eta N(s) - \beta(e(s), v(s)) \frac{S(s) I(s)}{[1 + \rho I(s)] + \eta N(s)} - \kappa S(s) - v(s) + \zeta R(s) \right\} ds + \sigma_1 [S(s) - S^*] dB_1(s), \\ dI(s) = \left\{ \beta(e(s), v(s)) \frac{S(s) I(s)}{[1 + \rho I(s)] + \eta N(s)} - (\mu + \kappa) I(s) - e(s) \right\} ds + \sigma_2 [I(s) - I^*] dB_2(s), \\ dR(s) = \{ \mu v(s) I(s) - [\kappa + \zeta] e(s) R(s) \} ds + \sigma_3 [R(s) - R^*] dB_3(s), \quad (2)$$

with stochastic differential infection rate β , which is a function of vaccination rate v and lock-down intensity e . In equation (1), $r \in (0, 1)$ is a continuous discounting factor; S and I represent the percentage of total population (N) susceptible to and infected with COVID-19; R is the percentage of people removed from N , where $S + I + R = N$; R includes people who got completely recovered from COVID-19 and also those people who passed away because of this pandemic. As S , I , and R are represented in terms of percentages, therefore, $N = 100$. Furthermore, in equation (1) $u^* = (v^*, e^*)$ represents the optimal level of vaccination rate and lock-down intensity, respectively. The coefficients a_{ij} for all $i = 1, 2$ and $j = 1, 2, 3$ are determined by the overall cost functions with $a_{11} > 0$ [55]. Finally, $\mathcal{F}_0$ is the σ -algebra and R starting at time $0 \in [0, t]$. Hence, $\mathbb{E}_0[\cdot] = \mathbb{E}[\cdot | S(0), I(0), R(0); \mathcal{F}_0]$, where $S(0)$, $I(0)$, and $R(0)$ are the initial conditions.

In the system of equation (2), η is the birth rate, $1/[1 + \rho I(s)]$ is a measure of inhibition effect from behavioral change of the susceptible individual, κ is the natural death rate, ζ is the rate at which recovered person loses immunity and returns to the susceptible class, and μ is the natural recovery rate. σ_1 , σ_2 , and σ_3 are assumed to be real constants and are defined as the intensity of stochastic environment, and $B_1(s)$, $B_2(s)$, and $B_3(s)$ are the standard one-dimensional Brownian motions [55]. It is important to note that the system dynamics (2) is a very general case of a standard SIR model. The variables S^* , I^* , and R^* are the steady-state level of the state variables in this system.

Assumption 1. Following set of assumptions regarding the objective function is considered:

- $\{\mathcal{F}_s\}$ takes the values from a set $\mathcal{X} \subset \mathbb{R}^4$. $\{\mathcal{F}_s\}_{s=0}^t$ is an exogenous Markovian stochastic processes defined on the probability space $(\mathcal{X}_\infty, \mathcal{F}_0, \mathcal{P})$, where $\mathcal{P}$ is the probability measure and $\mathcal{F}_\infty$ is the functional state space where each function is coming from a smooth manifold.
- For all $\{e(s), v(s), \beta(s), S(s), I(s), R(s)\}$, there exist an optimal vaccination rate and lock-down intensity $\{e^*(s), v^*(s)\}_{s=0}^t$, with initial conditions $\beta(0)$, $S(0)$, $I(0)$, and $R(0)$, which satisfy the stochastic dynamics represented by equations (1) and (2) for all continuous time $s \in [0, t]$.
- The function

$$\exp(-rs) \left[S(s) \left(\frac{1}{2} a_{11} v^2(s) + a_{12} v(s) + a_{13} \right) + I(s) \left(\frac{1}{2} a_{21} e^2(s) + a_{22} e(s) + a_{23} \right) \right] + \beta(e(s), v(s)) S(s) I(s)$$

is uniformly bounded, continuous on both the state and control spaces and, for a given $\{e(s), v(s), \beta(s), S(s), I(s), R(s)\}$, they are $\mathcal{P}$ -measurable.

- The function

$$\exp(-rs) \left[S(s) \left(\frac{1}{2} a_{11} v^2(s) + a_{12} v(s) + a_{13} \right) + I(s) \left(\frac{1}{2} a_{21} e^2(s) + a_{22} e(s) + a_{23} \right) \right] + \beta(e(s), v(s)) S(s) I(s)$$

is strictly convex with respect to the state and the control variables.

- There exists an $\varepsilon > 0$ such that for all $\{e(s), v(s), S(s), I(s), R(s)\}$,

$$\mathbb{E}_0 \left[\left| \exp(-rs) \left[S(s) \left(\frac{1}{2} a_{11} v^2(s) + a_{12} v(s) + a_{13} \right) + I(s) \left(\frac{1}{2} a_{21} e^2(s) + a_{22} e(s) + a_{23} \right) \right] + \beta(e(s), v(s)) S(s) I(s) \right| \middle| \mathcal{F}_0 \right] \geq \varepsilon.$$

The aforementioned assumption guarantees the integrability of the cost function.

Define $\mathbf{X}(s) = [\beta(s), S(s), I(s), R(s)]^T$, where T represents the transposition of a matrix such that the dynamic cost function is

$$c[u(s), \mathbf{X}(s)] = \exp(-rs) \left[S(s) \left(\frac{1}{2} a_{11} v^2(s) + a_{12} v(s) + a_{13} \right) + I(s) \left(\frac{1}{2} a_{21} e^2(s) + a_{22} e(s) + a_{23} \right) \right] + \beta(e(s), v(s)) S(s) I(s),$$

where $u(s) = [e(s), v(s)]^T$.

2.2 Spread of the pandemic

In this section, we will be discussing the spread of COVID-19 through social interactions and the differing levels of immunity among humans. The immune system is the body's primary defense against pathogens such as viruses, bacteria, fungi, protozoa, and worms. It supports the body's natural ability to defend itself and resist infections [12]. As long as the immunity level of a human is functioning properly, infections like COVID-19 go unnoticed. There are three main types of immunity levels: innate immunity (rapid response), adaptive immunity (slow response), and passive immunity [12]. To determine the interaction among people with different levels of immunity, we randomly chose a network of 30 people. We classified the immunity levels into five categories: very low, somewhat low, medium, somewhat high, and very high. The subcategories somewhat high and very high go under innate immunity, while the subcategories very low and somewhat low go under adaptive immunity. We kept passive immunity as a medium category and did not subdivide this category into natural immunity, received from the maternal side, and artificial immunity, received from medicine [12], as it is beyond the scope of this study. In Figure 1, we have created an Erdos-Renyi random network [41] of 30 agents. The colors in the figure represent the immunity level of each agent: deep magenta for very low, lighter magenta for somewhat low, white for medium, lighter green for somewhat high, and deep green for very high.

Let us take a look at Figure 1 and examine the interaction of agent 25. This agent has the lowest level of immunity against the pandemic according to our setting. Since information is perfect and complete, everyone in the network is aware of COVID-19. Agent 25 is connected to agents 2, 8, 11, 16, 19, 27, and 28, where agents 2 and 16 have the highest level of immunity. If COVID-19 hits the network and agent 25 gets infected, this person will be isolated from some of their adjacent ties. The probability of this happening is based on the level of dissimilarity in their immune systems. Additionally, agent 25 will stay with some non-adjacent agents. Figure 2 shows the random removal of the tie between agents 25 and 16, who are completely opposite in terms of their immune systems. In contrast, Figure 3 displays the random addition of a new tie between agent 25 and a previously nonadjacent agent 1. It is intuitive to think that due to COVID-19, agents with similar immune systems tend to come closer.

The temperature plays a crucial role in the spread of pandemics. When the temperature is high, more people tend to come outside of their homes and interact with each other, leading to a faster spread of the disease. To observe interactions between agents in a large network, we opted for an Erdos-Renyi random network consisting of 100 agents [42]. Among these, 21 have very low, 24 have somewhat low, 18 have medium, 20 have somewhat high, and 17 have very high immunity systems [62]. Figure 4 illustrates this type of network,

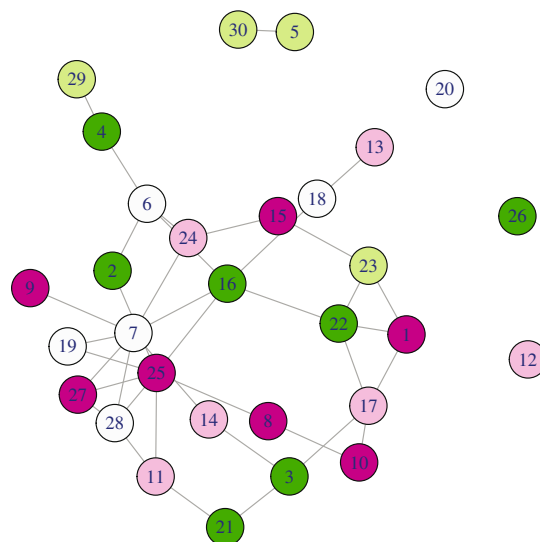


Figure 1: An Erdos-Renyi random network of 30 agents with five different immunity levels.

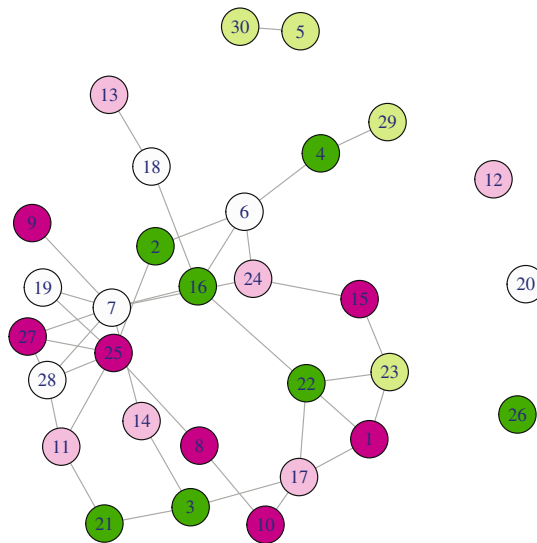


Figure 2: Tie between agents 16 and 25 is removed randomly.

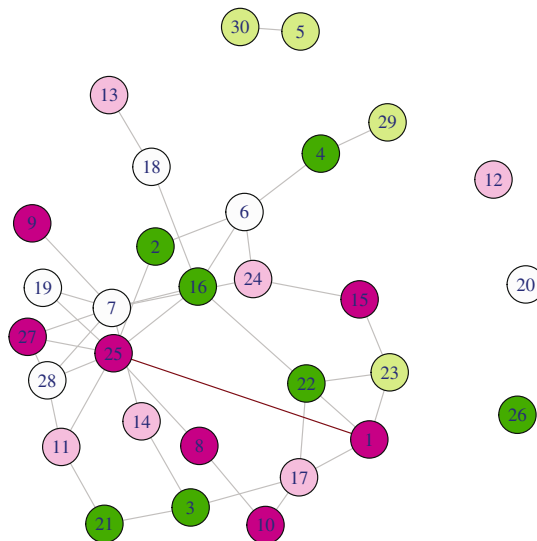


Figure 3: Tie between agents 1 and 25 is added randomly.

where agents are randomly connected and disconnected over time-based on probabilities, which are weighted by dissimilar immunity levels and the temperature of the region.

For the creation of Figure 4, an abstract concept of time is used. The process involves the selection of edges at random, with the assumption that some time has passed between each update. Initially, a list of objects is created to store an updated network [36]. Within a loop, a random node is selected and the update function is called. This function removes an existing edge and adds a new one [30]. However, there are two limitations to this procedure [19,25]. First, the loop can be replaced by a vectorized function. Second, the update function stores the entire network in each step, which results in very large objects being returned [36]. In Figure 5, we updated this large network 1,000 times. Before beginning the updating process, we assumed that this random network would become more homophilous over time, as the updates of edges are partially driven by the similarity of the immunity levels between two agents.

