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Theme:

Stochastic Modelling and Optimal Control of Epidemic Models

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Dedication

To my beloved parents, whose constant support and sacrifices.

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Abstract

This work considers some mathematical methods for formulation and numerical simulation of stochastic epidemic models. Specifically, models are formulated by stochastic differential equations. Some well-known examples are used for illustration such as a SIR epidemic model.

An optimal control analysis of a dynamical system of optimal control is presented. The stochastic and deterministic control systems are studied. For the deterministic optimal control problem the Pontryagins Maximum Principle is used. The stochastic optimal control problem is performed by using Stochastic Maximum Principle. The comparative results are obtained numerically through simulation, with a specific application to influenza using the Susceptible-Infected-Recovered (SIR) model.

The results highlight the advantages of stochastic optimal control in mitigating Epidemics outbreaks, offering insights for policymakers on resource allocation and intervention strategies in the presence of uncertainty.

Key words: Epidemic models, Optimal control theory, Pontryagins Maximum Principle, Stochastic Maximum Principle, Numerical simulations, Stochastic Differential Equations.

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Introduction

In recent years, the world has witnessed a wide spread of infectious diseases, such as the COVID-19 pandemic, which had a negative impact on public health and global economy. These epidemics require intervention and for this reason we have to understand the dynamics of disease transmission and develop effective strategies to combat epidemics and reducing infection.
Mathematics modeling plays an important role in finding solutions through epidemic models, which are powerful tools for analyzing and understanding the spread of diseases. These models describe the dynamics of infections over time using Ordinary differential equations (ODEs) and stochastic differential equations (SDEs)[11][27]. They also help decision-makers to assess the impact of various interventions, such as vaccination, quarantine, and social distancing.
Epidemic models aim to produce exact epidemic progression forecasts which then support the development of efficient strategies for disease spread containment. Through the combination of mathematical frameworks with actual health data these models generate specific guidelines for global and local epidemic responses that reduce both human and economic damage. Stochastic modeling stands as one of the multiple epidemic modeling methods which provides a structure for integrating random elements to capture the unpredictable nature of real disease processes. Through the implementation of optimal control theory, experts can create successful epidemic management strategies which maintain a proper balance between health protection and economic considerations [1][50][12]. The optimal control problem for deterministic systems can be solved by using Pontryagin Maximum Principle, and for the stochastic optimal control problem is performed by using Stochastic Maximum Principle [29, 22].
This thesis is organized in four chapters: The first chapter (1) provides a foundational understanding of epidemic modeling and optimal control, essential for addressing the complex dynamics of infectious disease spread. It begins by introducing the basic epidemic models, including the Susceptible-Infectious-Recovered (SIR) model, which captures the core mechanisms of disease transmission and recovery within a population. These models serve as critical tools for predicting outbreaks and guiding public health interventions. To accurately simulate these

models, we explore numerical methods for solving ordinary differential equations (ODEs), with a focus on the Runge-Kutta (RK) method. The chapter then transitions to the deterministic optimal control framework, which seeks to minimize the spread and impact of infectious diseases through strategic interventions [12, 47, 50]. We introduce the Pontryagin Maximum Principle (PMP), which is a fundamental method for deriving optimal control strategies.

The second chapter (2) delves into the mathematical foundations of Forward-Backward Stochastic Differential Equations (FBSDEs), which play a critical role in modeling and controlling systems influenced by randomness. We first present the general structure of FBSDEs. To solve these complex equations, we discuss numerical methods such as the Forward-Backward Euler-Maruyama scheme and the Least Squares Regression based method, which provide practical approaches for approximating the solutions of FBSDEs. Building on this foundation, we introduce stochastic epidemic models, which extend traditional deterministic frameworks by incorporating random perturbations. These models provide a more realistic representation of disease spread, accounting for the unpredictable nature of infection. Then we will recall the strong formulation of the stochastic optimal control problem. A statement of the stochastic maximum principle is given in which the stochastic Hamiltonian system is introduced [22].

In chapter (3), we apply the theoretical framework of optimal control to influenza epidemic. We analyze the comparative performance of deterministic and stochastic optimal control approaches for epidemic management using the Susceptible-Infected-Recovered (SIR) model with media awareness, the inclusion treatment and vaccination as controls. We begin by formulating both deterministic and stochastic models to capture the spread dynamics of influenza and show the effect of media awareness program without controls on the spread of the disease. Then we formulate both deterministic and stochastic optimal control models and we focus on finding the successful intervention strategy which decreases the number of infections with a minimum cost.

Finally, in chapter (4) we perform a numerical analysis of optimal control to influenza epidemic, evaluating the impact of various control strategies, such as vaccination, and treatment, on the spread of the disease. We explore different scenarios to assess the effectiveness of these strategies under varying epidemiological and intervention conditions. For simulations we use Python programming language.

Chapter 1

Deterministic Epidemic Models and Optimal Control

1.1 Epidemics outbreaks

Infectious diseases are those that can be transmitted from one person to another person and can cause epidemic outbreaks for examples, Covid19, Influenza, Measles, HIV, Tuberculosis, Malaria, Yellow fever. These are caused by viral and bacterial agents. Even with advanced medical treatments and vaccinations in place, the high number of people affected becomes an issue. The infectious viruses are capable of constant adaptation which causes new diseases to surface and, most of the time, leads to epidemics. Mathematical models can be used to represent how an infection spreads across a population over time, and generally come in two forms: stochastic and deterministic models [11][14].

1.2 The basic epidemic models

Throughout this section, we introduce the foundational concepts of mathematical modeling in epidemiology, focusing on some of the most widely used epidemic models. The choice of the most appropriated model depends on the precision or generality required.

Epidemic models provide valuable insights into disease transmission and help predict outbreak dynamics. Historical advancements in this field began with John Graunt in 1662, who systematically analyzed mortality causes in London, laying the foundation for epidemiology's "theory of competing risks."

The first step is to represent the epidemiology of the disease being studied by dividing the pop-

ulation into subpopulations, called compartments, that represent the various stages of disease progression. For example, individuals are identified as 'susceptible' (S) to a disease if they don't currently have the disease nor any immunity to the disease, e.g., they have not been vaccinated. Individuals are 'exposed' (E) if they have been infected with the disease pathogen but are not able to infect others, and they are 'infectious' (I) if they are infected and infectious with the disease pathogen. Finally, they are 'removed' (R) if they have cleared the infection and have immunity to recurrence for at least some period of time [2, 9, 10, 28, 31]. the epidemiology of a disease is represented as a series of subpopulations connected by the flow from one compartment to another that is dictated by the disease dynamics. The rates of flow between compartments are estimated from experiments and data analysis [35, 45, 43].

Now we will consider that population is constant, neglecting the tourism an immigration factors. Also it is considered that the population is homogeneously mixed, which means that every individual interacts with another at the same level and therefore all individuals have the same risk of contracting the disease. The compartment changes are expressed by a system of differential equations [1].let us present a method for solving ODEs before introducing the basic epidemic models

Runge-Kutta method for ODE's

$Runge-Kutta$ method is based on the concept of using weighted averages of different slopes to estimate the value of the derivative at each step. The most commonly used form of the $Runge-Kutta$ method is the $fourth-order$ RungeKutta ($RK4$) method [13] which is given by this algo-

Algorithm 1: Fourth-Order Runge-Kutta (RK4) Method

Δt is the step size, t_n is the current time, x_n is the current solution, and f(t,x) is the ODE function. Set $N \leftarrow T/\Delta t$ (Number of steps) ;

for $n = 0$ *to* $N - 1$ **do**

Compute:

$$k_1 = \Delta t f(x_n, t_n)$$

$$k_2 = \Delta t f\left(x_n + \frac{\Delta t}{2} k_1, t_n + \frac{\Delta t}{2}\right)$$

$$k_3 = \Delta t f\left(x_n + \frac{\Delta t}{2} k_2, t_n + \frac{\Delta t}{2}\right)$$

$$k_4 = \Delta t f\left(x_n + \Delta t \cdot k_3, t_n + \Delta t\right)$$

Update solution:

$$x_{n+1} = x_n + \frac{1}{6}(k_1 + 2k_2 + 2k_3 + k_4)$$

return x_{n+1}

rithm.

The simple Kermack–McKendrick epidemic Model (SIR)

Description: The SIR model is suitable for diseases where recovered individuals acquire immunity. for example Measles can be modelled using the SIR framework, as individuals acquire long-term immunity after recovery[1][50].

The compartments are:

- **Susceptible(S):** Individuals who can contract the disease.
- **Infected (I):** Individuals who are infected and can spread the disease.
- **Recovered (R):** Individuals who have recovered and acquire immunity.
- β is the transmission rate and γ is the recovery rate.

The total population is constant: $N = S + I + R$. We consider the following equations

$$\begin{cases} \frac{dS}{dt} = -\beta SI, \\ \frac{dI}{dt} = \beta SI - \gamma I, \\ \frac{dR}{dt} = \gamma I \end{cases} \tag{1.1}$$

where: $S(0) > 0, I(0) > 0$, and $R(0) \geqslant 0$.

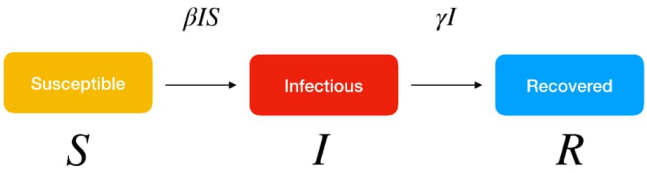


Figure 1.1: The SIR principal scheme

Example:

Consider an epidemic of influenza in a British boarding school in early 1978 . Three boys were reported to the school with the typical symptoms of influenza. Over the next few days, a very large fraction of the 763 boys in the school had contact with the infection. Within two weeks, the infection had become extinguished. The best parameters yield an estimated active infectious period $\gamma = \frac{1}{2.2}$, and a transmission rate $\beta = 1.66$ per day[1].

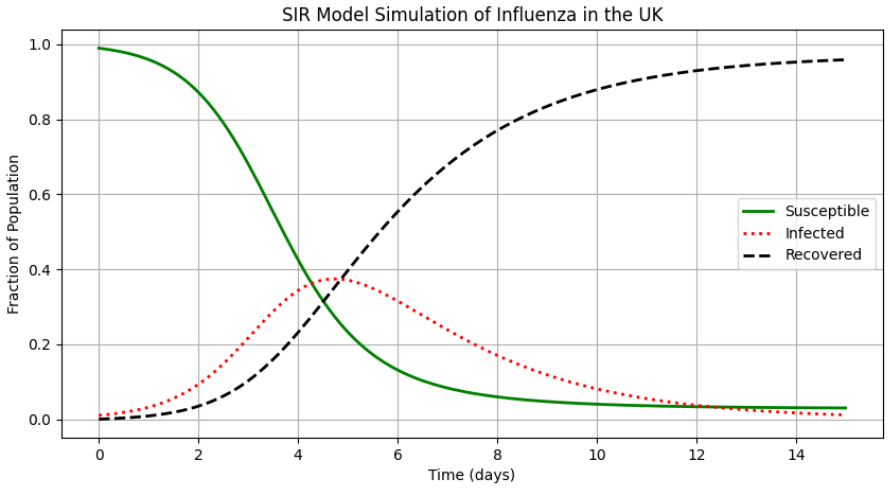


Figure 1.2: The time-evolution of in influenza over 15 days

From the graph of Figure 1.2 behavior we observe that the susceptible S decreases over time as individuals get infected and infected I increases initially due to new infections, peaks, and then decreases as individuals recover. Then recovered R increases as more infected individuals recover.

SI Model:

Description: The SI model is one of the simplest epidemic models used to describe the spread of infectious diseases. In this model, there is no recovery or removal process, meaning

once individuals become infected, they remain in the infected compartment indefinitely[1]. This model is suitable for diseases where there is no immunity or cure, such as certain viral infections (HIV). The disease spreads through interactions between susceptible and infected individuals, governed by the following differential equations:

$$\begin{cases} \frac{dS}{dt} = -\beta SI \\ \frac{dI}{dt} = \beta SI \end{cases}$$

where: $S(0) > 0, I(0) > 0$

The total population is constant: $N = S + I$.