In Figure 5, the right panel depicts that the modularity is lower in the initial network compared to the final network after 1,000 updates. On the other hand, the left panel shows the density of the infection network. One

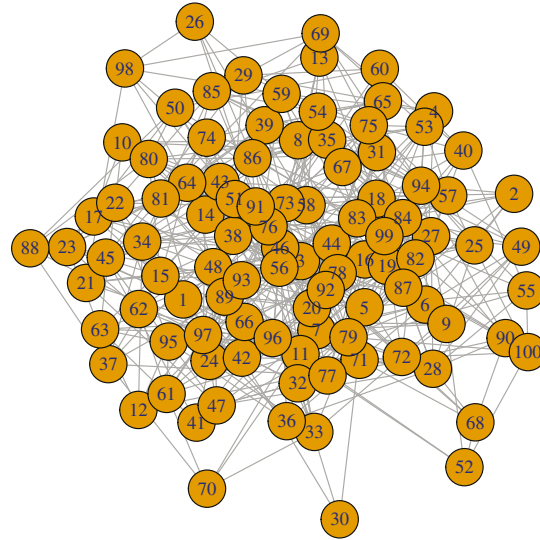


Figure 4: An Erdos-Renyi random network of 100 agents with five different immunity levels.

might assume that following the first instance of the pandemic, a greater part of the network would become segregated. The higher modularity at the end, however, suggests that edges between vertices with similar immunity levels are more likely than edges between different immunity levels.

Based on the aforementioned discussions, we construct an stochastic differential equation (SDE) of transmission rate of the pandemic β . Consider an Erdos-Renyi random network with total number of vertices V and edges $\mathcal{E}$ such that the graph becomes $G(V, \mathcal{E})$. Let $A(s)$ be the adjacency matrix with each element $a_{m_1 m_2}$ for agents m_1 and m_2 . We define the modularity as:

$$Q = \frac{1}{2\mathcal{E}} \sum_{m_1, m_2} \left[a_{m_1 m_2} - \frac{b_{m_1} b_{m_2}}{2\mathcal{E}} \right] \delta(c_{m_1}, c_{m_2}),$$

where b_{m_i} is the degree of the vertex m_i (i.e., agent m_i) for all $i = 1, 2$; c_{m_i} is the community corresponding to m_i with Kronecker delta function $\delta(.,.)$ such that if two different communities merge, δ takes the value of 1. Since, higher temperature influences the transmission rate, and higher lock-down intensity and vaccination rate reduces the transmission rate, the SDE becomes

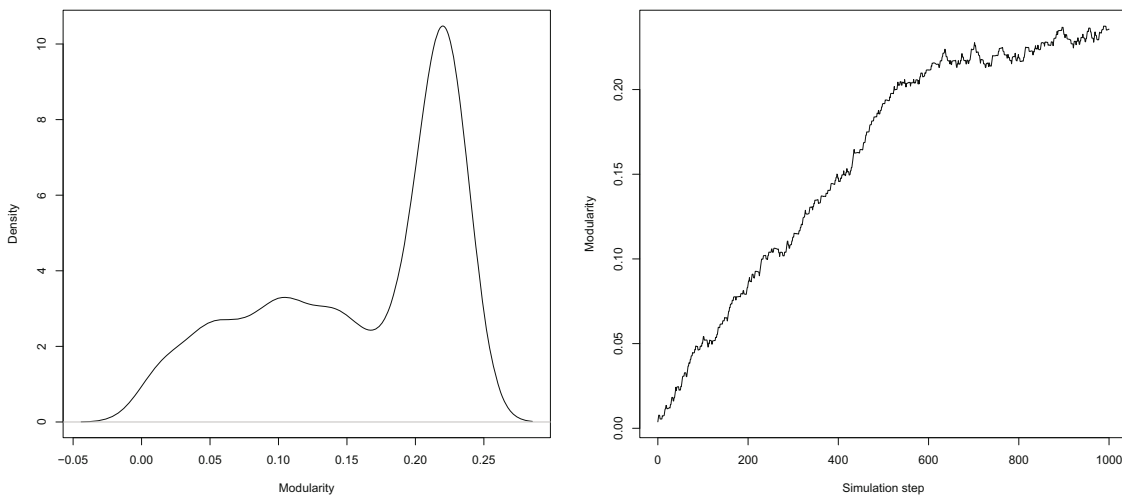


Figure 5: An Erdos-Renyi random network of 100 agents with 1,000 updates.

$$d\beta(s) = QI(s)[\beta_0 T(s) + \beta_1 M[1 - e(s)]^{\theta_1} - \beta_2 v^{\theta_2}(s)]ds + \sigma_4[\beta(s) - \beta^*]MdB_4(s), \quad (3)$$

where $\beta \in (0, 1)$ are the coefficients, $\theta_l > 1$ for all $l = 1, 2$ make the transmission function $\beta(v, e)$ a convex to e and v . Moreover, β_0 is the minimum level of infection risk produced if only the essential activities take place; β_1 is the increment in the level of infection; β_2 is the reduction in the level of infection due to vaccination; M is fine particulate matter ($PM_{2.5} > 12 \mu g/m^3$), which is an air pollutant and has a significant contribution in degrading a person's health; σ_4 is a known diffusion coefficient of infection dynamics; $dB_4(s)$ is a one-dimensional standard Brownian motion of $\beta(e, v)$ with steady-state at β^* ; and $T(s)$ is the temperature in that region at time s .

2.3 Stochastic SIR dynamics

For a complete probability space $(\mathcal{X}_\infty, \mathcal{F}_0, \mathbb{P})$ with filtration starting from $\{\mathcal{F}_s\}_{0 \leq s \leq t}$, let $\mathbf{X}(s) = [\beta(s), S(s), I(s), R(s)]^T$ be a state vector with L^2 -norm $\|\mathbf{X}(s)\|_2 = \sqrt{\beta^2(s) + S^2(s) + I^2(s) + R^2(s)}$. Furthermore, let $C^{2,1}(\mathbb{R}^4 \times (0, \infty), \mathbb{R}_+)$ be a family of all non-negative functions $Z(s, \mathbf{X})$ defined on $\mathbb{R}^4 \times (0, \infty)$ so that they are twice continuously differentiable in $\mathbf{X}$ and once in s . Define a differential operator $\mathfrak{D}$ associated with four-dimensional stochastic differential equation explained in the system of equations (2) and (3) as:

$$d\mathbf{X}(s) = \boldsymbol{\mu}(s, u, \mathbf{X})ds + \boldsymbol{\sigma}(s, \mathbf{X})d\mathbf{B}(s), \quad (4)$$

so that

$$\mathfrak{D} = \frac{\partial}{\partial s} + \sum_{j=1}^4 \boldsymbol{\mu}_j(s, u, \mathbf{X}) \frac{\partial}{\partial X_j} + \frac{1}{2} \sum_{j=1}^4 \sum_{j'=1}^4 \left[[\boldsymbol{\sigma}^T(s, \mathbf{X}) \boldsymbol{\sigma}(s, \mathbf{X})]_{jj'} \frac{\partial^2}{\partial X_j \partial X_{j'}} \right],$$

where

$$\boldsymbol{\mu} = \begin{bmatrix} \eta N(s) - \beta(e, v) \frac{S(s)I(s)}{[1 + \rho I(s)] + \eta N(s)} - \kappa S(s) - v(s) + \zeta R(s) \\ \beta(e, v) \frac{S(s)I(s)}{[1 + \rho I(s)] + \eta N(s)} - (\mu + \kappa)I(s) - e(s) \\ \mu v(s)I(s) - [\kappa + \zeta]e(s)R(s) \\ QI(s)[\beta_0 T(s) + \beta_1 M[1 - e(s)]^{\theta_1} - \beta_2 v^{\theta_2}(s)] \end{bmatrix}$$

and

$$\boldsymbol{\sigma} = \begin{bmatrix} \sigma_1(S - S^*) & 0 & 0 & 0 \\ 0 & \sigma_2(I - I^*) & 0 & 0 \\ 0 & 0 & \sigma_3(R - R^*) & 0 \\ 0 & 0 & 0 & \sigma_4(\beta - \beta^*)M \end{bmatrix}.$$

If the differential operator $\mathfrak{D}$ operates on a function $Z \in C^{2,1}(\mathbb{R}^4 \times (0, \infty); \mathbb{R}_+)$, such that

$$\mathfrak{D}Z(s, \mathbf{X}) = \frac{\partial}{\partial s}Z(s, \mathbf{X}) + \boldsymbol{\mu}(s, u, \mathbf{X}) \frac{\partial}{\partial \mathbf{X}}Z(s, \mathbf{X}) + \frac{1}{2} \text{trace} \left\{ \boldsymbol{\sigma}^T(s, \mathbf{X}) \left[\frac{\partial^2}{\partial X^T \partial X} Z(s, \mathbf{X}) \right] \boldsymbol{\sigma}(s, \mathbf{X}) \right\}, \quad (5)$$

where T represents a transposition of a matrix.

Assumption 2. For $t > 0$, let $\boldsymbol{\mu}(s, u, \mathbf{X}) : [0, t] \times [0, 1]^2 \times \mathbb{R}^4 \rightarrow \mathbb{R}$ and $\boldsymbol{\sigma}(\mathbf{X}) : \mathbb{R}^4 \rightarrow \mathbb{R}$ be some measurable functions, and for some positive constant K_0 , $\mathbf{X} \in \mathbb{R}^4$, the linear growth of the state variable is

$$|\boldsymbol{\mu}(s, u, \mathbf{X})| + |\boldsymbol{\sigma}(\mathbf{X})| \leq K_0(1 + |\mathbf{X}|),$$

such that there exists another positive, finite, constant K_1 , and for a different state variable vector $\tilde{\mathbf{X}}$, there exists the Lipschitz condition:

$$|\boldsymbol{\mu}(s, u, \mathbf{X}) - \boldsymbol{\mu}(s, u, \tilde{\mathbf{X}})| + |\boldsymbol{\sigma}(\mathbf{X}) - \boldsymbol{\sigma}^k(\tilde{\mathbf{X}})| \leq K_1 |\mathbf{X} - \tilde{\mathbf{X}}|,$$

for all $\tilde{\mathbf{X}} \in \mathbb{R}^4$, and

$$|\boldsymbol{\mu}(s, u, \mathbf{X})|^2 + |\boldsymbol{\sigma}(\mathbf{X})|^2 \leq K_1^2(1 + |\tilde{\mathbf{X}}|^2).$$

Remark 1. The Lipschitz condition of Assumption 2 guarantees that the stochastic dynamics represented by equation (4) has a unique solution by the Picard-Lindelöf theorem. The beauty of this simplistic assumption is its powerful intuitive meaning: in a particular region, the government implements all the available measures that make medical sense, comes up with a unique “lock-down intensity” and “vaccination rate.”

Assumption 3. Assume that $(\mathcal{X}_\infty, \mathcal{F}_0, \mathcal{P})$ is the stochastic basis where the filtration $\{\mathcal{F}_s\}_{0 \leq s \leq t}$ supports a four-dimensional Brownian motion $\mathbf{B}(s) = \{\mathbf{B}(s)\}_{0 \leq s \leq t}$. $\mathcal{F}_0$ is the σ -algebra generated by the Brownian motion $\mathbf{B}$, and the subspaces are

$$\mathbb{F}^2 := \left\{ \mathbf{X} \in \mathcal{F}_0; \mathbb{E}_0 \left[\int_0^t |\mathbf{X}(s)|^2 ds \right] < \infty \right\}$$

and,

$$\mathbb{S}^2 := \{ \mathbf{X} \in ; \mathbb{E}_0 [\sup_{0 \leq s \leq t} |\mathbf{X}(s)|^2] < \infty,$$

where $\mathcal{F}_0$ is a Borel σ -algebra and $\mathcal{P}$ is a probability measure [8]. Furthermore, the four-dimensional Brownian motion corresponding to the vector of state variables in this system is defined as:

$$\mathbf{B} := \{ \mathbf{X} \in \mathcal{F}_0; \sup_{0 \leq s \leq t} |\mathbf{X}(s)| < \infty; \mathcal{P}\text{-a.s.} \}.$$

Remark 2. The first part of Assumption 3 defines two subspaces $\mathbb{F}^2$ and $\mathbb{S}^2$. The first subspace $\mathbb{F}^2$ says that all expected values of the squared integrals of $\mathbf{X}(s)$ are finite. Along with the probability space $(\mathcal{X}_\infty, \mathcal{F}_0, \mathcal{P})$, this guarantees that the integral form of equation (4) is a strict contraction in the Hilbert space. $\mathbf{B}$ in Assumption 3 defines a subspace where absolute value of $\mathbf{X}(s)$ is finite with probability measure $\mathcal{P}$ almost surely. By combining the aforementioned two assumptions, make sure that equation (4) has a finite unique solution in the Hilbert space.