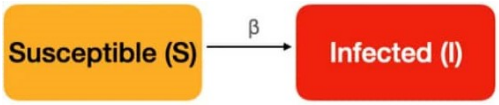


Figure 1.3: The SI principal scheme

Example:

We take a closer look at the course of the Corona virus pandemic in Croatia. We will consider the period from October 11, 2020 to February 8, 2021. We assume that $N = 235473$, with the parameter $\alpha = 0.00015$. [28]

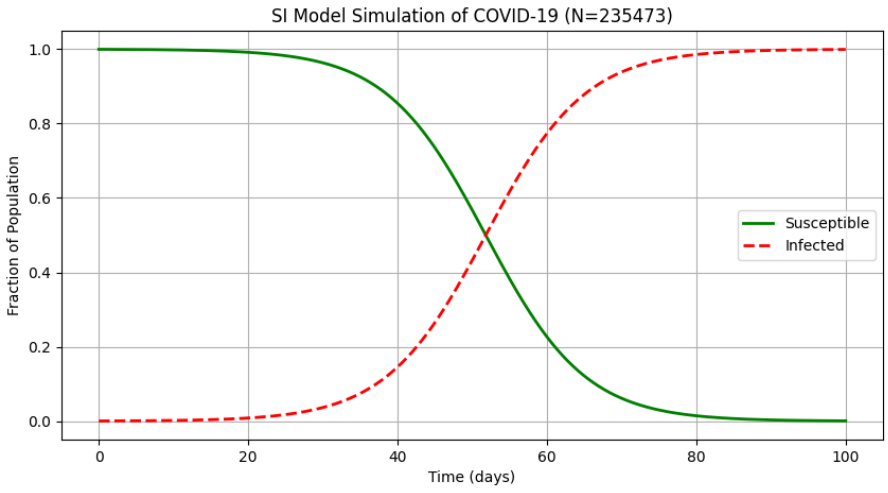


Figure 1.4: Simulation of coronavirus infection in Croatia

From the graph behavior we can observe that susceptible decreases as individuals get infected. Then the infected increases as the entire population becomes infected over time. So the infection

spreads until everyone is infected.

SIS Model:

Description: The SIS model describes diseases where infected individuals recover but do not gain immunity and can become susceptible again[1]. The common cold can be modeled using the SIS framework, as individuals can recover and become susceptible to reinfection. The total population is constant: $N = S + I$. So that lead us to the following system:

$$\begin{cases} \frac{dS}{dt} = -\beta SI + \gamma I \\ \frac{dI}{dt} = \beta SI - \gamma I \end{cases}$$

where:where:$S(0) > 0, I(0) > 0$

- β: Transmission rate.
- γ: Recovery rate.

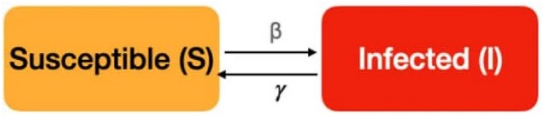


Figure 1.5: The SIS principal scheme

Each arrow pointing towards the inside of the compartment represents a positive term in the differential equation, and the opposite direction introduces a negative term.

Example:

Trachoma is an infectious disease causing a characteristic roughening of the inner surface of the eyelids. Also called granular conjunctivitis or Egyptian ophthalmia, it is the leading cause of infectious blindness in the world, with parameters $\beta = 0.047$ as transmission rate and the recovery rate $\gamma = 0.017$ [1].

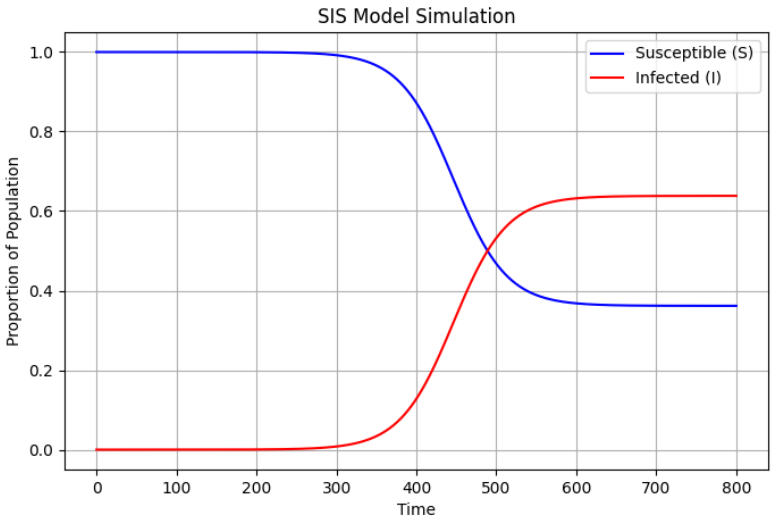


Figure 1.6: Deterministic SIS model for trachoma disease

From the graph behavior we observe that the susceptible decreases initially but stabilizes as individuals cycle back from the infected group. Then the infected increases and stabilizes as the infection rate and recovery rate balance. So the infection persists over time, reaching a steady state where a constant proportion of the population remains infected.

SEIR Model:

Description: The SEIR model extends the SIR model by adding an **E (Exposed)** compartment, representing individuals who have been exposed to the disease but are not yet infectious. For example COVID-19 can be modeled using the SEIR framework[1], as there is an incubation period where individuals are exposed but not yet infectious. The total population is constant: $N = S + E + I + R$. This is described by the following set of differential equations:

$$\begin{cases} \frac{dS}{dt} = -\beta SI, \\ \frac{dE}{dt} = \beta SI - \sigma E, \\ \frac{dI}{dt} = \sigma E - \gamma I, \\ \frac{dR}{dt} = \gamma I \end{cases}$$

where:$S(0) > 0$, $I(0) > 0$, $E(0) > 0$ and $R(0) \geq 0$

- β: Transmission rate.
- σ: Rate at which exposed individuals become infectious.

- γ: Recovery rate.

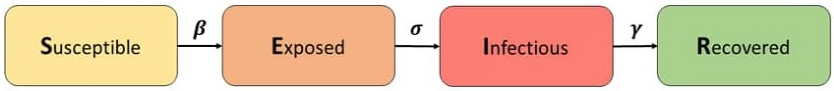


Figure 1.7: The SEIR principal scheme

Example:

Rubella, commonly known as German Measles, is a most common in child age, caused by the rubella virus. Children recover more quickly than adults, and can be very serious in pregnancy. The virus is contracted through the respiratory tract and has an incubation period of 2 to 3 weeks. The primary symptom of rubella virus infection is the appearance of a rash on the face which spreads to the trunk and limbs and usually fades after three days. Other symptoms include low grade fever, swollen glands, joint pains, headache and conjunctivitis. From [1], the parameters are $\beta = 0.52759$, $\sigma = 0.65$, $\gamma = 0.012$.

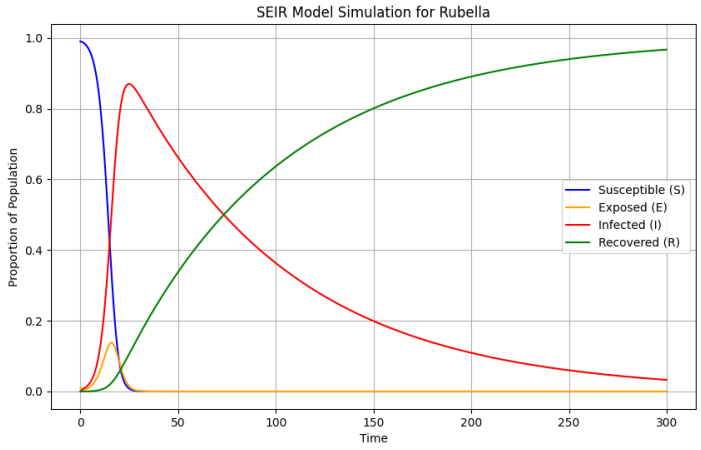


Figure 1.8: Deterministic SEIR model for Rubella disease

Adding an exposed (E) compartment for individuals in the incubation phase who are infected but not yet infectious, the susceptible decreases as individuals are exposed and infected. The exposed increases initially, then decreases as their individuals become infectious.

The basic reproduction number

In epidemiology there are many used threshold values, the most important is the basic reproduction number which is defined as fellows.

Definition 1.2.1 *: The basic reproduction number R_0 is a famous result due to Kermack and McKendrick, it is called also "threshold phenomenon" because initially proportion of susceptible in population must be exceed this critical for an infected to invade and it is defined as the average number of secondary infections that occurs when one infective is introduced into a completely susceptible population [6, 14, 15]. If $R_0 < 1$ the disease cannot invade the population and the infection will die out over a period of time, if $R_0 > 1$ the disease will invade the population.*

Example:

The classical SIR model has been utilized to determine, the basic reproduction number (R_0) of $COVID19$ in Algeria basing on the daily reported confirmed cases by the Algerian Ministry of Health from February 25th, 2020 to August 12th, 2020 [17]. The SIR model is as follows:

$$\begin{cases} \frac{dS}{dt} = -\frac{\beta}{N}SI, \\ \frac{dI}{dt} = \frac{\beta}{N}SI - \gamma I, \\ \frac{dR}{dt} = \gamma I \end{cases} \tag{1.2}$$

At $t = 0$ we have $S = N$, $I \approx 0$, and $R = 0$. The disease will die out over a period of time, if

$$\frac{\beta}{N}SI - \gamma I = \beta I - \gamma I < 0.$$

Then

$$R_0 = \frac{\beta}{\gamma} < 1.$$

The parameters of SIR epidemic model $\beta = 0.0561215$, $\gamma = 0.0455331$ are estimated by using least squares. After substituting these parameters, we find the basic reproduction number of $COVID19$ in Algeria

$$R_0 = \frac{\beta}{\gamma} = 1.23254$$

Since ($R_0 > 1$), then the disease will spread in the population.

Calculating R_0 using the next-generation matrix:

In complex epidemic models, R_0 can be computed using the *Next-Generation Matrix* (NGM) method. It is obtained by taking the largest (dominant) eigenvalue (spectral radius). This involves constructing two matrices [15][14]:

$$R_0 = \left[\frac{\partial F_i(x_0)}{\partial x_j}\right]\left[\frac{\partial V_i(x_0)}{\partial x_j}\right]^{-1} = \rho(FV^{-1})$$

Where

- F_i is the rate of appearance of new criminals in compartments.
- V_i is the transfer of individuals out of the compartment.
- x_0 is the equilibrium state.

1.3 Equilibrium points and stability in epidemic models

Definition 1.3.1 *An **equilibrium point** (or **steady-state**) in an epidemic model is a state where the system does not change over time. Mathematically, this means that all derivatives in the system of differential equations are zero:*

$$\frac{dX}{dt} = 0$$

where X represents the state variables (e.g., the number of susceptible, infected, and recovered individuals). At equilibrium, the number of individuals in each compartment remains constant over time, meaning there is no net change in infections or recoveries [6, 8, 24].

Types of equilibrium in epidemic models:

Theorem 1.3.1 *The disease-free equilibrium (DFE) represents a situation where the infection has been eradicated ($I^* = 0$). It exists in all epidemic models because a population without infection is always a possible state. Stability of the DFE depends on the basic reproduction number R_0. If $R_0 < 1$, the disease-free equilibrium is globally asymptotically stable, meaning the epidemic will eventually fade out [18].*

Theorem 1.3.2 *The endemic equilibrium represents a situation where the disease persists at a constant level ($I^* > 0$). It exists if $R_0 > 1$, meaning that on average, each infected individual transmits the disease to more than one person. Stability of the endemic equilibrium depends on model parameters such as recovery rate, transmission rate, and control measures [18].*

Stability of equilibrium points:

The stability of an equilibrium point determines whether small perturbations (e.g., introduction of a few infected individuals) will cause the system to return to equilibrium or move away from it. If explicit solution to a system of differential equations can be calculated, then presumably the question of solution stability can be resolved by inspection of the solution formulas, but

the most system it is not possible to generate explicit solution formulas, so we have to find methods of determining stability without the use of explicit solution [51].

Local stability analysis (Jacobian Method):

To check the local stability of an equilibrium point X^*, we linearize the system by computing the Jacobian matrix J:

$$J = \left.\frac{\partial F}{\partial X}\right|_{X^*}$$

where $F(X)$ represents the system of differential equations.

- If all eigenvalues of J have negative real parts, then the equilibrium is locally asymptotically stable.
- If at least one eigenvalue has a positive real part, then the equilibrium is unstable [48].

Corollary: [48] (Corollary of Gershgorin Circle Theorem) Let A be an $(n \times n)$ matrix with real entries. If the diagonal elements a_{ii} of A satisfy $a_{ii} < -r_i$, where

$$r_i = \sum_{j=1, j\neq i}^{n} \mid a_{ij} \mid$$

for $i = 1.....n$; then the eigenvalues of A are negative or have negative real parts.

1.4 Deterministic optimal control problem

Optimal Control (OC) is the process of determining control and state trajectories for a dynamic system over a period of time in order to minimize the cost function. In epidemic control, a government want to minimize the spread of a disease by controlling vaccination or social distancing policies or treatments. Then the goal is to find the best strategy to minimize infections while keeping costs low by finding a continuous control u(t) and the associated state variable x(t) to maximize or minimize a given cost (objective) functional J [23]. The control $u(\cdot)$ is taken from the set

$$\mathcal{U}[0,T] \triangleq \left\{u : [0,T] \to U \subset \mathbb{R}^d | u(\cdot) \ \ measurable\right\},$$

and we Consider the following control system:

$$\begin{cases} \dot{x}(t) = b(t, x(t), u(t)) \\ x(0) = x_0, \end{cases} \tag{1.3}$$

where $b : [0,T] \times \mathbb{R}^d \times U \to \mathbb{R}^d$. We consider the associated cost functional:

$$J(u(\cdot)) = \int_0^T f(t, x(t), u(t))\, dt + h(x(T)). \tag{1.4}$$

where f and h are called the running cost and the terminal cost, respectively.

Definition 1.4.1 *:[22] A control $u(\cdot)$ is called an admissible control, and $(x(\cdot), u(\cdot))$ called an admissible pair, if:*

(i) $u(\cdot) \in \mathcal{U}[0,T]$;

(ii) $x(\cdot)$ *is the unique solution of the dynamic system under* $u(\cdot)$;

(iii) $x(t) \in S(t)$ *where* $S(t)$ *is a given multifunction such that for each* $t \in [0,T]$, $S(t) \subset \mathbb{R}^d$.

(iv) $t \mapsto f(t, x(t), u(t)) \in L^1[0,T]$.

The set of all admissible controls is denoted by $\mathcal{U}_{ad}[0,T]$*:*

Our deterministic optimal control problem can be stated as follows.

Problem (D).[22] Minimize J over $\mathcal{U}_{\text{ad}}[0,T]$.

Problem (D) is said to be finite if J has a finite lower bound, and is said to be uniquely solvable if there is a unique $u^*(\cdot) \in \mathcal{U}_{\text{ad}}[0,T]$ satisfying

$$J(u^*(\cdot)) = \inf_{u(\cdot) \in \mathcal{U}_{\text{ad}}[0,T]} J(u(\cdot)).$$

Any $u^*(\cdot) \in \mathcal{U}_{\text{ad}}[0,T]$ satisfying J is called an optimal control, with the corresponding state trajectory $x^*(\cdot) = x(\cdot; u^*(\cdot))$, and $(x^*(\cdot), u^*(\cdot))$ are called an optimal state trajectory and an optimal pair, respectively.

1.4.1 Existence of optimal controls

In this subsection we are going to discuss the existence of optimal controls.

Theorem 1.4.1 *[20] Let J be an objective functional on a given control set $\mathcal{U}[0,T]$. Suppose J is subject to the state system having non-negative initial conditions at $t = 0$, then there exists an optimal control u^* such that*

$$J(u^*) = \min\{J(u) : u \in \mathcal{U}[0,T]\}.$$

Proof:[20] It is necessary to verify the following four properties in proving the Theorem 1.4.1:

(i) Convexity and closure of the control set $\mathcal{U}[0,T]$.

(ii) Boundedness of the state system by a linear function in the state and control variables.

(iii) Convexity of the integrand of the objective functional with respect to the control.

(iv) There exist constants $c_1, c_2 > 0$ and $c_3 > 1$ such that the running cost is bounded below by:

$$c_1 \left(|u|^2\right)^{\frac{c_3}{2}} - c_2.$$

See [20] for more details about the proof.

Theorem 1.4.2 *[19] Let $\mathcal{U}$ be convex, and let J be strictly convex on $\mathcal{U}$. Then there exists at most one $u^* \in \mathcal{U}$ such that J has a minimum at u^*.*

Proof:[19] Let u^* and u be two solutions of the optimization problem such that $u^* \neq u$. Set $v = \frac{u^*+u}{2}$. Since J is strictly convex, we have:

$$J(u) = J(u^*) \leq J(v) < \frac{1}{2}(J(u^*) + J(u)),$$

that is $J(u) < J(u^*) < J(u)$, which is a contradiction since $J(u) = J(u^*) = \min_{u\in\mathcal{U}} J$.

1.5 Pontryagin's Maximum Principle (PMP)

Optimal control problems may be regarded as optimization problems in infinite-dimensional spaces which make them substantially difficult to solve. The maximum principle, formulated and derived by Pontryagin and his group in the 1950s, is truly a milestone of optimal control theory. It states that any optimal control along with the optimal state trajectory must solve the so-called Hamiltonian system [27, 3], which is a two-point boundary value problem (and can also be called a forward-backward differential equation, to be able to compare with the stochastic case), plus a maximum condition of a function called the Hamiltonian. Let us assume the following:

- **(D1)** (U,d) is a separable metric space and $T > 0$.
- **(D2)** The maps $b : [0,T] \times \mathbb{R}^n \times U \to \mathbb{R}^n$, $f : [0,T] \times \mathbb{R}^n \times U \to \mathbb{R}$, and $h : \mathbb{R}^n \to \mathbb{R}$ are measurable, and there exist a constant $L > 0$ and a modulus of continuity $\omega : [0,\infty) \to [0,\infty)$ such that for $\varphi(t,x,u) = b(t,x,u), f(t,x,u), h(x)$, we have

$$\begin{cases} |\varphi(t,x,u) - \varphi(t,\hat{x},\hat{u})| \leq L|x-\hat{x}| + \omega(d(u,\hat{u})), \\ |\varphi(t,0,u)| \leq L, \end{cases} \quad \forall t \in [0,T],\ x,\hat{x} \in \mathbb{R}^n,\ u,\hat{u} \in U.$$

- **(D3)** The maps b, f, and h are C^1 in x, and there exists a modulus of continuity $\omega : [0,\infty) \to [0,\infty)$ such that for $\varphi(t,x,u) = b(t,x,u), f(t,x,u), h(x)$, we have

$$|\varphi_x(t,x,u) - \varphi_x(t,\hat{x},\hat{u})| \leq \omega(|x-\hat{x}| + d(u,\hat{u})), \forall t \in [0,T],\ x,\hat{x} \in \mathbb{R}^n,\ u,\hat{u} \in U.$$

- It is clear that under **(D1)-(D2)**, for any $u(\cdot) \in \mathcal{U}_{\text{ad}}[0,T]$, equation (1.3) admits a unique solution $x(\cdot)$. Hence the preblem (1.3)–(1.4) is well-defined [22].

Theorem 1.5.1 *[22] (Deterministic Maximum Principle) Let $(D1)-(D3)$ hold . Let $(x^*(\cdot),u^*(\cdot)) \in \mathcal{U}_{\text{ad}}[0,T]$ be an optimal pair of Problem (D). Then there exists a $p(\cdot) : [0,T] \to \mathbb{R}^n$ satisfying the following adjoint equation:*

$$\begin{cases} \dot{p}(t) = -b_x(t,x^*(t),u^*(t))^T p(t) + f_x(t,x^*(t),u^*(t)), \\ p(T) = -h_x(x^*(T)), \end{cases} \tag{1.5}$$

and

$$H(t,x^*(t),u^*(t),p(t)) = \max_{u\in U} H(t,x^*(t),u,p(t)), \tag{1.6}$$

where Hamiltonian function is

$$H(t,x,u,p) \triangleq \langle p, b(t,x,u)\rangle - f(t,x,u), \quad (t,x,u,p) \in [0,T]\times\mathbb{R}^n \times U \times \mathbb{R}^n.$$

We call $p(\cdot)$ the adjoint function. The necessary condition (1.6), corresponding to the optimal adjoint equation, along with, the maximization condition, can be written as:

$$\begin{cases} \dot{x}(t) = H_p(t,x(t),u(t),p(t)), \\ \dot{p}(t) = -H_x(t,x(t),u(t),p(t)), \\ x(0) = x_0 \quad p(T) = h_x(x(T)) \\ H(t,x(t),u(t),p(t)) = \max_{u\in U} H(t,x(t),u,p(t)), \end{cases}$$

The above system is called an Hamiltonian system.

Remark 1.5.1 *[1] We can switch back and forth between maximization and minimization:*

$$min(J) = -max(-J).$$

Example: Let $x(t)$ represent the number of tumor cells at time t, with exponential growth factor α, and $u(t)$ the drug concentration. The aim is to minimize the number of tumor cells

at the end of the treatment period and the accumulated harmful effects of the drug on the body[1]. This problem is formulated as minimize the cost function J:

$$J = x(T) + \int_0^T u^2 dt$$
$$\dot{x} = \alpha x - u, \quad x(0) = x_0$$

Let us consider the Hamiltonian

$$H(t, x, u, \lambda) = u^2 + \lambda(\alpha x - u).$$

The optimality condition is given by

$$\frac{\partial H}{\partial u} = 0 \implies u^* = \frac{\lambda}{2}.$$

The adjoint condition is given by

$$\dot{\lambda} = -\frac{\partial H}{\partial x} \iff \dot{\lambda} = -\alpha\lambda \implies \lambda = Ce^{-\alpha t},$$

Using the transversality condition $p(T) = 1$ (note that $h(x(T)) = x$, so $h_x(x(T)) = 1$), we obtain

$$\lambda(t) = e^{\alpha(T-t)}.$$

and

$$u^* = \frac{e^{\alpha(T-t)}}{2}.$$

The optimal state trajectory is (using $\dot{x} = \alpha x - u$ and $x(0) = x_0$):

$$x^* = x_0 e^{\alpha t} + e^{\alpha T} \frac{e^{-\alpha T} - e^{\alpha t}}{4\alpha}.$$

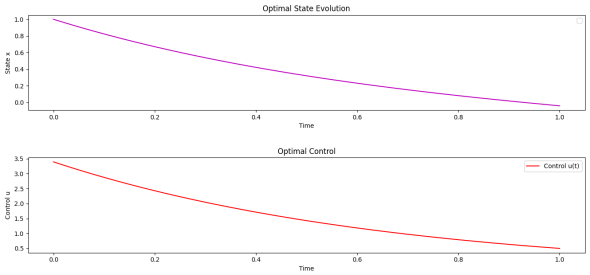


Figure 1.9: The optimal curves for tumor cells and the drug concentration

Chapter 2

Stochastic Epidemic Models and Optimal Control

A stochastic epidemic model is a mathematical framework used to describe the spread of infectious diseases in a population while accounting for random variations and uncertainties. Unlike deterministic models, which use fixed equations to predict disease dynamics, stochastic models incorporate probabilistic elements to represent real-world randomness, such as variations in transmission, recovery, and other epidemiological factors [51][43].
These models typically use stochastic processes, such as stochastic differential equations (SDEs), to simulate the unpredictable nature of disease transmission. They are particularly useful for studying outbreaks in small populations, rare events, or cases where randomness plays a significant role in disease dynamics.

2.1 Forward stochastic differential equations

Let $T > 0$ and $(\Omega, \mathcal{F}, \mathbb{P})$ be a filtered probability space, where $W : [0, T] \times \Omega \to \mathbb{R}^d$ is a d-dimensional standard $\mathbb{F}$-Brownian motion on $(\Omega, \mathcal{F}, \mathbb{P})$, $\mathbb{F} = \{\mathcal{F}_t\}_{t \geq 0}$ is the natural filtration generated by the Brownian motion W. Suppose that $(\Omega, \mathcal{F}, \mathbb{P})$ is complete, $\mathcal{F}_0$ contains all the $\mathbb{P}$-null sets in $\mathcal{F}$, and $\mathbb{F}$ is right continuous [21]. A forward stochastic differential equation FSDE or simply SDE $E_x(b, \sigma)$ is an equation formally written

$$dx_t = b(t, x_t)dt + \sigma(t, x_t)dW_t$$

b, σ are called the drift coefficient and the diffusion coefficient, respectively, these coefficients are locally bounded measurable functions. The SDE Equation is interpreted as the stochastic

integral equation

$$x_t = x_0 + \int_0^t b(t, x_s)ds + \int_0^t \sigma(t, x_s)dW_s$$

Theorem 2.1.1 *(Cauchy-Lipschitz for SDE_s) We assume that there exist constant K positive such that $\forall t \geq 0, x, y \in \mathbb{R}^d$*

$$|b(t,x) - b(t,y)| + |\sigma(t,x) - \sigma(t,y)| \leq K|x-y|$$

$$|b(t,x)| \leq K(1+|x|), \quad |\sigma(t,x)| \leq K(1+|x|)$$

then the $E_x(b,\sigma)$ has a unique continuous solution.