Proposition 1. Consider a small continuous time interval $[s, \tau] \in (0, t)$. Assume that the left-hand side of equation (5) is zero, and $\mathcal{Z}$ solves the Cauchy problem (5) with terminal condition $\mathcal{Z}(\tau, \mathbf{X}) = \Phi(\mathbf{X})$. Let the vector of pandemic state variables $\mathbf{X}$ follow the stochastic differential equation (4) so that

$$\boldsymbol{\sigma}(s, \mathbf{X}) \frac{\partial}{\partial \mathbf{X}} \mathcal{Z}(s, \mathbf{X}) \in L^2, \quad \text{for all } s \leq \tau, \quad \mathbf{X} \in \mathbb{R}^4,$$

then $\mathcal{Z}$ has the stochastic Feynman-Kac representation:

$$\mathcal{Z}(s, \mathbf{X}) = \mathbb{E}_s[\Phi(\mathbf{X}(\tau))].$$

Proposition 2. If Assumption 2 holds, then for $s \in [0, t]$, the pandemic dynamics expressed in equation (4) has a strong unique solution.

Proposition 3. Let the initial state variable of SIR model with stochastic infection $\mathbf{X}(0) \in \mathbb{L}^2$ be independent of Brownian motion $\mathbf{B}(s)$, and the drift and diffusion coefficients $\boldsymbol{\mu}(s, u, \mathbf{X})$ and $\boldsymbol{\sigma}(s, \mathbf{X})$, respectively, follow Assumptions 2 and 3. Then, the pandemic dynamics in equation (4) is in space of the real-valued process

with filtration $\{\mathcal{F}_s\}_{0 \leq s \leq t}$, and this space is denoted by $\mathcal{F}_0$. Moreover, for some finite constant $c_0 > 0$, continuous time $s \in [0, t]$, and Lipschitz constants μ and σ , the solution satisfies,

$$\mathbb{E}\left\{\sup_{0 \leq s \leq t} |\mathbf{X}(s)|^2\right\} \leq c_0\{1 + \mathbb{E}[|\mathbf{X}(0)|^2]\} \exp(c_0 t). \quad (6)$$

Propositions 1–3 tell us about the uniqueness and measurability of the system of stochastic SIR dynamics with infection dynamics. It is important to know that we assume the information available regarding the pandemic is complete and perfect and all the agents in the system risk-averse person. Therefore, once a person in a community gets infected by COVID 19, everybody gets information immediately, and that agent becomes isolated from the rest.

2.4 Main results

An agent's objective is to minimize the quadratic cost function expressed in equation (1) subject to the dynamic system represented by equations (2) and (3). Following [43], the quantum Lagrangian of an agent in a pandemic environment is

$$\mathcal{L}(s, u, \mathbf{X}) = \mathbb{E}_s\{c[u(s), \mathbf{X}(s)] + \lambda[\mu(s, u, \mathbf{X})ds + \sigma(s, \mathbf{X})d\mathbf{B}(s)]\}, \quad (7)$$

where $\mathbb{E}_s[\cdot] = \mathbb{E}[\cdot | \mathbf{X}(s), \mathcal{F}_s]$. In equation (7), $\lambda > 0$ is a time-independent quantum Lagrangian multiplier. At time s , an agent can predict the severity of pandemic based on all information available regarding state variables at that time; moreover, throughout interval $[s, s + \varepsilon]$, the agent has the same conditional expectation, which ultimately gets rid of the integration.

Proposition 4. For any two different immunity groups, if the probability measures of getting affected by the pandemic are $\mathcal{P}_1$ and $\mathcal{P}_2$, respectively, on $H \in (\mathcal{X}_\infty, \mathcal{P}, \mathcal{F}_0)$ so that the total variation difference between $\mathcal{P}_1$ and $\mathcal{P}_2$ is

$$\|\mathcal{P}_1 - \mathcal{P}_2\|_{tv} = \sup\{|\mathcal{P}_1(\mathcal{L}) - \mathcal{P}_2(\mathcal{L})|, \text{ for all } \mathcal{L} \in H\} \quad (8)$$

$$= 1 - \sup_{\eta \in \mathcal{P}_1, \mathcal{P}_2} \eta(H) \quad (9)$$

$$= 1 - \inf \sum_{k=1}^K [\mathcal{P}_1(B_k) \wedge \mathcal{P}_2(B_k)], \quad (10)$$

where $B_k \subset H$ so that $\bigcap_{k=1}^K B_k = \emptyset$ for all $k \in [1, K]$ and $K \geq 1$.

Remark 3. In Proposition 4, B_k is a set of communities of agents such that no two of them never socially interact. Furthermore, Proposition 4 tells us that if the same variant of COVID-19 hits a community with two agents differed by their immunities, total variation of infection is the supremum of two infection probabilities of their quantum Lagrangians.

Theorem 1. Consider an agent's objective is to minimize $M[\mathbf{X}(s)]$ subject to the stochastic dynamic system explained in equation (4) such that Assumptions 1–3 and Propositions 1–4 hold. For a C^2 -function $\tilde{f}(s, \bar{\mathbf{Z}})$ and for all $s \in [0, t]$, there exists a function $g(s, \mathbf{X}) \in C^2([0, t] \times \mathbb{R}^4)$ such that $\tilde{\mathbf{Y}} = g(s, \mathbf{X})$ with an Itô process $\tilde{\mathbf{Y}}$ optimal “lock-down” intensity e^* and vaccination rate v^* are the solutions

$$-\frac{\partial}{\partial u} \tilde{f}(s, \bar{\mathbf{Z}}) \Psi_s^\tau(\mathbf{X}) = 0, \quad (11)$$

where Ψ_s^τ is some transition wave function in $\mathbb{R}^4$.

3 Computation

Theorem 1 gives the solution of an optimal “lock-down” intensity and vaccination rate for a generalized stochastic pandemic system. Consider a function $g(s, \mathbf{X}) \in C^2([0, t] \times \mathbb{R}^4)$ such that [55]

$$g(s, \mathbf{X}) = [s\beta - 1 - \ln(\beta)] + [sS - 1 - \ln(S)] + [sI - 1 - \ln(I)] + [sR - 1 - \ln(R)],$$

with $\partial g / \partial s = S + I + R + \beta$, $\partial g / \partial x_i = s - 1/x_i$, $\partial^2 g / \partial x_i^2 = -1/x_i^2$, and $\partial^2 g / \partial x_i \partial x_j = 0$, for all $i \neq j$, where x_i is i th state variable of $\mathbf{X}$ for all $i = 1, \dots, 4$, and $\ln$ stands for natural logarithm. In other words, $x_1 = \beta$, $x_2 = S$, $x_3 = I$, and $x_4 = R$. Therefore,

$$\begin{aligned} \tilde{f}(s, \bar{Z}) = & \exp(-rs) \left[S \left(\frac{1}{2} a_{11} v^2 + a_{12} v + a_{13} \right) + I \left(\frac{1}{2} a_{21} e^2 + a_{22} e + a_{23} \right) \right] + \beta SI + [s\beta - 1 - \ln(\beta)] \\ & + [sS - 1 - \ln(S)] + [sI - 1 - \ln(I)] + [sR - 1 - \ln(R)] + (\beta + S + I + R) + \left(s - \frac{1}{\beta} \right) \\ & \times QI[\beta_0 T + \beta_1 M(1 - e)^{\theta_1} - \beta_2 v^{\theta_2}] + \left(s - \frac{1}{S} \right) \left[\eta N - \frac{\beta SI}{(1 + \rho I) + \eta N} - \kappa S - v + \zeta R \right] \\ & + \left(s - \frac{1}{I} \right) \left[\frac{\beta SI}{(1 + \rho I) + \eta N} - (\mu + \kappa) I - e \right] + \left(s - \frac{1}{R} \right) [\mu v I - (\kappa + \zeta) e R] \\ & - \frac{1}{2} \left\{ \sigma_1 (S - S^*) \frac{1}{S^2} + \sigma_2 (I - I^*) \frac{1}{I^2} + \sigma_3 (R - R^*) \frac{1}{R^2} + \sigma_4 (\beta - \beta^*) \frac{1}{\beta^2} \right\}. \end{aligned}$$

In order to satisfy equation (A8), either $\frac{\partial \tilde{f}}{\partial u} = 0$ or $\Psi_s^\tau = 0$. As Ψ_s^τ is a wave function, it cannot be zero. Therefore, $\frac{\partial \tilde{f}}{\partial u} = 0$ for all $u = \{e, v\}$. Therefore, for $\theta_1 = 2$, the lock-down intensity is,

$$e^* = \frac{A_2 + A_3}{A_1 + A_2},$$

Table 1: Parametric values taken from [9] and [55]

Parameter values and initial state variable values		
Variable	Value	Description
η	0.001	Birth rate
β	1	Initial infection
β_0	0	Minimal level of infection
β_1	0.2	Increment in the level of infection
β_3	0.2	Reduction in the level of infection due to vaccination
$e(0)$	1	Initial lock-down intensity
κ	0.2	Death rate
ζ	0.001	Rate by which recovered get susceptible again
μ	0.3	Natural recovery rate
ρ	0.5	Psychological or inhibitory coefficient
θ_l	2	Convexity coefficient of transmission function
M	12.5	Fine particulate matter
Q	0.5	Modularity of network
$S(0)$	99.8	Initial susceptible population
$I(0)$	0.1	Initial infected population
$R(0)$	0.1	Initial recovered population
v	0.674	Stable fully vaccination rate
a_{ij}	$\frac{1}{3}$	Coefficients of cost function

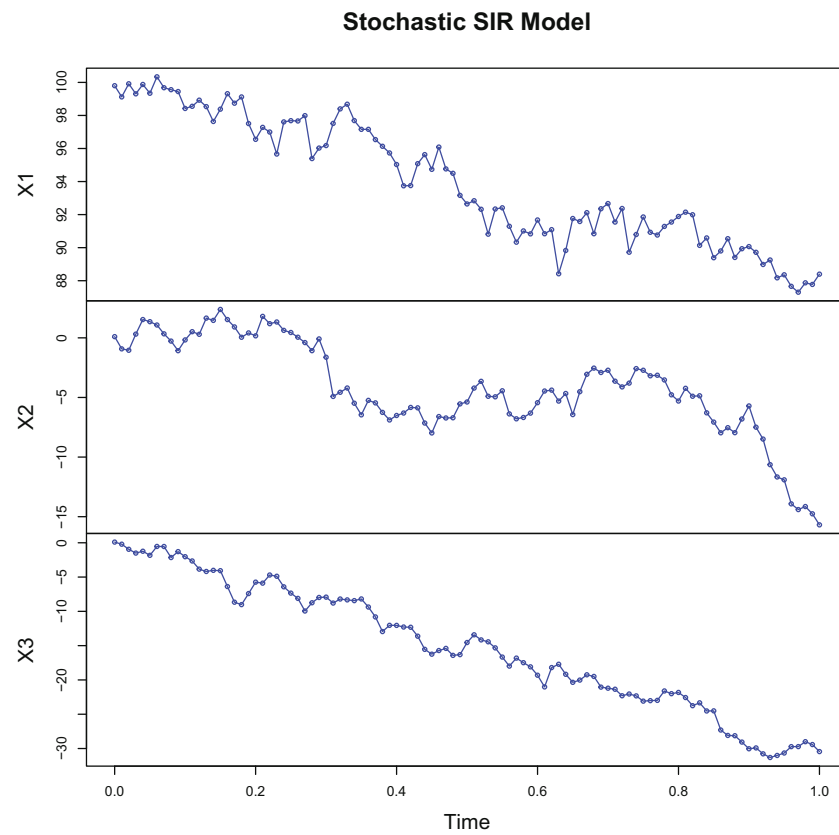


Figure 6: SIR model with higher volatility.