2.2 Backward stochastic differential equations

For ordinary differential equations (ODEs), both initial value and terminal value problems can be well-posed under suitable regularity conditions. In fact, a terminal value problem can be transformed into an equivalent initial value problem through the time reversal, where denotes the time variable and is the terminal time. However, the situation is more subtle for stochastic differential equations (SDEs), as such a time transformation would break the the adaptivity of the solution. In this section, we aim to provide an intuitive motivation for formulating a backward stochastic differential equation (BSDE) that maintains the adaptedness of its solution [38].

Notations

1. $S^2(\mathbb{R}^k)$: is the vector space consisting of processes Y, progressively measurable, with values in $\mathbb{R}^k$, such that:
$$\|Y\|_{S^2}^2 := \mathbb{E}\left[\sup_{0\leq t\leq T} |Y_t|^2\right] < \infty,$$
and $S_c^2(\mathbb{R}^k)$ is the subspace formed by continuous processes. Two indistinguishable processes are always identified.

2. $M^2(\mathbb{R}^{k\times d})$: is the vector space consisting of processes Z, progressively measurable, with values in $\mathbb{R}^{k\times d}$, such that:
$$\|Z\|_{M^2}^2 := \mathbb{E}\left[\int_0^T \|Z_t\|^2 dt\right] < \infty,$$
where if $z \in \mathbb{R}^{k\times d}$, then $\|z\|^2 = \text{trace}(zz^*)$. $M^2(\mathbb{R}^{k\times d})$ denotes the equivalence classes in $M^2(\mathbb{R}^{k\times d})$.

3. The spaces S^2, S_c^2, and M^2 are Banach spaces under the norms defined above. We denote the product space $S_c^2(\mathbb{R}^k) \times M^2(\mathbb{R}^{k\times d})$ by $\mathcal{B}^2$.

4. We define an application f defined on $[0,T]\times\Omega\times\mathbb{R}^k\times\mathbb{R}^{k\times d}$ with values in $\mathbb{R}^k$, such that for any $x\in\mathbb{R}^k$, $z\in\mathbb{R}^{k\times d}$, the process $(f(t,\omega,x,z))_{0\le t\le T}$ is progressively measurable with respect to $\mathcal{F}_T$ (or $\mathcal{F}_t$-measurable), and with values in $\mathbb{R}^k$.

We start by a simple example (but it's illustrative) to know $f\equiv 0$. Let $m=1$, $T>0$, and $\xi\in L^2(\Omega;\mathbb{R})$. Consider the following stochastic differential equation [26]

$$dY(t)=0,\quad t\in[0,T]; Y(T)=\xi.$$

It's impossible to find an $\mathcal{F}_t$-adapted solution $Y(\cdot)$, since the only solution of this equation is

$$Y(t)=\xi\quad t\in[0,T].$$

A natural way to making this equation $\mathcal{F}_t$-adapted is to redefine $Y(\cdot)$ as follows:

$$Y(t)=\mathbb{E}(\xi|\mathcal{F}_t),\quad t\in[0,T].$$

Then, $Y(\cdot)$ is $\mathcal{F}_t$-adapted and satisfies the terminal condition $Y(T)=\xi$. Noting that the process $Y(\cdot)$ defined by the last equation is a square integrable $\mathcal{F}_t$-martingale. By the martingale representation theorem, we can find an $\mathcal{F}_t$-adapted process $Z(\cdot)\in L^2([0,T];\mathbb{R})$ such that

$$Y(t)=Y(0)+\int_0^t Z(s)dW(s),\quad \forall t\in[0,T],\quad P\text{-a.s.}$$

then

$$\xi=Y(T)=Y(0)+\int_0^T Z(s)dW(s).$$

Hence,

$$Y(t)=\xi-\int_t^T Z(s)dW(s),\quad \forall t\in[0,T].$$

Here, the role of the process Z is to make the process Y adapted.

We allow f to depend on the process Z, the equation therefore becomes:

$$-dY_t=f(t,Y_t,Z_t)dt-Z_tdW_t,$$

with

$$Y_T=\xi,$$

or, equivalently, the BSDE is

$$Y_t=\xi+\int_t^T f(s,Y_s,Z_s)ds-\int_t^T Z_sdW_s,\quad 0\le t\le T.$$

Definition 2.2.1 *[26] A solution of BSDE is a pair* $(Y_t, Z_t)_{0\leq t\leq T}$ *satisfying:*

1. Y *and* Z *are progressively measurable at values respectively in* $\mathbb{R}^k$ *and* $\mathbb{R}^{k\times d}$.
2. $\int_0^T |f(s, Y_s, Z_s)|ds + \int_0^T \|Z_s\|^2 ds < \infty$ *P-a.s.*
3. *P-a.s., we have*

$$Y_t = \xi + \int_t^T f(s, Y_s, Z_s)ds - \int_t^T Z_s dW_s, \quad 0 \leq t \leq T.$$

Proposition [26] We suppose that exists a process $(f_t)_{0\leq t\leq T} \in M^2(\mathbb{R})$, and a constant $\lambda > 0$ such as

$$|f(t, y, z)| \leq f_t + \lambda(|y| + ||z||), \quad \forall (t, y, z) \in [0, T] \times \mathbb{R}^k \times \mathbb{R}^{k\times d}.$$

If $(Y_t, Z_t)_{0\leq t\leq T}$ is a solution of BSDE such as $Z \in M^2$, then Y belongs to S_c^2.

Lipschitz case

Pardoux−Peng result

We give some assumption [26]. *There exists a constant* λ *P-a.s. such that :*

1. **(H1)**: **Lipschitz condition in (y,z):** for all $t, y, \bar{y}, z, \bar{z}$,

$$|f(t, y, z) - f(t, \bar{y}, \bar{z})| \leq \lambda(|y - \bar{y}| + \|z - \bar{z}\|).$$

2. **(H2)**: **Integrability condition**

$$\mathbb{E}\left[|\xi|^2 + \int_0^T |f(s, 0, 0)|^2 ds\right] < \infty.$$

Theorem 2.2.1 *[26](Pardoux−Peng) Under* ***(H1)****and* ***(H2)*** *the BSDE has a unique solution* (Y, Z).

2.3 Numerical method for forward backward stochastic differential equations

Most forward backward stochastic differential equations can not be solved analytically and thus numerical methods must be applied in order to approximate their solutions. There have been many numerical methods proposed over the past few decades for the most part, in a complex

and scattered manner, with each requiring a variety of different and similar assumptions and conditions. In this section we will introduce the Euler scheme for the forward SDEs then we will focus on the backward Euler methods and least-squares approach for BSDEs to estimate the conditional expectations.

Forward Euler methods

Now, provide a brief explanation of the numerical approximation technique that was employed to resolve FSDEs. The Japanese mathematician G. Maruyama developed the Euler-Maruyama method, which is an extension of the Euler method, as a numerical integration methodology for estimating solutions for a system of stochastic differential equations from a given initial value $X_0 = x_0$ [49].

Let $0 = t_0 < t_1 < \cdots < t_{k-1} < t_k = T$ be a partition of the interval $[0, T]$, where the length of each subinterval is $\Delta t = t_{i+1} - t_i = T/k$, which implies that $t_{i+1} = t_i + \Delta t = i\Delta t$ and

$$\Delta W_i = W(t_i + \Delta t) - W(t_i).$$

For each stochastic process trajectory, the value of $X_{t_{i+1}}$ is approximated using only the value of the previous time step, X_{t_i}. Then, to find the trajectories or approximate solutions of a stochastic differential equation by the Euler-Maruyama method, the following equation is implemented:

$$X_{t_{i+1}} = X_{t_i} + b(t_i, X_{t_i})\Delta t + \sigma(t_i, X_{t_i})\Delta W_i$$

for all $i = 0, 1, \ldots, k-1$. In order to carry out the method computationally, it is essential to understand how to compute ΔW_i. Since the partition is made up of equal intervals, the differences ΔW_i, $i = 0, 1, \ldots, k-1$ have the same distribution, $\Delta W_i \sim N(0, \Delta t)$. Let η be a random variable with a standard normal distribution $\eta \sim N(0, 1)$. Then $\sqrt{\Delta t}\eta$ has a normal distribution with zero mean and variance Δt, that is, $\sqrt{\Delta t}\eta \sim N(0, \Delta t)$.

When the diffusion coefficient is identically zero, that is when $\sigma \equiv 0$, the stochastic iterative scheme reduces to the deterministic Euler scheme for the ordinary differential equation.

Backward Euler methods

The first class of backward numerical methods we review are the so called backward Euler schemes for BSDEs. In the present context, there are certainly two general categories of explicit and implicit discretization schemes for the FBSDE [36] which can be summarized as follows:

Algorithm 2: Backward Scheme for BSDE

1 **Initialization:** Approximate the terminal condition $Y^n_{t_n} = \Phi(X^n_{t_n})$ with the Euler-Maruyama scheme X^n.

2 **for** $k = (n-1)$ *to* 0 **do**

3 $$Z^n_{t_k} = \frac{1}{t_{k+1} - t_k} \mathbb{E}\left[Y^n_{t_{k+1}} (W_{t_{k+1}} - W_{t_k})^\top \middle| \mathcal{F}_{t_k} \right],$$

4 Compute $Y^n_{t_k}$ using either:

$$Y^n_{t_k} = \begin{cases} \mathbb{E}\left[Y^n_{t_{k+1}} + f(t_k, X_{t_k}, Y_{t_{k+1}}, Z^n_{t_k})(t_{k+1} - t_k) \middle| \mathcal{F}_{t_k} \right], & \text{(explicit)} \\ \mathbb{E}\left[Y^n_{t_{k+1}} \middle| \mathcal{F} t_k \right] + f(t_k, X_{t_k}, Y_{t_k}, Z^n_{t_k})(t_{k+1} - t_k). & \text{(implicit)} \end{cases}$$

5 **end for**

Note: The implicit scheme often provides better properties and performance relative to the explicit scheme, with these benefits coming in exchange for the additional computing effort for solving the defining equation for $Y^n_{t_k}$.

Least-squares regression based methods for BSDEs

Least-squares regression based methods was applied to solve BSDEs. The basic idea here is to replace the conditional expectations by projections on finite-dimensional subspaces which are spanned by pre-selected basis functions. The coefficients for the projection on the finite-dimensional subspaces are approximated by the solution of a linear least-squares problem making use of simulated sample paths [52]. It can be applied to compute conditional expectations of the form $E[Y|X]$ for square-integrable random variables X and Y numerically, provided a machinery for sampling independent copies of the pair (X, Y) [53][36]. The method builds upon the elementary property that $E[Y|X] = u(X)$, where the function u solves:

$$u = \arg\min_v E[|v(X) - Y|^2]$$

and v runs over all measurable functions with $E[|v(X)|^2] < \infty$.