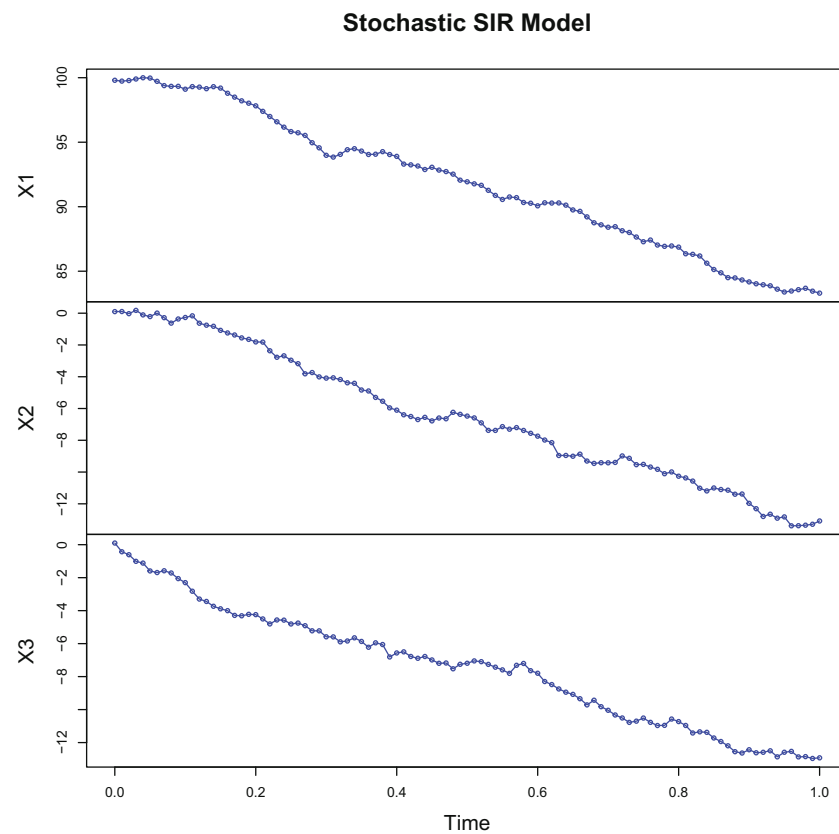


Figure 7: Model with $\sigma_1 = 0.05$, $\sigma_2 = 0.01$, and $\sigma_3 = 0.03$.

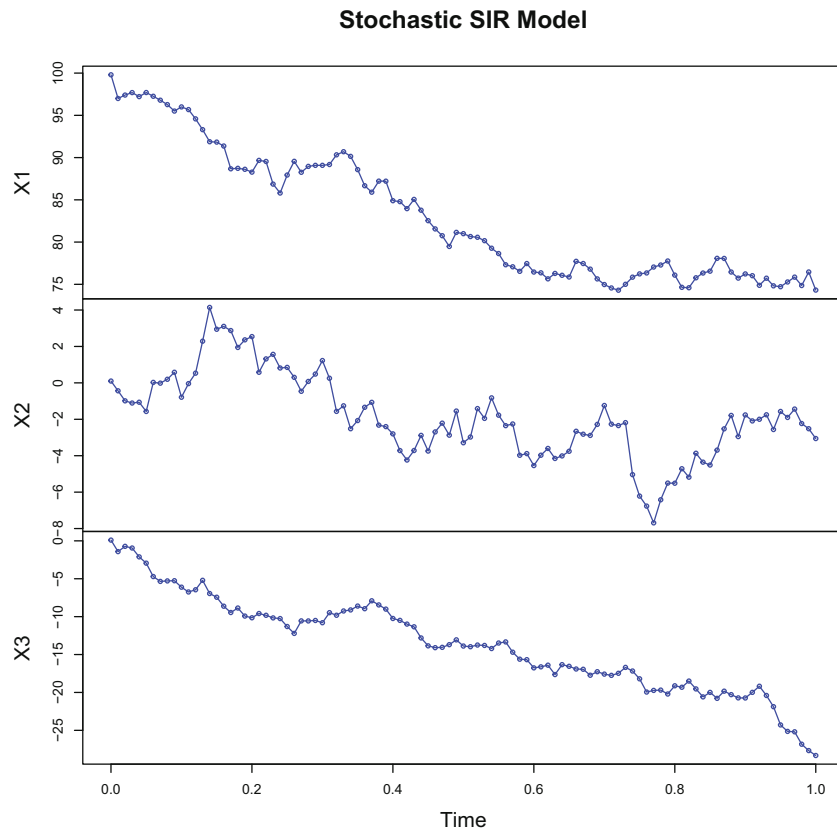


Figure 8: Model with $\sigma_1 = 0.1$, $\sigma_2 = 0.06$, and $\sigma_3 = 0.12$.

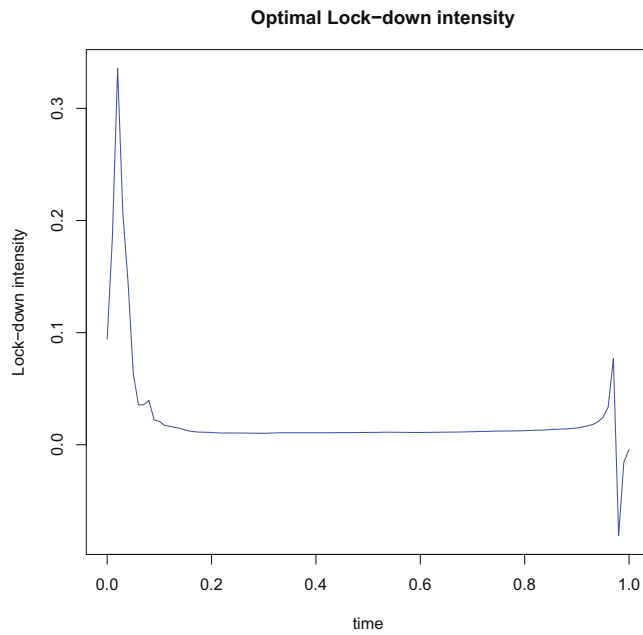


Figure 9: Lock-down with diffusion coefficients $\sigma_2 = 0.06$ and $\sigma_3 = 0.12$.

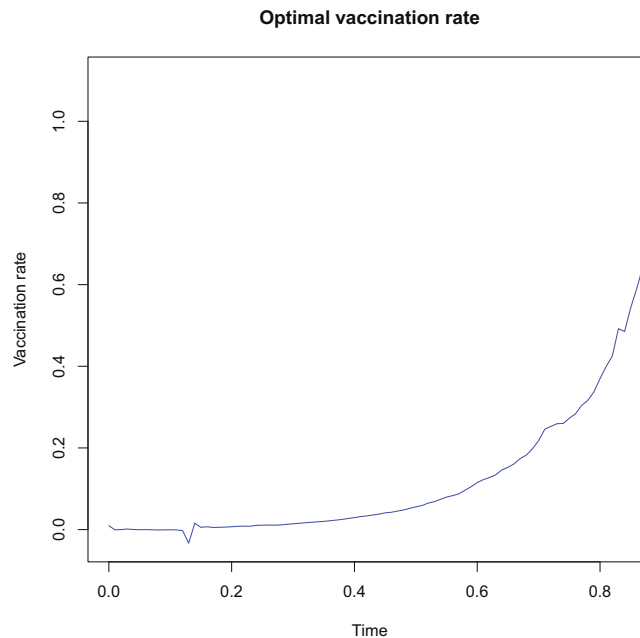


Figure 10: Vaccination with $\sigma_1 = 0.1$, $\sigma_2 = 0.06$, and $\sigma_3 = 0.12$.

where $A_1 = \exp(-rs)Ia_{21}$, $A_2 = 2QI\beta_1 M \left(s - \frac{1}{\beta} \right)$, and $A_3 = \left(s - \frac{1}{I} \right) + R \left(s - \frac{1}{R} \right) (\kappa + \zeta) - \exp(-rs)Ia_{22}$. On the other hand, for $\theta_2 = 2$, the vaccination rate is

$$v^* = \frac{B_3}{B_1 - B_2},$$

where $B_1 = \exp(-rs)Sa_{11}$, $B_2 = 2QI\beta_2 \left(s - \frac{1}{\beta} \right)$, and $B_3 = \left(s - \frac{1}{S} \right) - \mu I \left(s - \frac{1}{R} \right) - \exp(-rs)Sa_{12}$ so that $B_1 > B_2$.

Values from Table 1 have been used to do the simulation studies. These values and initial state variables are obtained from [9] and [55]. We did simulate the stochastic SIR model 100 times with different diffusion coefficients. Figure 6 assumes $\sigma_1 = 0.1$, $\sigma_2 = 0.06$, and $\sigma_3 = 0.12$.

Since the diffusion coefficients are relatively high, we can see more fluctuations. In order to observe the behavior of each of the susceptible (S), infected (I), and recovered (R) curves, we construct Figures 7 and 8. In these figures, X_1 , X_2 , and X_3 curves represent S, I, and R, respectively. When the diffusion coefficients are low, all the three curves have a downward pattern as in Figure 7. Once these coefficients increased to $\sigma_1 = 0.1$, $\sigma_2 = 0.06$, and $\sigma_3 = 0.12$, the X_2 curve in Figure 8 starts to behave ergodically, while X_1 and X_3 keep their downward trends with more fluctuations. Figures 9 and 10 represent the behavior of optimal lock-down intensity and vaccination rate over time. Our model says, under higher volatility of the pandemic, the vaccination rate is increasing over time because people are *risk-averse* and the information regarding this disease is *perfect* and *complete*.

On the other hand, under $\sigma_2 = 0.06$ and $\sigma_3 = 0.12$, Figure 9 implies that initially, people did not know about the severity of the disease, and therefore, they come outside their homes and work. Slowly, they become afraid of being infected and stopped going out, and finally, very close to the terminal point, the intensity increased because of high vaccination rate (i.e., Figure 10).

4 Conclusion

In this research article, we analyze a stochastic pandemic SIR model that incorporates a non-linear incidence rate of $\beta SI/(1 + \rho I + \eta N)$ and a stochastic dynamic infection rate. Due to the curse of dimensionality, we use a

Feynman-type path integral approach to determine the optimal lock-down intensity and vaccination rate, as simulating an HJB equation is nearly impossible. The main contribution of this study is the demonstration of the global stability and uniqueness of the control variables when the information is both perfect and complete.

To study the infection dynamics, we categorized the immunity level into five subcategories: very low, somewhat low, medium, somewhat high, and very high. We used the Erdos-Renyi random graph model to investigate the infection rate among agents with different levels of immunity. Our goal was to minimize an agent's individual cost of COVID-19 while taking into account the stochastic SIR and infection dynamics. Using a Feynman-type path integral approach, we were able to determine a Fokker-Plank type equation and obtain an optimal lock-down intensity and vaccination rate. We also conducted simulation studies based on the parameters in [9] and [55]. Since we assumed that all agents in the pandemic environment are risk-averse, the optimal lock-down intensity was initially high and then decreased to almost zero. This is because, due to the availability of perfect and complete information, individuals do not want to go out and risk getting infected by the pandemic. At the end of our study, we observed that the lock-down intensity had slightly improved, although the optimal vaccination rate had increased over the time interval we studied.

Funding information: This research received no specific grant from any funding agency, commercial, or nonprofit sectors.

Conflict of interest: The authors have no conflicts of interest to disclose.

Ethical approval: This research did not require ethical approval.

Data availability statement: Data sharing is not applicable to this article as no new data were created or analyzed in this study.

References

- [1] Acemoglu, D., Chernozhukov, V., Werning, I., & Whinston, M. D. (2020). *A multi-risk SIR model with optimally targeted lock-down* (Vol. 2020). Cambridge, MA: National Bureau of Economic Research.
- [2] Albrecht, L., Czarnecki, P., & Sakelaris, B. (2021). *Investigating the relationship between air quality and covid-19 transmission*. arXiv: <http://arXiv.org/abs/arXiv:2103.13494>.
- [3] Allen, L. J. (2017). A primer on stochastic epidemic models: Formulation, numerical simulation, and analysis. *Infectious Disease Modelling*, 2(2), 128–142.
- [4] Anderson, R. M., Heesterbeek, H., Klinkenberg, D., & Hollingsworth, T. D. (2020). How will country-based mitigation measures influence the course of the covid-19 epidemic? *The Lancet*, 395(10228), 931–934.
- [5] Anderson, R. N., Boulanger, A., Powell, W. B., & Scott, W. (2011). Adaptive stochastic control for the smart grid. *Proceedings of the IEEE*, 99(6), 1098–1115.
- [6] Baaquie, B. E. (1997). A path integral approach to option pricing with stochastic volatility: Some exact results. *Journal de Physique I*, 7(12), 1733–1753.
- [7] Bayraktar, E., Cohen, A., & Nellis, A. (2021). A macroeconomic sir model for covid-19. *Mathematics*, 9(16), 1901.
- [8] Carmona, R. (2016). *Lectures on BSDEs, stochastic control, and stochastic differential games with financial applications*. Philadelphia: SIAM.
- [9] Caulkins, J. P., Grass, D., Feichtinger, G., Hartl, R. F., Kort, P. M., Prskawetz, A., ..., Wrzaczek, S. (2021). The optimal lock-down intensity for covid-19. *Journal of Mathematical Economics*, 93, 102489.
- [10] Charpentier, A., Elie, R., Laurière, M., & Tran, V. C. (2020). Covid-19 pandemic control: Balancing detection policy and lock-down intervention under icu sustainability. *Mathematical Modelling of Natural Phenomena*, 15, 57.
- [11] Chow, G. C. (1996). The Lagrange method of optimization with applications to portfolio and investment decisions. *Journal of Economic Dynamics and Control*, 20(1–3), 1–18.
- [12] Chowdhury, M. A., Hossain, N., Kashem, M. A., Shahid, M. A., & Alam, A. (2020). Immune response in covid-19: A review. *Journal of Infection and Public Health*, 13(11), 1619–1629.
- [13] Del Moral, P. (2004). *Feynman-Kac formulae: Genealogical and interacting particle systems with applications* (Vol. 88). New York: Springer.

- [14] Elie, R., Hubert, E., & Turinici, G. (2020). Contact rate epidemic control of covid-19: An equilibrium view. *Mathematical Modelling of Natural Phenomena*, 15, 35.
- [15] Ferguson, N., Laydon, D., Nedjati-Gilani, G., Imai, N., Ainslie, K., Baguelin, M., ..., Dighe, A. A. (2020). Report 9: Impact of non-pharmaceutical interventions (NPIs) to reduce covid19 mortality and healthcare demand. *Imperial College London*, 10(77482), 491–497.
- [16] Feynman, R. P. (1949). Space-time approach to quantum electrodynamics. *Physical Review*, 76(6), 769.
- [17] Fujiwara, D. (2017). *Rigorous time slicing approach to Feynman path integrals*. Tokyo: Springer.
- [18] Hatchimonji, J. S., Swendiman, R. A., Seamon, M. J., & Nance, M. L. (2020). Trauma does not quarantine: Violence during the covid-19 pandemic. *Annals of Surgery*, 272(2), e53.
- [19] Hertweck, K. L., Vikramdeo, K. S., Galeas, J. N., Marbut, S. M., Pramanik, P., Yunus, F., ..., Dasgupta, S. (2023). Clinicopathological significance of unraveling mitochondrial pathway alterations in non-small-cell lung cancer. *The FASEB Journal*, 37(7), e23018.
- [20] Hua, L., Polansky, A., & Pramanik, P. (2019). Assessing bivariate tail non-exchangeable dependence. *Statistics & Probability Letters*, 155, 108556.
- [21] Hubert, E., Mastrolia, T., Possamai, D., & Warin, X. (2022). Incentives, lock-down, and testing: From Thucydides analysis to the covid-19 pandemic. *Journal of Mathematical Biology*, 84(5), 1–48.
- [22] Jiang, Y., Wu, X.-J., & Guan, Y.-J. (2020). Effect of ambient air pollutants and meteorological variables on COVID-19 incidence. *Infection Control & Hospital Epidemiology*, 41(9), 1011–1015.
- [23] Johnson, G. W., & Lapidus, M. L. (2000). *The Feynman integral and Feynman operational calculus*. New York: Clarendon Press.
- [24] Kac, M. (1949). On distributions of certain wiener functionals. *Transactions of the American Mathematical Society*, 65(1), 1–13.
- [25] Kakkat, S., Pramanik, P., Singh, S., Singh, A. P., Sarkar, C., & Chakroborty, D. (2023). Cardiovascular complications in patients with prostate cancer: Potential molecular connections. *International Journal of Molecular Sciences*, 24(8), 6984.
- [26] Kantner, M., & Koprucki, T. (2020). Beyond just “flattening the curve”: Optimal control of epidemics with purely non-pharmaceutical interventions. *Journal of Mathematics in Industry*, 10(1), 1–23.
- [27] Kappen, H. J. (2005). Path integrals and symmetry breaking for optimal control theory. *Journal of Statistical Mechanics: Theory and Experiment*, 2005(11), P11011.
- [28] Kappen, H. J. (2007). An introduction to stochastic control theory, path integrals and reinforcement learning. In *AIP Conference Proceedings* (Vol. 887, pp. 149–181). American Institute of Physics.
- [29] Karatzas, I., & Shreve, S. (2012). *Brownian motion and stochastic calculus* (Vol. 113). New York: Springer Science & Business Media.
- [30] Khan, M. A., Acharya, S., Anand, S., Sameeta, F., Pramanik, P., Keel, C., ..., Singh, A. P. (2023). Myb exhibits racially-disparate expression, clinicopathologic association, and predictive potential for biochemical recurrence in prostate cancer. *iScience*.
- [31] Lasry, J.-M., & Lions, P.-L. (2007). Mean field games. *Japanese Journal of Mathematics*, 2(1), 229–260.
- [32] Lesniewski, A. (2020). *Epidemic control via stochastic optimal control*. arXiv: <http://arXiv.org/abs/arXiv:2004.06680>.
- [33] Li, J., Lindberg, D. V., Smith, R. A., & Reluga, T. C. (2017). Provisioning of public health can be designed to anticipate public policy responses. *Bulletin of Mathematical Biology*, 79(1), 163–190.
- [34] Lindström, E., Madsen, H., & Nielsen, J. N. (2018). *Statistics for finance: Texts in statistical science*. Boca Raton: Chapman and Hall/CRC.
- [35] Ljungqvist, L., & Sargent, T. J. (2012). *Recursive macroeconomic theory*. Cambridge, Massachusetts: MIT Press.
- [36] Luke, D. A. (2015). *A user as guide to network analysis in R* (Vol. 72). Springer Switzerland: Springer.
- [37] Marcat, A., & Marimon, R. (2019). Recursive contracts. *Econometrica*, 87(5), 1589–1631.
- [38] Morzfeld, M. (2015). Implicit sampling for path integral control, Monte Carlo localization, and slam. *Journal of Dynamic Systems, Measurement, and Control*, 137(5), 051016.
- [39] Øksendal, B. (2003). Stochastic differential equations. In: *Stochastic Differential Equations* (pp. 65–84). Berlin: Springer.
- [40] Piguillem, F., & Shi, L. (2020). *The optimal covid-19 quarantine and testing policies*. 2020. Oxford: Einaudi Institute for Economics and Finance (EIEF).
- [41] Polansky, A. M., & Pramanik, P. (2021). A motif building process for simulating random networks. *Computational Statistics & Data Analysis*, 162, 107263.
- [42] Pramanik, P. (2016). *Tail non-exchangeability*. DeKalb, Illinois: Northern Illinois University.
- [43] Pramanik, P. (2020). Optimization of market stochastic dynamics. In: *Operations Research Forum* (Vol. 1, p. 1–17). Springer Switzerland: Springer.
- [44] Pramanik, P. (2021a). Effects of water currents on fish migration through a Feynman-type path integral approach under $\sqrt{8/3}$ Liouville-like quantum gravity surfaces. *Theory in Biosciences*, 140(2), 205–223.
- [45] Pramanik, P. (2021b). *Optimization of dynamic objective functions using path integrals*. (PhD thesis), DeKalb, Illinois: Northern Illinois University.
- [46] Pramanik, P. (2022a). *On lock-down control of a pandemic model*. arXiv: <http://arXiv.org/abs/arXiv:2206.04248>.
- [47] Pramanik, P. (2022b). *Stochastic control of a sir model with non-linear incidence rate through euclidean path integral*. arXiv: <http://arXiv.org/abs/arXiv:2209.13733>.
- [48] Pramanik, P. (2023a). Consensus as a Nash equilibrium of a stochastic differential game. *European Journal of Statistics*, 3, 10–10.
- [49] Pramanik, P. (2023b). *Path integral control in infectious disease modeling*. arXiv: <http://arXiv.org/abs/arXiv:2311.02113>.
- [50] Pramanik, P. (2023c). Path integral control of a stochastic multi-risk sir pandemic model. *Theory in Biosciences*, 142(2), 107–142.
- [51] Pramanik, P., & Polansky, A. M. (2020). *Motivation to run in one-day cricket*. arXiv: <http://arXiv.org/abs/arXiv:2001.11099>.

- [52] Pramanik, P., & Polansky, A. M. (2022). Optimal estimation of Brownian penalized regression coefficients. *International Journal of Mathematics, Statistics and Operations Research*, 2(2), 79–99.
- [53] Pramanik, P., & Polansky, A. M. (2023a). Scoring a goal optimally in a soccer game under Liouville-like quantum gravity action. In: *Operations Research Forum* (Vol. 4, p. 66). Springer Switzerland: Springer.
- [54] Pramanik, P., & Polansky, A. M. (2023b). Semicooperation under curved strategy spacetime. *The Journal of Mathematical Sociology*, 1–35.
- [55] Rao, F. (2014). Dynamics analysis of a stochastic sir epidemic model. In: *Abstract and Applied Analysis* (Vol. 2014). London: Hindawi.
- [56] Reluga, T. C. (2010). Game theory of social distancing in response to an epidemic. *PLoS Computational Biology*, 6(5), e1000793.
- [57] Reluga, T. C. (2013). Equilibria of an epidemic game with piecewise linear social distancing cost. *Bulletin of Mathematical Biology*, 75(10), 1961–1984.
- [58] Richardson, S., Hirsch, J. S., Narasimhan, M., Crawford, J. M., McGinn, T., Davidson, K. W., ..., Cookingham, J. (2020). Presenting characteristics, comorbidities, and outcomes among 5700 patients hospitalized with Covid-19 in the New York city area. *Jama*, 323(20), 2052–2059.
- [59] Simon, B. (1979). *Functional integration and quantum physics*, (Vol. 86). Providence, Rhode Island: Academic Press.
- [60] Theodorou, E., Buchli, J., & Schaal, S. (2010). *Reinforcement learning of motor skills in high dimensions: A path integral approach*. In: *2010 IEEE International Conference on Robotics and Automation (ICRA)* (pp. 2397–2403). IEEE.
- [61] Theodorou, E. A. (2011). *Iterative path integral stochastic optimal control: Theory and applications to motor control*. Los Angeles: University of Southern California.
- [62] Vikramdeo, K. S., Anand, S., Sudan, S. K., Pramanik, P., Singh, S., Godwin, A. K., ..., Dasgupta, S. (2023). Profiling mitochondrial DNA mutations in tumors and circulating extracellular vesicles of triple-negative breast cancer patients for potential biomarker development. *FASEB Bio Advances*, 5(10), 412.
- [63] Wilder-Smith, A., Chiew, C. J., & Lee, V. J. (2020). Can we contain the covid-19 outbreak with the same measures as for SARS? *The Lancet Infectious Diseases*, 20(5), e102–e107.
- [64] Yang, I., Morzfeld, M., Tomlin, C. J., & Chorin, A. J. (2014). Path integral formulation of stochastic optimal control with generalized costs. *IFAC Proceedings Volumes*, 47(3), 6994–7000.
- [65] Yang, J., Zheng, Y., Gou, X., Pu, K., Chen, Z., Guo, Q., ..., Zhou, Y. (2020). Prevalence of comorbidities and its effects in patients infected with SARS-COV-2: A systematic review and meta-analysis. *International Journal of Infectious Diseases*, 94, 91–95.
- [66] Yeung, D. W., & Petrosjan, L. A. (2006). *Cooperative stochastic differential games*. New York: Springer Science & Business Media.
- [67] Zhu, Y., Xie, J., Huang, F., & Cao, L. (2020). Association between short-term exposure to air pollution and covid-19 infection: Evidence from china. *Science of the Total Environment*, 727, 138704.