Methods that fit into this category are ones which use a form of least-squares regression to evaluate the conditional expectations appearing in a discretization. Here, we proceed with representative methods of [40] to illustrate this type of method in a clear and concise manner. Let $\Delta_k = T/n$, that is, $t_k = kT/n$ and $\Delta W_k = W_{t_{k+1}} - W_{t_k}$ for $k \in \{0, 1, \cdots, n\}$. For

each time point t_k, we take $\mathbb{R}^K$-valued deterministic function bases $(e^K_{i,k})_{i\in\{0,1,\cdots,d\}}$. For every $k \in \{1, \cdots, n\}$, let $\{\Delta W^m_k\}_{m\in\{1,\cdots,M\}}$ be independent copies of ΔW_k. For the time discretization we consider a partition $\pi = \{t_0.....t_k\}$ of the interval $[0, T]$ i.e $0 = t_0 < t_1 < \cdots < t_{k-1} < t_k = T$ and let $\{X^{\pi,m}_k\}_{m\in\{1,\cdots,M\}}$ be corresponding copies of X^π_k. This method can be summarized in the following algorithm:

Algorithm 3: Least-squares regression based methods for BSDEs

Set $y^{n,M,K}_n(\cdot) = \Phi(\cdot)$

for $k = (n-1)$ *to* 1 **do**

$$\alpha^{M,K}_{i,k} = \underset{\alpha}{\operatorname{argmin}} \frac{1}{M}\sum_{m=1}^{M}\left| y^{n,M,K}_{k+1}(X^{\pi,m}_{k+1})\frac{\Delta W^{i,m}_k}{\Delta_k} - \alpha \cdot e^K_{i,k}(X^{\pi,m}_k)\right|^2,$$

for$(i \in \{1, \cdots, d\})$

$z^{n,M,K}_k = (z^{n,M,K}_{1,k}, \cdots, z^{n,M,K}_{d,k})$ where $z^{n,M,K}_{i,k}(\cdot) = \alpha^{M,K}_{i,k} \cdot e^K_{i,k}(\cdot)$

$$\alpha^{M,K}_{0,k} = \underset{\alpha}{\operatorname{argmin}} \frac{1}{M}\sum_{m=1}^{M} |y^{n,M,K}_{k+1}(X^{\pi,m}_{k+1})$$
$$+\Delta_k f(t_k, X^{\pi,m}_k, y^{n,M,K}_{k+1}(X^{\pi,m}_{k+1}), z^{n,M,K}_k(X^{\pi,m}_k)) - \alpha \cdot e^K_{0,k}(X^{\pi,m}_k)|^2.$$

$y^{n,M,K}_k(\cdot) = \alpha^{M,K}_{0,k} \cdot e^K_{0,k}(\cdot)$

end for

Return

$$Y^{\pi,M,K}_0 = \frac{1}{M}\sum_{m=1}^{M}\left(y^{n,M,K}_1(X^{\pi,m}_1) + \Delta_1 f(t_0, \mathbf{x}, y^{n,M,K}_1(X^{\pi,m}_1), z^{n,M,K}_1(X^{\pi,m}_1))\right)$$

Under the Lipschitz continuity in the state variables, the (1/2)-Hölder continuity in time of the coefficients of the Markovian BSDE, and the condition that for all measurable functions φ such that $\varphi(X^\pi_k) \in L^2(\Omega)$, there is $(\beta^K_k)_k$ such that $\beta^K \cdot e^K_{i,k}(X^\pi_k) \to \varphi(X^\pi_k)$ in $L^2(\Omega, \mathcal{F}_k)$ as $K \to \infty$.

2.4 Stochastic Epidemic Models

Considering that white noise is in direct proportional to populations : S, E, I, R introduced in the first chapter. Thus, we get the following stochastic models [21][12].

Stochastic SIR Model

The stochastic SIR model includes **susceptible (S)**, **infected (I)**, and **recovered (R)** individuals

$$dS = -\beta SI\,dt + \epsilon S\,dW, \tag{2.1}$$

$$dI = (\beta SI - \gamma I)\,dt + \epsilon I\,dW, \tag{2.2}$$

$$dR = \gamma I\,dt + \epsilon R\,dW, \tag{2.3}$$

where:

- β is the transmission rate.
- γ is the recovery rate.
- W is Brownian motions.
- ϵ is noise intensity.

Stochastic SI Model

The SI model assumes no recovery, meaning individuals remain infected indefinitely.

$$dS = -\beta SI\,dt + \epsilon S\,dW, \tag{2.4}$$

$$dI = \beta SI\,dt + \epsilon I\,dW. \tag{2.5}$$

Stochastic SEIR Model

The SEIR model includes an **exposed (E)** compartment for individuals who are infected but not yet infectious.

$$dS = -\beta SI\,dt + \epsilon S\,dW, \tag{2.6}$$

$$dE = (\beta SI - \alpha E)\,dt + \epsilon E\,dW, \tag{2.7}$$

$$dI = (\alpha E - \gamma I)\,dt + \epsilon I\,dW, \tag{2.8}$$

$$dR = \gamma I\,dt + \epsilon R\,dW, \tag{2.9}$$

where α is the rate of progression from exposed to infectious.

Stochastic SIS Model

The SIS model assumes no immunity, meaning recovered individuals can become susceptible again.

$$dS = (-\beta SI + \gamma I)\,dt + \epsilon S\,dW, \tag{2.10}$$

$$dI = (\beta SI - \gamma I)\,dt + \epsilon I\,dW. \tag{2.11}$$

We use the following algorithm to simulate the stochastic epidemic models with parameters : $\beta = 0.0003, \alpha = 0.2, \gamma = 0.1, \epsilon = 0.02$

Algorithm 4: Stochastic epidemic models simulation

Input: Initial value ,t_0, X_{t_0},Δt,K,T

Output: Approximate solution $\{X_{t_{i+1}}\}$,$\mathbb{E}[X_{t_{i+1}}]$

Monte carlo simulation :

for $m = 0$ *to 200* **do**

  Set $k \leftarrow T/\Delta t$ (Number of steps) ;

  for $i = 0$ *to* $k - 1$ **do**

    Generate $\eta_i \sim \mathcal{N}(0, 1)$;

    $X_{t_{i+1}} = X_{t_i} + b(X_{t_i}, t_i)\Delta t + \sigma(X_{t_i}, t_i)\sqrt{\Delta t}\eta_i$;

    $t_{i+1} = t_i + \Delta t$;

    endFor

  compute the expected value $\mathbb{E}[X_{t_{i+1}}]$;

  endFor

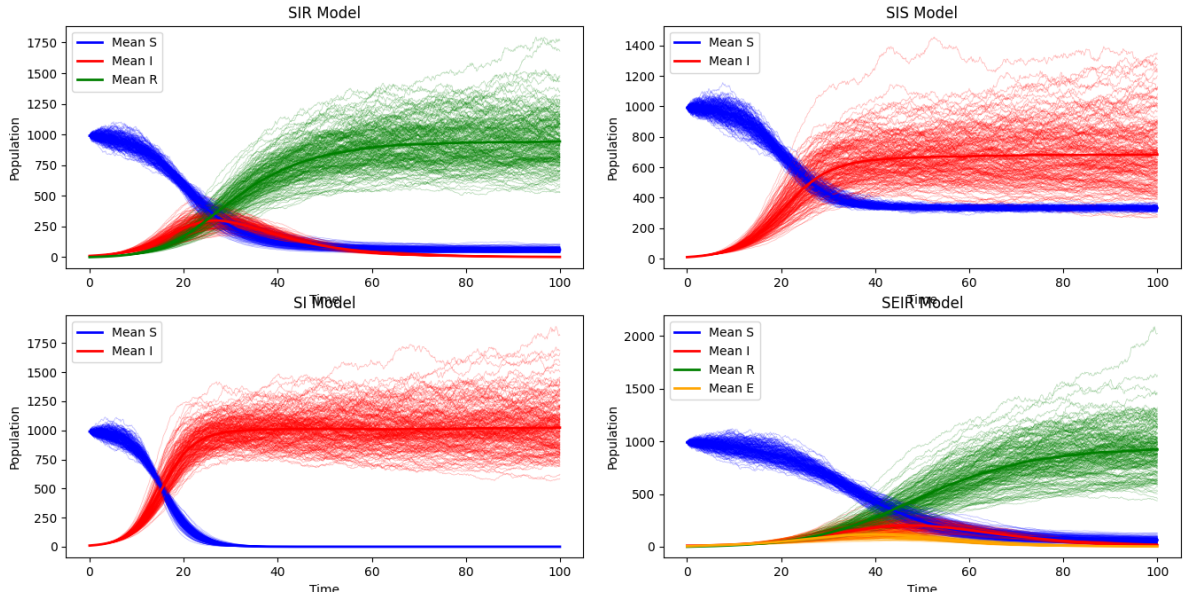


Figure 2.1: Stochastic epidemic models simulation. The program execution took 11.39 seconds to complete the full simulation.

Note: *The Monte Carlo method for SDEs involves generating random samples of the Brownian motion and using them to simulate the evolution of the stochastic process over time. By averaging the outcomes of multiple simulations, an approximation of the solution to the SDE can be obtained.*

2.4.1 Stochastic Lyapunov Function

Definition 2.4.1 *A* ***Stochastic Lyapunov Function*** $V(X)$ *is a non-negative function. It satisfies conditions similar to the deterministic Lyapunov method but considers the stochastic dynamics[11]. A function* $V(X)$ *is a* ***Lyapunov function*** *for the stochastic system if:*

- $V(X) > 0$ *for all* X *different from* X^* *such that* $V(X^*) = 0$.
- *The* ***Stochastic Differential Operator (Generator)*** $\mathcal{L}V(X)$ *is negative definite in a region containing* X^*.

Consider the stochastic differential equation (SDE) of n-dimensional form:

$$dX(t) = F(t, X(t))dt + G(t, X(t))dW(t), \tag{2.12}$$

where $F(t, X) : \mathbb{R}^+ \times \mathbb{R}^n \to \mathbb{R}^n$ *and* $G(t, X) : \mathbb{R}^+ \times \mathbb{R}^n \to \mathbb{R}^{n\times m}$ *are measurable functions, and* $W(t)$ *is an* $\mathbb{R}^m$*-valued standard Brownian motion. Given* $V(X, t) \in C^{2,1}(\mathbb{R}^n \times \mathbb{R}^+, \mathbb{R})$ *[39], we*

define the operator $\mathcal{L}$ corresponding to the SDE by

$$\mathcal{L}V = V_t(X,t) + V_X(X,t)F(X,t) + \frac{1}{2}trace\left[G^T(X,t)V_{XX}(X,t)G(X,t)\right],$$

where

$$V_t(X,t) = \frac{\partial V(X,t)}{\partial t}, \quad V_X(X,t) = \left(\frac{\partial V}{\partial x_1}, \frac{\partial V}{\partial x_2}, \ldots, \frac{\partial V}{\partial x_n}\right), \quad V_{XX}(X,t) = \left(\frac{\partial^2 V}{\partial x_i \partial x_j}\right)_{n\times n}.$$

- *The second term corresponds to the deterministic dynamics.*
- *The $\frac{1}{2}trace\left[G^T(X,t)V_{XX}(X,t)G(X,t)\right]$ term accounts for the stochastic perturbations.*

Then, the corresponding Itô formula can be obtained as :

$$dV(X,t) = \mathcal{L}V(X,t)dt + V_X(X,t)G(X,t)dW(t).$$

2.5 Stochastic optimal control problem

The stochastic optimal control problem is performed by using Stochastic Maximum Principle. The mathematical significance of the Maximum Principle lies in its ability to simplify the original infinite-dimensional optimal control problem into a more tractable form by focusing on the maximization of the Hamiltonian. This often enables the derivation of closed-form solutions for specific classes of control problems. Pontryagin's original formulation of the Maximum Principle was developed for deterministic systems, drawing upon ideas from classical calculus of variations. The standard approach involves perturbing an optimal control using a spike variation and analyzing the first-order term in a Taylor expansion [22]. As the perturbation tends to zero, a variational inequality emerges, leading to the Maximum Principle through a duality argument.

However, extending this method to stochastic control problems presents substantial challenges, particularly when the diffusion coefficient depends on the control . the standard first-order variation techniques insufficient. Addressing this issue requires considering both first- and second-order terms in the expansion, resulting in a more complex stochastic maximum principle involving a forward-backward stochastic differential system and an additional quadratic term in the diffusion coefficient [22]. In this thesis, we restrict our attention to the case where the diffusion term is independent from the control.

In the following subsection, we introduce the preliminaries of stochastic optimal control.

2.5.1 Controlled stochastic differential equations

Given a filtered probability space $(\Omega, \mathcal{F}, (\mathcal{F}_t)_{t\geq 0}, \mathbb{P})$ satisfying the usual condition on which an d-dimensional standard Brownian motion W(.) is defined [22], consider the following controlled stochastic differential equation:

$$\begin{cases} dx(t) = b(t, x(t), u(t))dt + \sigma(t, x(t))dW_t, \\ x(0) = x_0 \in \mathbb{R}^n \end{cases} \tag{2.13}$$

The cost functional is:

$$J(u(\cdot)) = \mathbb{E}\left[\int_0^T f(t, x(t), u(t))dt + h(x(T))\right]. \tag{2.14}$$

where $u(t) \in [0, T]$ is an admissible control process, i.e., a $\mathbb{F}$-adapted square-integrable process valued in a given subset U of $\mathbb{R}^d$. The drift coefficient b and the diffusion coefficient σ are deterministic functions:

$$b : [0, T] \times \mathbb{R}^n \times U \to \mathbb{R}^n,$$

$$\sigma : [0, T] \times \mathbb{R}^n \to \mathbb{R}^{n\times d}.$$

$$f : [0, T] \times \mathbb{R}^n \times U \to \mathbb{R},$$

$$h : [0, T] \times \mathbb{R}^n \to \mathbb{R},$$

$$\mathcal{U}[0, T] \triangleq \left\{u : [0, T] \times \Omega \to U \mid u \in L^2_{\mathbb{F}}(0, T; \mathbb{R}^d)\right\}, \tag{2.15}$$

where

$$L^2_{\mathbb{F}}(0, T; \mathbb{R}^d) \triangleq \left\{x : [0, T] \times \Omega \to \mathbb{R}^d \text{ is } \mathbb{F}\text{-adapted and } \mathbb{E}\left[\int_0^T |x_t|^2 dt\right] < \infty\right\}.$$

Definition 2.5.1 *[26],[22] Let $(\Omega, \mathcal{F}, \{\mathcal{F}_t\}_{t\geq 0}, \mathbb{P})$ be given satisfying the usual conditions and $W(t)$ be a d-dimensional standard $\{\mathcal{F}_t\}_{t\geq 0}$-Brownian motion. A control $u(\cdot)$ is called an s-admissible control, and $(x(\cdot), u(\cdot))$ an s-admissible pair, if*

(i) $u(\cdot) \in \mathcal{U}[0, T]$;

(ii) $x(\cdot)$ *is the unique solution of equation* (2.13)

(iii) some prescribed state constraints are satisfied.

(iv) $f(\cdot, x(\cdot), u(\cdot)) \in L^1_{\mathcal{F}}(0,T;\mathbb{R})$; and $h(x_T) \in L^1_{\mathcal{F}}(\Omega;\mathbb{R})$.

The set of all admissible controls is denoted by $\mathcal{U}^s_{\text{ad}}[0,T]$.

Problem (P): [22] Our stochastic optimal control problem can be stated as minimizing the cost function J over $\mathcal{U}^s_{\text{ad}}[0,T]$.

The goal is to find $u^*(\cdot) \in \mathcal{U}^s_{\text{ad}}[0,T]$ (if it exists) such that:

$$J(u^*(\cdot)) = \inf_{u(\cdot)\in\mathcal{U}^s_{\text{ad}}[0,T]} \mathbb{E}\left[\int_0^T f(t,x(t),u(t))dt + h(x(T))\right]. \tag{2.16}$$

- Problem (P) is said to be s-finite if the right-hand side of (2.16) is finite, and it is said to be (uniquely) s-solvable if there exists a (unique) $u^*(\cdot) \in \mathcal{U}^s_{\text{ad}}[0,T]$ such that (2.16) holds.
- Any $u^*(\cdot) \in \mathcal{U}^s_{\text{ad}}[0,T]$ satisfying (2.16) is called an *optimal control*.
- The corresponding state process $x^*(\cdot)$ and the state-control pair $(x^*(\cdot), u^*(\cdot))$ are called an optimal state process and an optimal pair, respectively.

2.5.2 Existence of optimal controls

Mazur's theorem: [54] *Let $(x_n) \rightharpoonup x$ weakly as $n \to \infty$ in a normed linear space $\mathcal{X}$. Then there exists, for any $\varepsilon > 0$, a convex combination $\sum_{j=1}^n \alpha_j x_j$ $\left(\alpha_j \geq 0,\ \sum_{j=1}^n \alpha_j = 1\right)$ of x_j such that*

$$\left\| x - \sum_{j=1}^n \alpha_j x_j \right\| \leq \varepsilon.$$

Now consider the following stochastic linear controlled system [26]:

$$\begin{cases} dx(t) = [Ax(t) + Bu(t)]dt + Cx(t)dW(t), \quad t \in [0,T], \\ x(0) = x_0, \end{cases} \tag{2.17}$$

where A, B, and C are matrices of suitable sizes. The state $x(t)$ takes values in $\mathbb{R}^n$, and the control $u(\cdot)$ is in $\mathcal{U}[0,T]$. We introduce the following assumptions:

(H1) The set $U \subset \mathbb{R}^k$ is convex and closed, and the functions f and h are convex and for some $\alpha, K > 0$

$$f(x,u) \geq \alpha|u|^2 - K, \quad h(x) \geq -K, \quad \forall (x,u) \in \mathbb{R}^n \times U. \tag{2.18}$$

(H2) The set $U \subset \mathbb{R}^k$ is convex and compact, and the functions f and h are convex.

Theorem 2.5.1 *[26] Under either (H1) or (H2), if **Problem (P)** is finite, then it admits an optimal control.*

Proof. We suppose that (H1) holds. Let $x_j(\cdot), u_j(\cdot)$ be a minimizing sequence. By (2.18), we have

$$E\int_0^T |u_j(t)|^2 dt \leq K, \quad \forall j \geq 1.$$

for some positive constant K. Thus, there is a subsequence which is still labeled by $u_j(\cdot)$, such that

$$u_j(\cdot) \rightharpoonup u^*(\cdot), \quad \text{in } L^2_{\mathcal{F}}(0,T;\mathbb{R}^k), \quad \text{weakly convergence.}$$

By Mazur's theorem, we have a sequence of convex combinations

$$\tilde{u}_j(\cdot) \triangleq \sum_{i\geq 1} \alpha_{ij} u_{i+j}(\cdot), \quad a_{ij} \geq 0 \quad \text{and} \quad \sum_{i\geq 1} \alpha_{ij} = 1,$$

such that

$$\tilde{u}_j(\cdot) \to u^*(\cdot), \quad \text{in } L^2_{\mathcal{F}}(0,T;\mathbb{R}^k), \quad \text{Strongly convergence.}$$

Since the set $U \subseteq \mathbb{R}^k$ is convex and closed, it follows that $u^*(\cdot) \in \mathcal{U}^s[0,T]$.
On the other hand, if $\tilde{x}_j(\cdot)$ is the state under the control $\tilde{u}_j(\cdot)$, here we have the convergence

$$\tilde{x}_j(\cdot) \to x^*(\cdot), \quad \text{strongly in } C_{\mathcal{F}}([0,T],\mathbb{R}^n).$$

So, $(x^*(\cdot), u^*(\cdot))$ is admissible, and the convexity of f and h implies

$$\begin{aligned} J(u^*(\cdot)) &= \lim_{j\to\infty} J(\tilde{u}_j(\cdot)) \leq \lim_{j\to\infty} \sum_{i\geq 1} \alpha_{ij} J(u_{i+j}(\cdot)), \\ &= \inf_{u(\cdot)\in\mathcal{U}^s[0,T]} J(u(\cdot)). \end{aligned}$$

Hence, $(x^*(\cdot), u^*(\cdot))$ is an optimal pair. □

2.5.3 Statement of Stochastic maximum principle

Let us make the following assumptions:

- **(S0):** $\{\mathcal{F}_t\}_{t>0}$ is the natural filtration generated by $W(t)$.
- **(S1):** (U,d) is a separable metric space and $T > 0$.
- **(S2):** The maps b, σ, f, h are measurable, and there exist a constant $L > 0$ and a modulus of continuity $\bar{\omega} : [0,\infty) \to [0,\infty)$ such that for σ and $\varphi = b, h, f$, we have

$$\begin{cases} |\varphi(t,x,u) - \varphi(t,\bar{x},\bar{u})| \leq L|x-\bar{x}| + \bar{\omega}(d(u,\bar{u})), \\ |\sigma(t,x) - \sigma(t,\bar{x})| \leq L|x-\bar{x}| \\ |\varphi(t,0,u)| \leq L, \quad \forall t \in [0,T], x, \bar{x} \in \mathbb{R}^n, u, \bar{u} \in U, \end{cases}$$

- **(S3):** The maps b, σ, f, h are C^1 in x. Moreover, there exist a constant $L > 0$ and a modulus of continuity $\bar{\omega} : [0, \infty) \to [0, \infty)$ such that for σ and $\varphi = b, h, f$, we have:

$$\begin{cases} |\varphi_x(t, x, u) - \varphi_x(t, \bar{x}, \bar{u})| \leq L|x - \bar{x}| + \bar{\omega}(d(u - \bar{u})) \\ |\sigma_x(t, x) - \sigma_x(t, \bar{x})| \leq L|x - \bar{x}|, \quad \forall t \in [0, T], x, \bar{x} \in \mathbb{R}^n, u, \bar{u} \in U, \end{cases}$$

- Given $u(\cdot) \in \mathcal{U}[0, T]$, equation (2.13) is an SDE with random coefficients. We see that under assumptions $(S0) - (S2)$, for any $u(\cdot) \in \mathcal{U}[0, T]$, the state equation (2.13) admits a unique solution $x(\cdot) \equiv x(\cdot; x(\cdot))$ and the cost functional (2.14) is well defined. In the case where $x(\cdot)$ is the solution of (2.13) corresponding to $u(\cdot) \in \mathcal{U}[0, T]$, we call $(x(\cdot), u(\cdot))$ an admissible pair, and $x(\cdot)$ an admissible state process (trajectory).

Before introducing a set of sufficient conditions for the Stochastic Maximum Principle (SMP), we firstly introduce the adjoint equations involved in a SMP.

Adjoint equation

Let $(x^*(\cdot), u^*(\cdot))$ be a given optimal pair [22]. We introduce the adjoint BSDE as follows:

$$\begin{cases} dp(t) = -\left[b_x(t, x^*(t), u^*(t))^\top p(t) + \sum_{j=1}^d \sigma_x^j(t, x^*(t))^\top q_{t,j} - f_x(t, x^*(t), u^*(t))\right] dt + q(t) dW_t, \\ p(T) = -h_x(x^*(T)), \quad t \in [0, T], \end{cases} \tag{2.19}$$

- where $p(\cdot)$ and $q(\cdot)$ are two $\mathcal{F}$ adapted processes which should be solved.
- Any pair of processes $(p(\cdot), q(\cdot)) \in \mathcal{L}^2(0, T; \mathbb{R}^n) \times (\mathcal{L}^2(0, T; \mathbb{R}^n))^d$ satisfying (2.19) is called an adapted solution of (2.19).
- We refer to (2.19) as the first-order adjoint equations and to $p(\cdot)$ as the first-order adjoint process.
- Under $(S0) - (S3)$, for any $(x^*(\cdot), u^*(\cdot))$ admits a unique adapted solution $(p(\cdot), q(\cdot))$.

The sufficient conditions for the SMP given by the following theorem.

Theorem 2.5.2 *Let Assumptions $(S0) - (S3)$ hold. Let $(x^*(\cdot), u^*(\cdot), p(\cdot), q(\cdot))$ be an admissible 4-tuple. Suppose that $h(\cdot)$ is convex, $H(t, \cdot, \cdot, p(t), q(t))$ defined by*

$$H(t, x, u, p, q) = \langle p, b(t, x, u) \rangle + tr[q^\top \sigma(t, x)] - f(t, x, u),$$

where

$$(t, x, u, p, q) \in [0, T] \times \mathbb{R}^n \times U \times \mathbb{R}^n \times \mathbb{R}^{n \times d}$$

is concave for all $t \in [0, T]$ *almost surely and*

$$H(t, x^*(t), u^*(t), p(t), q(t)) = \max_{u \in U} H(t, x^*(t), u, p(t), q(t)), \quad P\text{-a.s.}$$

holds. Then $(x^*(\cdot), u^*(\cdot))$ *is an optimal pair of* (2.16) *[22].*

Stochastic Hamiltonian system

Note that the partial differentials of the Hamiltonian satisfy $b(t, x, u) = H_p(t, x, u, p, q)$ and $\sigma(t, x) = H_q(t, x, p, q)$, then we have :

$$\begin{cases} dx(t) = H_p(t, x(t), u(t), p, q))dt + H_q(t, x, p, q)dW_t, \\ dp(t) = -H_x(t, x(t), u(t), p(t), q(t))dt + q(t)dW_t, \qquad t \in [0, T], \\ x(0) = x_0, \quad (T) = -h_x(x(T)), \\ H(t, x(t), u(t), p(t), q(t)) = \max_{u \in U} H(t, x(t), u, p(t), q(t)), \end{cases} \tag{2.20}$$

which is called a *(extended) stochastic Hamiltonian system* and it is also called a forward-backward stochastic differential equation (FBSDE, for short).Therefore,It is seen from the following theorem that optimal control theory can be used to solve stochastic Hamiltonian systems.