Appendix

Proof of Proposition 1. For small continuous time interval $[s, \tau]$, Itô formula yields

$$\begin{aligned} \mathcal{Z}(\tau, \mathbf{X}(\tau)) &= \mathcal{Z}(s, \mathbf{X}(s)) + \int_s^\tau \left\{ \frac{\partial}{\partial s} \mathcal{Z}(v, \mathbf{X}) + \boldsymbol{\mu}(v, u, \mathbf{X}) \frac{\partial}{\partial \mathbf{X}} \mathcal{Z}(v, \mathbf{X}) + \frac{1}{2} \text{trace} \left[\boldsymbol{\sigma}^T(v, \mathbf{X}) \left[\frac{\partial^2}{\partial X^T \partial X} \mathcal{Z}(v, \mathbf{X}) \right] \boldsymbol{\sigma}(v, \mathbf{X}) \right] \right\} dv \\ &\quad + \int_s^\tau \boldsymbol{\sigma}(s, \mathbf{X}) \frac{\partial}{\partial \mathbf{X}} \mathcal{Z}(s, \mathbf{X}) d\mathbf{B}(v). \end{aligned}$$

As we have already assumed that $\boldsymbol{\sigma}(s, \mathbf{X}) \frac{\partial}{\partial \mathbf{X}} \mathcal{Z}(s, \mathbf{X})$ is in Hilbert space L^2 , then in the aforementioned equation, integral part with respect to time vanishes [34]. Applying boundary condition $\mathcal{Z}(\tau, \mathbf{X}) = \Phi(\mathbf{X})$, the initial condition $\mathbf{X}(s) = \mathbf{X}_s$ and taking conditional expectation on the remainder part of the aforementioned equation yield

$$\mathbf{Z}(s, \mathbf{X}_s) = \mathbb{E}_s[\Phi(\mathbf{X}(\tau))].$$

This completes the proof. $\square$

Proof of Proposition 2. Suppose $\mathbf{X}$ and $\tilde{\mathbf{X}}$ both are strong solutions on the four-dimensional Brownian motion $\mathbf{B}(s)$ for all $s \in [0, t]$ under complete probability space $\{\mathcal{X}_\infty, \mathcal{F}_0, \mathcal{P}\}$. Define stopping times

$$s_\rho := \inf\{s \geq 0; \|\mathbf{X}(s)\| \geq \rho, \quad \forall \rho \geq 1\},$$

$$\tilde{s}_\rho := \inf\{s \geq 0; \|\tilde{\mathbf{X}}(s)\| \geq \rho, \quad \forall \rho \geq 1\}$$

Setting $S_\rho \triangleq s_\rho \wedge \tilde{s}_\rho$ yields $P[\lim_{\rho \rightarrow \infty} S_\rho] \xrightarrow{a.s.} \infty$ and

$$\mathbf{X}(s \wedge S_\rho) - \tilde{\mathbf{X}}(s \wedge S_\rho) = \int_0^{s \wedge S_\rho} \{\boldsymbol{\mu}[v, u(v), \mathbf{X}(v)] - \boldsymbol{\mu}[v, u(v), \tilde{\mathbf{X}}(v)]\} dv + \int_0^{s \wedge S_\rho} \{\boldsymbol{\sigma}[v, \mathbf{X}(v)] - \boldsymbol{\sigma}[v, \tilde{\mathbf{X}}(v)]\} d\mathbf{B}(v).$$

For any finite constant $\mathcal{K}$, the Hölder inequality for Lebesgue integrals, property 3.2.27 of [29] and Assumption 2 imply

$$\begin{aligned} \mathbb{E}_0\{\|\mathbf{X}(s \wedge S_\rho) - \tilde{\mathbf{X}}(s \wedge S_\rho)\|^2 | \mathcal{F}_0\} &\leq 9\mathbb{E}_0\left\{\left[\int_0^{s \wedge S_\rho} \|\boldsymbol{\mu}[v, u(v), \mathbf{X}(v)] - \boldsymbol{\mu}[v, u(v), \tilde{\mathbf{X}}(v)]\| dv\right]^2 \middle| \mathcal{F}_0\right\} \\ &\quad + 9\mathbb{E}_0\left\{\left[\sum_{k=1}^d \left[\sum_{l=1}^r \int_0^{s \wedge S_\rho} [\sigma_{kl}[v, \mathbf{X}(v)] - \sigma_{kl}[v, \tilde{\mathbf{X}}(v)]] d\mathbf{B}^{(l)}(v)\right]^2 \right] \middle| \mathcal{F}_0\right\} \\ &\leq 9s\mathbb{E}_0\left\{\int_0^{s \wedge S_\rho} \|\boldsymbol{\mu}[v, u(v), \mathbf{X}(v)] - \boldsymbol{\mu}[v, u(v), \tilde{\mathbf{X}}(v)]\|^2 dv \middle| \mathcal{F}_0\right\} \\ &\quad + 9\mathbb{E}_0\left\{\int_0^{s \wedge S_\rho} \|\boldsymbol{\sigma}[v, \mathbf{X}(v)] - \boldsymbol{\sigma}[v, \tilde{\mathbf{X}}(v)]\|^2 dv \middle| \mathcal{F}_0\right\} \\ &\leq 9(1+t)\mathcal{K}^2 \int_0^t \mathbb{E}_0\{\|\mathbf{X}(s \wedge S_\rho) - \tilde{\mathbf{X}}(s \wedge S_\rho)\|^2 dv | \mathcal{F}_0\}. \end{aligned}$$

Following [29], we know for $s \in [0, t]$, the aforementioned condition implies

$$\mathbb{E}_0\{\|\mathbf{X}(s \wedge S_\rho) - \tilde{\mathbf{X}}(s \wedge S_\rho)\|^2 | \mathcal{F}_0\} \leq 9t\mathcal{K}^2 + 9\mathcal{K}^2 \int_0^t \mathbb{E}_0\{\|\mathbf{X}(s \wedge S_\rho) - \tilde{\mathbf{X}}(s \wedge S_\rho)\|^2 dv | \mathcal{F}_0\}.$$

Therefore, $\{\mathbf{X}(s \wedge S_\rho); s \in [0, t]\}$ and $\{\tilde{\mathbf{X}}(s \wedge S_\rho); s \in [0, t]\}$ are the modification of each other and hence are indistinguishable. Allowing $\rho \rightarrow \infty$ gives us $\{\mathbf{X}(s); s \in [0, t]\}$ and $\{\tilde{\mathbf{X}}(s); s \in [0, t]\}$ are indistinguishable. $\square$

Proof of Proposition 3. For each optimal solution $\mathbf{X}^* \in \mathbb{F}^2$ of equation (4), define a squared integrable progressively measurable process $Z(\mathbf{X}^*)$ by:

$$Z(\mathbf{X}^*)_s = \mathbf{X}(0) + \int_0^s \boldsymbol{\mu}(s, u, \mathbf{X}) ds + \int_0^s \boldsymbol{\sigma}(s, \mathbf{X}) d\mathbf{B}(s). \quad (\text{A1})$$

We will show that $Z(\mathbf{X}^*) \in \mathbb{F}^2$. Furthermore, as $\mathbf{X}^*$ is a solution of equation (4) iff $Z(\mathbf{X}^*) = \mathbf{X}^*$, we will show that Z is the strict contraction of the Hilbert space $\mathbb{F}^2$. Using the fact that

$$|\boldsymbol{\mu}(s, u, \mathbf{X})|^2 \leq c_0[1 + |\mathbf{X}|^2 + |\boldsymbol{\mu}(s, u, \mathbf{X}(0))|^2]$$

yields

$$\|Z(\mathbf{X})\|^2 \leq 4 \left[t\mathbb{E}[|\mathbf{X}(0)|^2] + \mathbb{E} \left[\int_0^t \left| \int_0^s \boldsymbol{\mu}(s', u, \mathbf{X}) ds' \right|^2 ds \right] + t\mathbb{E} \left[\sup_{0 \leq s \leq t} \left| \int_0^s \boldsymbol{\sigma}(s', \mathbf{X}(s')) d\mathbf{B}(s') \right|^2 ds \right] \right]. \quad (\text{A2})$$

Assumption 3 implies $t\mathbb{E}[|\mathbf{X}(0)|^2] < \infty$. It will be shown that the second and third terms of the right-hand side of Inequality (6) are also finite. Assumption 2 implies,

$$\begin{aligned} \mathbb{E} \left[\int_0^t \left| \int_0^s \boldsymbol{\mu}(s', u, \mathbf{X}) ds' \right|^2 ds \right] &\leq \mathbb{E} \left[\int_0^t \int_0^s |\boldsymbol{\mu}(s', u, \mathbf{X})|^2 ds' ds \right] \\ &\leq c_0 \mathbb{E} \left[\int_0^t \int_0^s (1 + |\boldsymbol{\mu}(s', u, \mathbf{X}(0))|^2 + |\mathbf{X}(s)|^2) ds' ds \right] \\ &\leq c_0 t^2 \{1 + \|\boldsymbol{\mu}(s', u, \mathbf{X}(0))\|^2 + \mathbb{E}[\sup_{0 \leq s \leq t} |\mathbf{X}(s)|^2]\} < \infty. \end{aligned}$$

Doob's maximal inequality and Lipschitz assumption (i.e., Assumption 2) implies,

$$\begin{aligned} t\mathbb{E} \left[\sup_{0 \leq s \leq t} \left| \int_0^s \boldsymbol{\sigma}(s', \mathbf{X}(s')) d\mathbf{B}(s') \right|^2 ds \right] &\leq 4t\mathbb{E} \left[\int_0^t |\boldsymbol{\sigma}(s, \mathbf{X}(s))|^2 ds \right] \\ &\leq 4c_0 \mathbb{E} \left[\int_0^t (1 + |\boldsymbol{\sigma}(\mathbf{X}(0))|^2 + |\mathbf{X}(s)|^2) ds \right] \\ &\leq 4c_0 t^2 \{1 + \|\boldsymbol{\sigma}(\mathbf{X}(0))\|^2 + \mathbb{E}[\sup_{0 \leq s \leq t} |\mathbf{X}(s)|^2]\} < \infty. \end{aligned}$$

As Z maps $\mathbb{F}^2$ into itself, we show that it is strict contraction. To do so, we change Hilbert norm $\mathbb{F}^2$ to an equivalent norm. Following [8] for $a > 0$, define a norm on $\mathbb{F}^2$ by:

$$\|\xi\|_a^2 = \mathbb{E} \left[\int_0^t \exp(-as) |\xi_s|^2 ds \right].$$

If $\mathbf{X}(s)$ and $\mathbf{Y}(s)$ are the generic elements of $\mathbb{F}^2$, where $\mathbf{X}(0) = \mathbf{Y}(0)$, then

$$\begin{aligned}
\mathbb{E}[|Z(\mathbf{X}(s)) - Z(\mathbf{Y}(s))|^2] &\leq 2\mathbb{E}\left[\left|\int_0^\tau [\boldsymbol{\mu}(s', u, \mathbf{X}(s')) - \boldsymbol{\mu}(s', u, \mathbf{Y}(s'))]ds\right|^2\right] + 2\mathbb{E}\left[\left|\int_0^\tau [\boldsymbol{\sigma}_0^k(\mathbf{X}(s')) - \boldsymbol{\sigma}(s', \mathbf{Y}(s'))]d\mathbf{B}(s')\right|^2\right] \\
&\leq 2\tau\mathbb{E}\left[\int_0^\tau |\boldsymbol{\mu}(s', u, \mathbf{X}(s')) - \boldsymbol{\mu}(s', u, \mathbf{Y}(s'))|^2 ds'\right] + 2\mathbb{E}\left[\int_0^\tau |\boldsymbol{\sigma}(s', \mathbf{X}(s')) - \boldsymbol{\sigma}(s', \mathbf{Y}(s'))|^2 ds'\right] \\
&\leq c_0(1 + \tau)\int_0^\tau \mathbb{E}[|\mathbf{X}(s') - \mathbf{Y}(s')|^2]ds',
\end{aligned}$$

by Lipschitz's properties of drift and diffusion coefficients. Hence,

$$\begin{aligned}
\|Z(\mathbf{X}) - Z(\mathbf{Y})\|_a^2 &= \int_0^t \exp(-as)\mathbb{E}[|Z(\mathbf{X}(s)) - Z(\mathbf{Y}(s))|^2]ds \\
&\leq c_0t \int_0^t \exp(-as)\int_0^s \mathbb{E}[|\mathbf{X}(s') - \mathbf{Y}(s')|^2]ds'ds \\
&\leq c_0t \int_0^t \exp(-as)ds \int_0^s \mathbb{E}[|\mathbf{X}(s') - \mathbf{Y}(s')|^2]ds' \leq \frac{c_0t}{a}\|\mathbf{X} - \mathbf{Y}\|_a^2.
\end{aligned}$$