Theorem 2.5.3 *[22] Let (S0)-(S3) hold. Let Problem (P) admit an optimal pair* $(x^*(\cdot), u^*(\cdot))$*. Then the optimal 4-tuple* $(x^*(\cdot), u^*(\cdot), p(\cdot), q(\cdot))$ *of Problem (P) solves the stochastic Hamiltonian system.*

Now we state the Pontryagin-type maximum principle for optimal stochastic control. At first glance, one might naturally expect that a stochastic maximum principle should maximize the Hamiltonian H. However, this is *not* true when the diffusion coefficient σ depends on the control. The following example illustrates this point:

$$\begin{cases} dx(t) = u(t)dW(t), \quad t \in [0, 1], \\ x(0) = 0, \end{cases}$$

with the control domain $U = [-1, 1]$ and the cost functional:

$$J(u(\cdot)) = \mathbb{E}\left\{\int_0^1 \left|x(t)^2 - \frac{1}{2}u(t)^2\right| dt + x(1)^2\right\}.$$

Substituting $x(t) = \int_0^t u(s)dW(s)$ into the cost functional yields:

$$J = \mathbb{E}\int_0^1 \left(\frac{2}{3} - t\right) u(t)^2 dt.$$

The optimal control is $\overline{u}(t) \equiv 0$ with the optimal state trajectory $\overline{x}(t) \equiv 0$. However, the corresponding Hamiltonian is:

$$H(t, \overline{x}(t), u, p(t), q(t)) = \frac{1}{2}u^2 + q(t)u.$$

This convex function in u does not attain its maximum at $\overline{u}(t) = 0$ for any t.

To formulate the correct stochastic maximum principle, we must add a risk adjustment term to the Hamiltonian that accounts for the diffusion coefficient and reflects the controller's risk attitude. This leads to **generalized Hamiltonian** [22].

Chapter 3

Deterministic and Stochastic Optimal Control analysis for Influenza with media awareness programs under treatment and vaccination

Major influenza include the 1918 Spanish flu the mother of all pandemics [25], 1957 Asian flu, 1968 Hong Kong flu, and the 2009 H1N1 pandemic influenza spreads primarily through airborne droplets. When an infected person coughs, sneezes, or talks, tiny droplets containing the virus can be inhaled by others.

Common symptoms include fever, cough, sore throat, body aches, fatigue, and sometimes severe respiratory complications. Annual flu vaccines help prevent infection and reduce severity, especially for high-risk populations.

Antiviral medications like oseltamivir (Tamiflu) and supportive care (hydration, fever management) can help reduce symptoms and complications [25].

In this chapter, we analyze the comparative performance of deterministic and stochastic optimal control approaches for epidemic management using the Susceptible-Infected-Recovered (SIR) model with media awareness, the inclusion treatment and vaccination as controls [4] [33] [34] [46] [51].

We formulate and solve the optimal control problems using the Pontryagin's Maximum Principle (PMP) for the deterministic model and the Stochastic Maximum Principle (SMP) for the stochastic model.

3.1 Deterministic Model Formulation

We assume :

- **Recovered individuals** gain **a strong immunity** after infection.
- A **media awareness program** is incorporated into **deterministic and stochastic optimal control** analysis.
- In the model, the effective contact rate is defined as:

$$P(I) = \beta - \pi f(I),$$

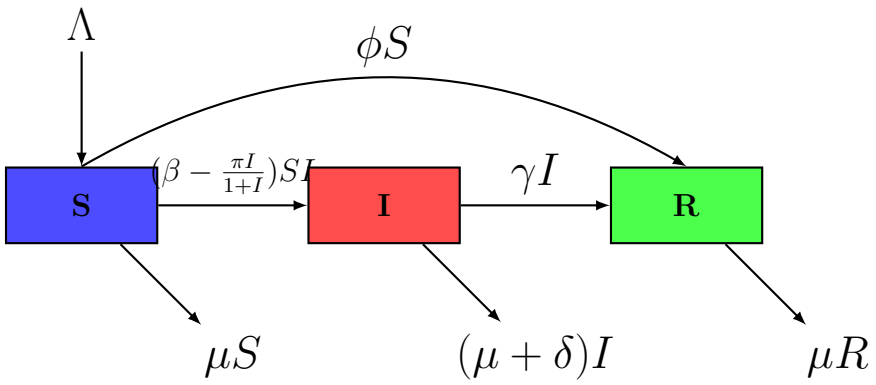
where:

- β Contact rate of susceptibles with infectives.
- π Rate of awareness. It is also assumed that $\beta \geq \pi$.
- $f(I)$ is the media coverage function, which is given by

$$f(I) = \frac{I}{1+I}$$

- As I increases, $f(I)$ increases, which causes $P(I)$ to decrease, reflecting the impact of media in reducing transmission through increased public awareness.

Then SIR principal scheme diagram with media awareness program :



Parameters	Explanation	values
Λ	Constant rate at which number of **susceptibles** increases continuously.	40
β	Contact rate of susceptible with infectives.	0.0003
π	Rate of awareness.	0.0002
ϕ	Rate of vaccination of susceptibles.	0.65
γ	Recovery rate of infectives.	0.045
δ	Disease-induced death rate.	0.02
μ	Natural death rate from each class.	0.05
A_1	positive constants	100
A_2	positive constant	150
A_3	positive constant	100
S(0)	initial susceptibles	100000
I(0)	initial infectived	100
R(0)	initial Recovered	0
ϵ	intensity of noise	0.005.

Table 3.1: Parameters values

Remark 3.1.1: we assume that the vaccinated individuals gain strong immunity as the recovered individuals from the epidemic. From a mathematical perspective, introducing a separate vaccinated compartment would increase the model's complexity by adding additional equations and parameters (e.g., vaccine efficacy, waning immunity, partial protection)[47][45]. While such extensions are necessary in certain contexts especially when studying vaccine hesitancy, varying efficacy, or multi-dose regimens in many standard models, the primary goal is to capture the overall dynamics efficiently [42]. Hence, placing vaccinated individuals in offers a simplified yet biologically meaningful representation of their immune status, facilitating both analysis and simulation.

The model equations are given by this system :

$$\begin{cases} \frac{dS}{dt} &= \Lambda - \left(\beta - \frac{\pi I}{1+I}\right) SI - (\phi + \mu)S \\ \frac{dI}{dt} &= \left(\beta - \frac{\pi I}{1+I}\right) SI - (\gamma + \delta + \mu)I \\ \frac{dR}{dt} &= \phi S + \gamma I - \mu R \end{cases} \tag{3.1}$$

Where $S(0) > 0$, $I(0) > 0$, $R(0) \geq 0$, and the total variable population at time t is given by:

$$N = S + I + R.$$

3.1.1 The effect of media awareness program

In order to show the effect of media awareness program we will solve numerically the system of ODEs of the SIR model (by using a Runge-Kutta method).

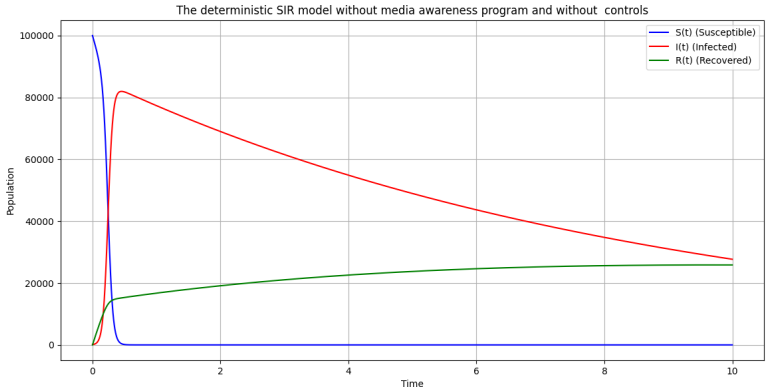


Figure 3.1: The deterministic SIR model without media awareness program ($\pi = 0$) (without controls)

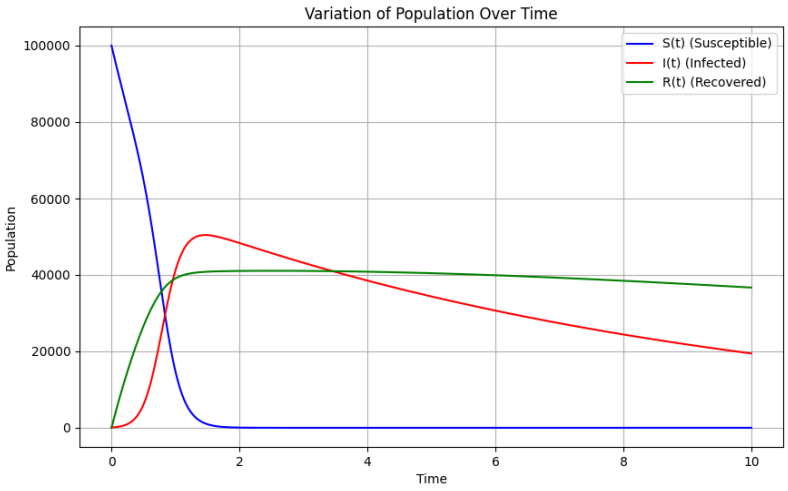


Figure 3.2: The deterministic SIR model with media awareness program (without controls)

Interpretation

The figure 3.1 shows the simulation of a deterministic SIR model without media awareness and without controls. It illustrates the typical epidemic curve, where the infected population increasing to a peak 80000, before gradually decreasing. The sharp initial peak indicates a rapid spread due to the absence of interventions.
The figure 3.2 shows the variation of the three categories susceptible, infectious, recovered over days. When the number of susceptible gets drop down, the number of infection increases to a peak 50000 even though there are still no control, because people more cautious and attentive, leading them to take measures such as social distancing or quarantine after becoming aware of the rise in infections and the danger of the epidemic through the media awareness.

We conclude that media awareness is essential in combating the disease. However, this alone is not enough to eliminate the epidemic. So assume the following controls have been used:

1. Let $u_1 \in [0, u_{1\max}]$ be the control representing the **strengthening effort made on the vaccination program**.

2. Let $u_2 \in [0, u_{2\max}]$ be the control representing the **controlling effort that alters infection cases receiving treatment per unit time**.