Furthermore, if c_0t is very large, Z becomes a strict contraction. Finally, for $s \in [0, t]$,

$$\begin{aligned}
\mathbb{E}[\sup_{0 \leq s \leq t} |\mathbf{X}(s)|^2] &= \mathbb{E}\left[\sup_{0 \leq s \leq t} \left|\mathbf{X}(0) + \int_0^{s'} \boldsymbol{\mu}(r, u, \mathbf{X}(r))dr + \int_0^{s'} \boldsymbol{\sigma}(r, \mathbf{X}(r))d\mathbf{B}(r)\right|^2\right] \\
&\leq 4\left[\mathbb{E}[|\mathbf{X}(0)|^2] + s\mathbb{E}\left[\int_0^s |\boldsymbol{\mu}(s', u, \mathbf{X}(s'))|^2 ds'\right] + 4\mathbb{E}\left[\int_0^s |\boldsymbol{\sigma}(s', \mathbf{X}(s'))|^2 ds'\right]\right] \\
&\leq c_0\left[1 + \mathbb{E}[|\mathbf{X}(0)|^2] + \int_0^s \mathbb{E}[\sup_{0 \leq r \leq s'} |\mathbf{X}(r)|^2]dr\right],
\end{aligned}$$

where the constant c_0 depends on t , $\|\boldsymbol{\mu}\|^2$, and $\|\boldsymbol{\sigma}\|^2$. Gronwall's inequality implies,

$$\mathbb{E}[\sup_{0 \leq s \leq t} |\mathbf{X}(s)|^2] \leq c_0\{1 + \mathbb{E}[|\mathbf{X}(0)|^2]\}\exp(c_0t). \square$$

Proof of Proposition 4. In order to show Condition 8, we will use the Hahn-Jordan orthogonal decomposition of total variation [13]:

$$\mathcal{P} = \mathcal{P}_1 - \mathcal{P}_2 = \mathcal{P}^+ - \mathcal{P}^-,$$

such that $\|\mathcal{P}_1 - \mathcal{P}_2\|_{tv} = \mathcal{P}^+(H) = \mathcal{P}^-(H)$. Therefore, for quantum Lagrangian $\mathcal{L} \in H$, we have

$$\begin{aligned}
|\mathcal{P}_1(\mathcal{L}) - \mathcal{P}_2(\mathcal{L})| &= \left|\int_{\mathbb{R}^4} \mathcal{L}(s, u, \mathbf{X})\mathcal{P}^+(d\mathbf{X}) - \int_{\mathbb{R}^4} \mathcal{L}(s, u, \tilde{\mathbf{X}})\mathcal{P}^-(d\tilde{\mathbf{X}})\right| \\
&= \|\mathcal{P}_1 - \mathcal{P}_2\|_{tv} \left|\int_{\mathbb{R}^4} [\mathcal{L}(s, u, \mathbf{X}) - \mathcal{L}(s, u, \tilde{\mathbf{X}})] \frac{\mathcal{P}^+(d\mathbf{X})}{\mathcal{P}^+(H)} \frac{\mathcal{P}^-(d\tilde{\mathbf{X}})}{\mathcal{P}^-(H)}\right|.
\end{aligned}$$

The aforementioned condition implies

$$|\mathcal{P}_1(\mathcal{L}) - \mathcal{P}_2(\mathcal{L})| \leq \|\mathcal{P}_1 - \mathcal{P}_2\|_{tv}.$$

Supremum over all $\mathcal{L} \in H$ yields

$$\sup\{|\mathcal{P}_1(\mathcal{L}) - \mathcal{P}_2(\mathcal{L})|; \mathcal{L} \in H\} \leq \|\mathcal{P}_1 - \mathcal{P}_2\|_{tv}.$$

The reverse inequality can be checked by using the simple function $\mathbb{1}_B$ such as $B \in \mathcal{E} \in H$. Now, we will show Condition 9. By the construction of this pandemic framework, there exist two non-interacting neighborhoods of agents H_+ and H_- so that $\mathcal{P}^+(H_-) = 0 = \mathcal{P}^-(H_+)$. Hence, for all $B \in \mathcal{E}$, we have

$$\mathcal{P}^+(B) = \mathcal{P}(B \cap H_+) \geq 0 \quad \text{and} \quad \mathcal{P}^-(B) = -\mathcal{P}^-(B \cap H_-) \geq 0,$$

which implies

$$\mathcal{P}_1(B \cap H_+) \geq \mathcal{P}_2(B \cap H_+) \quad \text{and} \quad \mathcal{P}_2(B \cap H_-) \geq \mathcal{P}_1(B \cap H_-). \quad (\text{A3})$$

For any $B \in \mathcal{E}$, define η as $\eta \triangleq \mathcal{P}_1(B \cap H_-) + \mathcal{P}_2(B \cap H_+)$. By construction of this pandemic network, we have

$$\eta(B) \leq \mathcal{P}_1(B) \wedge \mathcal{P}_2(B) \quad \text{and} \quad \eta(H) = \mathcal{P}_1(H_-) + \mathcal{P}_2(H_+). \quad (\text{A4})$$

As total variation distance between two immunity group is

$$\|\mathcal{P}_1 - \mathcal{P}_2\|_{tv} = \mathcal{P}^+(H) = \mathcal{P}(H_+) = \mathcal{P}_1(H_+) - \mathcal{P}_2(H_-) = 1 - [\mathcal{P}_1(H_+) + \mathcal{P}_2(H_-)],$$

Condition A4 implies

$$1 - \sup_{\gamma \leq \mathcal{P}_1, \mathcal{P}_2} \gamma(H) \leq 1 - \eta(H) = \|\mathcal{P}_1 - \mathcal{P}_2\|_{tv}.$$

In order to show the reverse inequality, assume that γ is a non-negative such as for all $B \in \mathcal{E}$ yields $\gamma(B) \leq \mathcal{P}_1(B) \wedge \mathcal{P}_2(B)$. Suppose, if we consider $B = H_+$ and $B = H_-$, respectively, then we have

$$\gamma(H_+) \leq \mathcal{P}_1(H_+) \quad \text{and} \quad \gamma(H_-) \leq \mathcal{P}_2(H_-),$$

which yields

$$\gamma(H) \leq \mathcal{P}_1(H_+) + \mathcal{P}_2(H_-) = 1 - \|\mathcal{P}_1 - \mathcal{P}_2\|_{tv}.$$

Therefore, $1 - \gamma(H) \geq \|\mathcal{P}_1 - \mathcal{P}_2\|_{tv}$. Finally, taking the infimum of over all the distributions of $\gamma \leq \mathcal{P}_1$ and $\mathcal{P}_2$, Condition 9 is obtained. To show Condition 10, we are going to use the similar idea like above. First using A3, define $\mathcal{P}_2(H_+) = \mathcal{P}_1(H_+) \wedge \mathcal{P}_2(H_+)$ and $\mathcal{P}_1(H_-) = \mathcal{P}_1(H_-) \wedge \mathcal{P}_2(H_-)$. This yields

$$\eta(H) = \mathcal{P}_1(H_-) + \mathcal{P}_2(H_+) = [\mathcal{P}_1(H_-) \wedge \mathcal{P}_2(H_-)] + [\mathcal{P}_1(H_+) \wedge \mathcal{P}_2(H_+)].$$

Non-interaction of agents between H_+ and H_- implies

$$\eta(H) \leq \inf \sum_{k=1}^K [\mathcal{P}_1(B_k) \wedge \mathcal{P}_2(B_k)],$$

where the infimum is taken over all resolutions of H into pairs of non-interacting subgroups B_k , $k \in [1, K]$, $K \geq 1$. To show the reverse inequality, we use the definition of η [13]. Using Condition A4, we know for any finite resolution $B_k \in \mathcal{E}$, the inequality $\eta(B_k) \leq \mathcal{P}_1(B_k) \wedge \mathcal{P}_2(B_k)$ holds. Thus,

$$\eta(H) = \sum_{k=1}^K \eta(B_k) \leq \sum_{k=1}^K \mathcal{P}_1(B_k) \wedge \mathcal{P}_2(B_k).$$

Taking the infimum of all resolutions and using $\eta(H) = 1 - \|\mathcal{P}_1 - \mathcal{P}_2\|_{tv}$ yield Condition 10. This completes the proof. $\square$

Proof of Theorem 1. From quantum Lagrangian function expressed in equation (7), the Euclidean action function for the agent in continuous time $[0, t]$ is given by:

$$\mathcal{A}_{0,t}(\mathbf{X}) = \int_0^t \mathbb{E}_s \{c[u(s), \mathbf{X}(s)] ds + \lambda[\boldsymbol{\mu}(s, u, \mathbf{X}) ds + \boldsymbol{\sigma}(s, \mathbf{X}) d\mathbf{B}(s)]\},$$

where vector $\lambda > 0$ is a time-independent quantum Lagrangian multiplier. As at the beginning of the continuous time interval $[s, s + \varepsilon]$, as the agent does not have any prior future knowledge, they make expectations based on their all current state variables represented by $\mathbf{X}$. Hence, $\mathbb{E}_s[\cdot] = \mathbb{E}[\cdot | \mathcal{F}_s]$, where $\mathcal{F}_s$ is the filtration process starting at time s . For a penalization constant $L_\varepsilon > 0$ and for time interval $[s, s + \varepsilon]$ with $\varepsilon \downarrow 0$, define a transition function from s to $s + \varepsilon$ as:

$$\Psi_{s,s+\varepsilon}(\mathbf{X}) = \frac{1}{L_\varepsilon} \int_{\mathbb{R}^4} \exp[-\varepsilon \mathcal{A}_{s,s+\varepsilon}(\mathbf{X})] \Psi_s(\mathbf{X}) d\mathbf{X}, \quad (\text{A5})$$

where $\Psi_s(\mathbf{X})$ is the value of the transition function at time s with the initial condition $\Psi_0(\mathbf{X}) = \Psi_0$ and the action function in $[s, s + \varepsilon]$ of the representative agent is,

$$\mathcal{A}_{s,s+\varepsilon}(\mathbf{X}) = \int_s^{s+\varepsilon} \mathbb{E}_v \{ c[u(v), \mathbf{X}(v)] dv + g[v + \Delta v, \mathbf{X}(v) + \Delta \mathbf{X}(v)] \},$$

where $g(\mathbf{X}) \in C^2([0, t] \times \mathbb{R}^4)$ such that Assumptions 1–3 hold and $\tilde{Y}(v) = g(\mathbf{X})$, where $\tilde{Y}$ is an Itô process [39], and

$$g(\mathbf{X}) = \lambda [\mu(s, u, \mathbf{X}) ds + \sigma(s, \mathbf{X}) d\mathbf{B}(s)] + o(1),$$

where $\Delta \mathbf{X}(v) = \mathbf{X}(v + \varepsilon) - \mathbf{X}(v)$. In equation (A5), L_ε is a positive penalization constant such that the value of $\Psi_{s,s+\varepsilon}^k(\cdot)$ becomes 1. One can think this transition function $\Psi_{s,s+\varepsilon}(\cdot)$ as some transition probability function on the Euclidean space. We have divided the time interval $[0, t]$ into n small equal sub-intervals $[s, s + \varepsilon]$ so that $\tau = s + \varepsilon$. Fubini's theorem implies,

$$\mathcal{A}_{s,\tau}(\mathbf{X}) = \mathbb{E}_s \left[\int_s^\tau c[u(v), \mathbf{X}(v)] dv + g[v + \Delta v, \mathbf{X}(v) + \Delta \mathbf{X}(v)] \right].$$

After using the fact that $[\Delta \mathbf{X}(s)]^2 = \varepsilon$, for $\varepsilon \downarrow 0$ (with initial condition $\mathbf{X}(0)$), Itô's formula and [6] imply,

$$\mathcal{A}_{s,\tau}(\mathbf{X}) = c[u(s), \mathbf{X}(s)] + g + \frac{\partial g}{\partial s} + \mu[s, u(s), \mathbf{X}(s)] \frac{\partial g}{\partial \mathbf{X}} + \frac{1}{2} \sigma^T[s, \mathbf{X}(s)] \frac{\partial^2 g}{\partial \mathbf{X}^T \partial \mathbf{X}} \sigma[s, \mathbf{X}(s)] + o(1),$$

where $g = g[s, \mathbf{X}(s)]$. Result in equation (A5) implies

$$\begin{aligned} \Psi_{s,\tau}(\mathbf{X}) &= \frac{1}{L_\varepsilon} \int_{\mathbb{R}^4} \exp \left[-\varepsilon \left[c[u(s), \mathbf{X}(s)] + g + \frac{\partial g}{\partial s} + \mu[s, u(s), \mathbf{X}(s)] \frac{\partial g}{\partial \mathbf{X}} + \frac{1}{2} \sigma^T[s, \mathbf{X}(s)] \frac{\partial^2 g}{\partial \mathbf{X}^T \partial \mathbf{X}} \sigma[s, \mathbf{X}(s)] \right] \right] \Psi_s(\mathbf{X}) d\mathbf{X} \\ &\quad + o(\varepsilon^{1/2}). \end{aligned}$$

For $\varepsilon \downarrow 0$, define a new transition probability Ψ_s^τ centered around time τ . A Taylor series expansion (up to second order) of the left-hand side of the aforementioned equation yields

$$\begin{aligned} \Psi_s^\tau(\mathbf{X}) + \varepsilon \frac{\partial}{\partial s} \Psi_s^\tau + o(\varepsilon) &= \frac{1}{L_\varepsilon} \int_{\mathbb{R}^4} \exp \left\{ -\varepsilon \left[c[u(s), \mathbf{X}(s)] + g + \frac{\partial g}{\partial s} + \mu[s, u(s), \mathbf{X}(s)] \frac{\partial g}{\partial \mathbf{X}} \right. \right. \\ &\quad \left. \left. + \frac{1}{2} \sigma^T[s, \mathbf{X}(s)] \frac{\partial^2 g}{\partial \mathbf{X}^T \partial \mathbf{X}} \sigma[s, \mathbf{X}(s)] \right] \right\} \Psi_s(\mathbf{X}) d\mathbf{X} + o(\varepsilon^{1/2}), \end{aligned}$$

as $\varepsilon \downarrow 0$. For fixed s and τ , let $\mathbf{X}(s) = \mathbf{X}(\tau) + \vartheta$. For some number $\bar{\vartheta}^* \in (0, \infty)$, assume $|\vartheta| \leq \bar{\vartheta}^* \varepsilon [\mathbf{X}(s)]^{-1}$. Therefore, we obtain upper bound of state variables in this SIR model as $\mathbf{X}(s) \leq \bar{\vartheta}^* \varepsilon / (\vartheta)^2$. Moreover, Fröhlich's reconstruction theorem [43,45,59] and Assumptions 1–3 imply

$$\begin{aligned} \Psi_s^\tau(\mathbf{X}) + \varepsilon \frac{\partial}{\partial s} \Psi_s^\tau + o(\varepsilon) &= \frac{1}{L_\varepsilon} \int_{\mathbb{R}^4} \exp \left\{ -\varepsilon \left[c[u(s), \mathbf{X}(s)] + g + \frac{\partial g}{\partial s} + \mu[s, u(s), \mathbf{X}(s)] \frac{\partial g}{\partial \mathbf{X}} \right. \right. \\ &\quad \left. \left. + \frac{1}{2} \sigma^T[s, \mathbf{X}(s)] \frac{\partial^2 g}{\partial \mathbf{X}^T \partial \mathbf{X}} \sigma[s, \mathbf{X}(s)] \right] \right\} \left[\Psi_s^\tau(\mathbf{X}) + \vartheta \frac{\partial}{\partial \mathbf{X}} \Psi_s^\tau(\mathbf{X}) + o(\varepsilon) \right] d\mathbf{X} + o(\varepsilon^{1/2}), \end{aligned} \quad (\text{A6})$$

as $\varepsilon \downarrow 0$. Define a C^2 function:

$$\tilde{f}(s, \bar{Z}) \triangleq c[u(s), \mathbf{X}(s)] + g + \frac{\partial g}{\partial s} + \mu[s, u(s), \mathbf{X}(s)] \frac{\partial g}{\partial \mathbf{X}} + \frac{1}{2} \sigma^T[s, \mathbf{X}(s)] \frac{\partial^2 g}{\partial \mathbf{X}^T \partial \mathbf{X}} \sigma[s, \mathbf{X}(s)].$$

Plugging in $\tilde{f}(s, \bar{Z})$ into equation (A6) yields

$$\Psi_s^\tau(\mathbf{X}) + \varepsilon \frac{\partial}{\partial s} \Psi_s^\tau(\mathbf{X}) + o(\varepsilon) = \frac{1}{L_\varepsilon} \int_{\mathbb{R}^4} \exp\{-\varepsilon \tilde{f}(s, \bar{Z})\} \left[\Psi_s^\tau(\mathbf{X}) + \vartheta \frac{\partial}{\partial \mathbf{X}} \Psi_s^\tau(\mathbf{X}) + o(\varepsilon) \right] d\mathbf{X} + o(\varepsilon^{1/2}). \quad (\text{A7})$$

Let $\tilde{f}(s, \bar{Z})$ be a C^2 function. A second-order Taylor series expansion yields

$$\tilde{f}(s, u, \vartheta(\tau)) = \tilde{f}(s, u, \vartheta(\tau)) + [\vartheta - \mathbf{X}(\tau)] \frac{\partial}{\partial \mathbf{X}} \tilde{f}(s, u, \vartheta(\tau)) + \frac{1}{2} [\vartheta - \mathbf{X}(\tau)]^T \frac{\partial^2}{\partial \mathbf{X}^T \partial \mathbf{X}} \tilde{f}(s, u, \vartheta(\tau)) [\vartheta - \mathbf{X}(\tau)] + o(\varepsilon),$$

as $\varepsilon \downarrow 0$ and $\Delta u(s) \downarrow 0$. Define $\hat{\vartheta} = \vartheta - \mathbf{X}$ so that $d\hat{\vartheta} = d\vartheta$. Thus, first integration of equation (A7) becomes

$$\begin{aligned} \int_{\mathbb{R}^4} \exp(-\varepsilon \tilde{f}(s, \bar{Z})) d\mathbf{X} &= \int_{\mathbb{R}^4} \exp\{-\varepsilon [\tilde{f}(s, u, \vartheta(\tau)) - \mathbf{J}^T \hat{\vartheta} + \hat{\vartheta}^T \mathcal{H}_{\mathbf{X}} \hat{\vartheta}]\} d\hat{\vartheta} \\ &= \exp\{-\varepsilon \tilde{f}(s, u, \vartheta(\tau))\} \int_{\mathbb{R}^4} \exp\{(\varepsilon \mathbf{J}^T) \hat{\vartheta} - \hat{\vartheta}^T (\varepsilon \mathcal{H}_{\mathbf{X}}) \hat{\vartheta}\} d\hat{\vartheta} \\ &= \frac{\pi}{\sqrt{\varepsilon |\mathcal{H}_{\mathbf{X}}|}} \exp\left\{ \frac{\varepsilon}{4} \mathbf{J}^T \mathcal{H}_{\mathbf{X}}^{-1} \mathbf{J} - \varepsilon \tilde{f}(s, u, \vartheta(\tau)) \right\}, \end{aligned}$$

where $\mathbf{J} = -\partial \tilde{f} / \partial \mathbf{X}$ and $\mathcal{H}_{\mathbf{X}}$ is a non-singular Hessian matrix. Therefore, first integral term of equation (A7) becomes

$$\frac{1}{L_\varepsilon} \Psi_s^\tau(\mathbf{X}) \int_{\mathbb{R}^4} \exp(-\varepsilon \tilde{f}) d\mathbf{X} = \frac{1}{L_\varepsilon} \Psi_s^\tau \frac{\pi}{\sqrt{\varepsilon |\mathcal{H}_{\mathbf{X}}|}} \exp\left\{ \frac{\varepsilon}{4} \mathbf{J}^T \mathcal{H}_{\mathbf{X}}^{-1} \mathbf{J} - \varepsilon \tilde{f}(s, u, \vartheta(\tau)) \right\},$$

where $\mathcal{H}_{\mathbf{X}} > 0$. In a similar fashion, we obtain the second integral term of equation (A7) as:

$$\frac{1}{L_\varepsilon} \frac{\partial \Psi_s^\tau(\mathbf{X})}{\partial \mathbf{X}} \int_{\mathbb{R}^4} \vartheta \exp(-\varepsilon \tilde{f}) d\mathbf{X} = \frac{1}{L_\varepsilon} \frac{\partial \Psi_s^\tau}{\partial \mathbf{X}} \frac{\pi}{\sqrt{\varepsilon |\mathcal{H}_{\mathbf{X}}|}} \left[\frac{1}{2} \mathcal{H}_{\mathbf{X}}^{-1} + \mathbf{X} \right] \exp\left\{ \frac{\varepsilon}{4} \mathbf{J}^T \mathcal{H}_{\mathbf{X}}^{-1} \mathbf{J} - \varepsilon \tilde{f}(s, u, \vartheta(\tau)) \right\}.$$

Using the aforementioned results and equation (A7), we obtain a Fokker-Plank-type equation as:

$$\Psi_s^\tau(\mathbf{X}) + \varepsilon \frac{\partial}{\partial s} \Psi_s^\tau(\mathbf{X}) + o(\varepsilon) = \frac{1}{L_\varepsilon} \frac{\pi}{\sqrt{\varepsilon |\mathcal{H}_{\mathbf{X}}|}} \exp\left\{ \frac{\varepsilon}{4} \mathbf{J}^T \mathcal{H}_{\mathbf{X}}^{-1} \mathbf{J} - \varepsilon \tilde{f}(s, u, \vartheta(\tau)) \right\} \left[\Psi_s^\tau(\mathbf{X}) + \left[\frac{1}{2} \mathcal{H}_{\mathbf{X}}^{-1} + \mathbf{X} \right] \frac{\partial \Psi_s^\tau}{\partial \mathbf{X}} \right] + o(\varepsilon^{1/2}),$$

as $\varepsilon \downarrow 0$. Assuming $L_\varepsilon = \pi / \sqrt{\varepsilon |\mathcal{H}_{\mathbf{X}}|} > 0$ yields

$$\Psi_s^\tau(\mathbf{X}) + \varepsilon \frac{\partial}{\partial s} \Psi_s^\tau(\mathbf{X}) + o(\varepsilon) = \left\{ 1 + \frac{\varepsilon}{4} \mathbf{J}^T \mathcal{H}_{\mathbf{X}}^{-1} \mathbf{J} - \varepsilon \tilde{f}(s, u, \vartheta(\tau)) \right\} \left[\Psi_s^\tau(\mathbf{X}) + \left[\frac{1}{2} \mathcal{H}_{\mathbf{X}}^{-1} + \mathbf{X} \right] \frac{\partial \Psi_s^\tau}{\partial \mathbf{X}} \right] + o(\varepsilon^{1/2}),$$

as $\varepsilon \downarrow 0$. Since $\mathbf{X} \leq \bar{\vartheta}^* \varepsilon / (\vartheta)^2$, assume $|\mathcal{H}_{\mathbf{X}}^{-1}| \leq 2\bar{\vartheta}^* \varepsilon (1 - \vartheta^{-1})$ such that $|(2\mathcal{H}_{\mathbf{X}})^{-1} + \mathbf{X}| \leq \bar{\vartheta}^* \varepsilon$. Therefore, $|\mathcal{H}_{\mathbf{X}}^{-1}| \leq 2\varepsilon \bar{\vartheta}^* (1 - \vartheta^{-1})$ so that $|(2\mathcal{H}_{\mathbf{X}})^{-1} + \mathbf{X}| \downarrow 0$. Hence,

$$\Psi_s^\tau(\mathbf{X}) + \varepsilon \frac{\partial}{\partial s} \Psi_s^\tau(\mathbf{X}) + o(\varepsilon) = (1 - \varepsilon) + \Psi_s^\tau + o(\varepsilon^{1/2}).$$

The Fokker-Plank-type equation of stochastic SIR model with infection dynamics is,

$$\frac{\partial}{\partial s} \Psi_s^\tau(\mathbf{X}) = -\tilde{f}(s, u, \vartheta(\tau)) \Psi_s^\tau(\mathbf{X}).$$

The solution of

$$-\frac{\partial}{\partial u} \tilde{f}(s, u, \vartheta(\tau)) \Psi_s^\tau(\mathbf{X}) = 0 \quad (\text{A8})$$

is an optimal “lock-down” intensity and vaccination rate. Since $\vartheta = \mathbf{X}(s) - \mathbf{X}(\tau)$ for all $\varepsilon \downarrow 0$, in equation (A8), ϑ can be replaced by $\mathbf{X}$. As the transition function $\Psi_s^\tau(\mathbf{X})$ is a solution of equation (A8), the result follows. $\square$