The controlled system of $ODEs$ is given as follow

$$\begin{cases} \frac{dS}{dt} &= \Lambda - \left(\beta - \frac{\pi I(t)}{1+I(t)}\right) S(t)I(t) - (1+u_1)\phi S(t) - \mu S(t) \\ \frac{dI}{dt} &= \left(\beta - \frac{\pi I(t)}{1+I(t)}\right) S(t)I(t) - (1+u_2)\gamma I(t) - \delta I(t) - \mu I(t) \\ \frac{dR}{dt} &= (1+u_1)\phi S(t) + (1+u_2)\gamma I(t) - \mu R(t) \end{cases} \tag{3.2}$$

where $S(0) > 0$, $I(0) > 0$, $R(0) \geq 0$.
To show the existence of the feasible set of system (3.2) which attracts all solutions initiating in the interior of the **positive orthant**. that is, **all solutions are uniformly bounded in a proper subset** $\Omega \subset \mathbb{R}^3$.
Using the fact that $N = S(t) + I(t) + R(t)$, the system reduces (3.2) to the following system

$$\frac{dN}{dt} = \Lambda - \mu N(t) - \delta I(t) \tag{3.3}$$

$$\leq \Lambda - \mu N(t) \tag{3.4}$$

Rewriting (3.3):

$$\frac{dN}{dt} + \mu N = \Lambda$$

This is a linear first-order differential equation. The integrating factor is $e^{\mu t}$. Multiplying both sides by $e^{\mu t}$:

$$e^{\mu t}\frac{dN}{dt} + \mu e^{\mu t}N = \Lambda e^{\mu t}$$

$$\frac{d}{dt}\left(Ne^{\mu t}\right) = \Lambda e^{\mu t}$$

$$Ne^{\mu t} = \int \Lambda e^{\mu t} dt$$

$$Ne^{\mu t} = \frac{\Lambda}{\mu}e^{\mu t} + C$$

Then

$$N = \frac{\Lambda}{\mu} + Ce^{-\mu t}$$

Using the initial condition $N(0) = N_0$,

$$N_0 = \frac{\Lambda}{\mu} + C$$

Thus,

$$C = N_0 - \frac{\Lambda}{\mu}$$

$$N(t) = \left(N_0 - \frac{\Lambda}{\mu}\right)e^{-\mu t} + \frac{\Lambda}{\mu}$$

We have

$$N(t) \leq N(0)e^{-\mu t} + \frac{\Lambda}{\mu}(1 - e^{-\mu t}) \tag{3.5}$$

where $N(0)$ is the sum of initial values $S(0), I(0), R(0)$.

$$\lim_{t\to\infty} N(t) = \frac{\Lambda}{\mu}$$

Thus, $\frac{\Lambda}{\mu}$ is the upper bound of N. Therefore, the region of attraction is given by the set:

$$\Omega = \left\{(S, I, R) \in \mathbb{R}^3 : 0 \leq S + I + R \leq \frac{\Lambda}{\mu}\right\} \tag{3.6}$$

and attracts all solutions initiating in the interior of the **positive orthant**

Theorem 3.1.1 *[14] The solutions S, I, R of the system* (3.2) *with initial values* $S(0) > 0, I > 0$*, and* $R \geq 0$ *in the feasible domain are positive in* Ω *for all time* $t > 0$*, because the model represents population of human being.*

proof: Given the differential equation for $S(t)$:

$$\dot{S(t)} = \Lambda - \left(\beta - \frac{\pi I(t)}{1+I(t)}\right) S(t)I(t) - ((1+u_1(t))\phi + \mu)\, S$$

$$\frac{dS}{dt} + \left[((1+u_1)\phi+\mu) + \left(\beta - \frac{\pi I}{1+I}\right) I\right] S = \Lambda$$

The integrating factor is:

$$IF = e^{-\int\left[((1+u_1)\phi+\mu)+\left(\beta-\frac{\pi I}{1+I}\right)I\right]dt}$$

Multiplying by the integrating factor:

$$\frac{d}{dt}\left(S \cdot IF\right) = \Lambda \cdot IF$$

$$S \cdot IF = \int \Lambda \cdot IF\, dt + C$$

$$S(t) = e^{\int\left[((1+u_1)\phi+\mu)+\left(\beta-\frac{\pi I(t)}{1+I(t)}\right)I(t)\right]dt}\left(\int \Lambda e^{-\int\left[((1+u_1)\phi+\mu)+\left(\beta-\frac{\pi I(t)}{1+I(t)}\right)I(t)\right]dt}dt + C\right)$$

$$= e^{-\left[((1+u_1)\phi+\mu)+\left(\beta-\frac{\pi I(t)}{1+I(t)}\right)I(t)\right]t}\left(\Lambda\int e^{\left[((1+u_1)\phi+\mu)+\left(\beta-\frac{\pi I(t)}{1+I(t)}\right)I(t)\right]t}dt + C\right)$$

$$= e^{-\left[((1+u_1)\phi+\mu)+\left(\beta-\frac{\pi I(t)}{1+I(t)}\right)I(t)\right]t}\left(\Lambda\frac{e^{\left[((1+u_1)\phi+\mu)+\left(\beta-\frac{\pi I(t)}{1+I(t)}\right)I(t)\right]t}}{\left[((1+u_1)\phi+\mu)+\left(\beta-\frac{\pi I(t)}{1+I(t)}\right)I(t)\right]} + C\right)$$

$$= \frac{\Lambda}{\left[((1+u_1)\phi+\mu)+\left(\beta-\frac{\pi I(t)}{1+I(t)}\right)I(t)\right]} + Ce^{-\left[((1+u_1)\phi+\mu)+\left(\beta-\frac{\pi I(t)}{1+I(t)}\right)I(t)\right]t}$$

$$\lim_{t\to 0} S(t) = \frac{\Lambda}{\left[((1+u_1)\phi+\mu)+\left(\beta-\frac{\pi I(t)}{1+I(t)}\right)I(t)\right]} + C > 0$$

Following the same method, we can easily verify that $I(t) > 0$ and $R(t) > 0$ for all $t > 0$.

Equilibrium points and stability :

The equilibria for the system (3.2) is obtained by equating each of the derivatives to zero. So, it is found that the model system (3.2) has **two non-negative equilibria**, i.e., **disease-free equilibrium (DFE)** when $I = 0$, and the EE endemic equilibrium when $I \neq 0$ [16], [4], [31], [37].

Disease-Free Equilibrium (DFE)

1. **Solve for** S **and** R at equilibrium ($\frac{dS}{dt} = 0$ and $\frac{dR}{dt} = 0$).

Solve for S^*. Setting $\frac{dS}{dt} = 0$:

$$\Lambda - (\phi(1+u_1)\mu)S = 0 \tag{3.7}$$

$$S^* = \frac{\Lambda}{\phi(1+u_1)+\mu} \tag{3.8}$$

For R^*, setting $\frac{dR}{dt} = 0$:

$$\phi(1+u_1)S + \gamma(1+u_2)I - \mu R = 0 \tag{3.9}$$

Substituting $I = 0$:

$$\phi(1+u_1)S - \mu R = 0 \tag{3.10}$$

Solving for R^*:

$$R^* = \frac{\phi(1+u_1)}{\mu}S^* = \frac{\phi(1+u_1)}{\mu} \cdot \frac{\Lambda}{\phi(1+u_2)+\mu} \tag{3.11}$$

Thus, the **disease-free equilibrium (DFE)** is:

$$E_0 = (S^*(t), I^*(t), R^*(t)) = \left(\frac{\Lambda}{\phi(1+u_1)+\mu}, 0, \frac{\phi(1+u_1)}{\mu} \cdot \frac{\Lambda}{\phi(1+u_1)+\mu}\right) \tag{3.12}$$

This point represents the state where the infection has been eradicated, and only susceptibles and recovered individuals remain.

Basic reproduction number R_0

To calculate the basic reproduction number R_0 at the Disease-Free Equilibrium (DFE), we use the **next-generation matrix matrix**.

$$R_0 = \left[\frac{\partial F_i(x_0)}{\partial x_j}\right]\left[\frac{\partial V_i(x_0)}{\partial x_j}\right]^{-1} = \rho(FV^{-1})$$

From the infection equation, we extract:

$$\frac{dI}{dt} = \left(\beta - \frac{\pi I(t)}{1+I(t)}\right)S(t)I(t) - ((1+u_2)\gamma + \delta + \mu)I(t)$$

$$(S^*(t), I^*(t), R^*(t)) = \left(\frac{\Lambda}{\phi(1+u_1)+\mu}, 0, \frac{\phi\Lambda}{\mu(\phi+\mu)}\right)$$

$$F_i = \left(\beta - \frac{\pi I(t)}{1+I(t)}\right)I(t)S(t)$$

At **DFE**,

$$F = \left[\frac{\partial F_i}{\partial I(t)}\right]_{DFE} = \beta\frac{\Lambda}{\phi(1+u_1)+\mu}$$
$$V_i = ((1+u_2)\gamma + \delta + \mu)I(t)$$

$$V = \left[\frac{\partial V_i}{\partial I(t)}\right]_{DFE} = (1+u_2)\gamma + \delta + \mu$$

$$R_0 = \frac{\beta\Lambda}{(\phi(1+u_1)+\mu)((1+u_2)\gamma+\delta+\mu)}$$

Now we going to analyze the stability of the DFE. To define **local stability** of E_0, the **Jacobian matrix** of model system (3.2) is evaluated at DFE.

$$J = \begin{bmatrix} -\left(\beta - \frac{\pi I(t)}{1+I(t)}\right) I(t) - \mu & -S(t)\left(\beta - \frac{\pi I(t)}{1+I(t)} - \frac{\pi I(t)}{(1+I(t))^2}\right) & 0 \\ \left(\beta - \frac{\pi I(t)}{1+I(t)}\right) I(t) & S\left(\beta - \frac{\pi I(t)}{1+I(t)} - \frac{\pi I(t)}{(1+I(t))^2}\right) - (\gamma(1+u_2)+\delta+\mu) & 0 \\ \phi(1+u_1) & \gamma(1+u_2) & -\mu \end{bmatrix}$$

$$J_{DFE} = \begin{bmatrix} -\mu & -\frac{\Lambda}{\phi(1+u_1)+\mu}\beta & 0 \\ 0 & \frac{\Lambda}{\phi(1+u_1)+\mu} - (\gamma(1+u_2)+\delta+\mu) & 0 \\ \phi(1+u_1) & \gamma(1+u_2) & -\mu \end{bmatrix}$$

The eigenvalues of the Jacobian matrix are

$$\lambda 1 = -\mu,\ \lambda 2 = \frac{\beta\Lambda}{\mu+\phi} - (\gamma(1+u_2)+\delta+\mu),\ \lambda 3 = -\mu.$$

the eigen values are negative Except $\lambda 2$, thus if

$$\frac{\beta\Lambda}{(\phi(1+u_1)+\mu)(\gamma(1+u_2)+\delta+\mu)} < 0$$

Then E_0 is locally asymptotically stable.

Remark 3.1.2: By (Corollary of Gershgorin Circle Theorem) The local stability can be established without the need to calculate the eigenvalues.

Remark 3.1.3: [18] The case $R_0 = 1$ is a critical threshold point where the disease free equilibrium E_0 loses its asymptotic stability and simply becomes (neutrally) stable. Moreover, it becomes unstable immediately $R_0 > 1$ and this will lead to the existence of a stable endemic equilibrium (EE). Note that $R_0 = 1$ can literarily be viewed as a transcritical bifurcation point where stability is exchanged between E_0 and Endemic Equilibrium (EE).

Endemic Equilibrium

We consider the system at equilibrium for $I(t) \neq 0$ ($\frac{dS}{dt} = 0$, $\frac{dI}{dt} = 0$, $\frac{dR}{dt} = 0$). We use the fact $S + I + R = N$, we have:

$$\begin{cases} \left(\beta - \frac{\pi I(t)}{1+I(t)}\right) S(t)I(t) - (\gamma(1+u_2)+\delta+\mu)I(t) = 0 \\ \phi(1+u_1)S(t) + \gamma(1+u_2)I(t) - \mu R = 0 \\ \Lambda - \mu N - \delta I(t) = 0 \end{cases} \tag{3.13}$$

and

$$S = N - I - R. \tag{3.14}$$

By solving the system we find

$$N^* = \frac{\Lambda - \delta I(t)}{\mu}$$

$$R^*(t) = \frac{\phi(1+u_1)(\Lambda - \delta I(t) - \mu I(t)) + (1+u_2)\gamma\mu I(t)}{\mu(\phi(1+u_1)+\mu)}$$

Then

$$EE = (I(t), \frac{\phi(1+u_1)(\Lambda - \delta I(t) - \mu I(t)) + (1+u_2)\gamma\mu I(t)}{\mu(\phi(1+u_1)+\mu)}, \frac{\Lambda - \delta I(t)}{\mu})$$

For the stability of endemic Equilibrium as same kind of DFE [37](See also [24, 4]).

Let the basic reproduction number R_0

$$R_0 = \frac{\beta\Lambda}{(\phi(1+u_1)+\mu)((1+u_2)\gamma + \delta + \mu)}$$

By definition $R_0 < 1$ the disease cannot invade the population and the infection will die out over a period of time. When $R_0 > 1$ The disease will invade

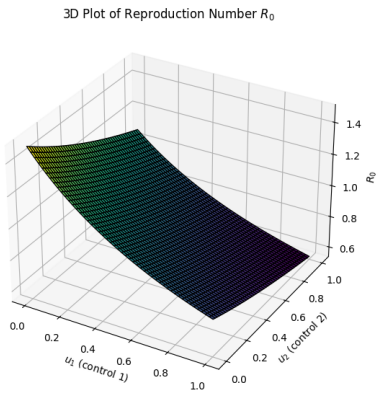


Figure 3.3: the basic reproduction number (R_0).

It is clear that the greater of controls u_1 and u_2, the lower the reproduction number. That mean increasing control effectively reduces the spread of the disease, demonstrating their role in reducing infections and eventually eliminating the disease. However, excessive control can have negative consequences moreover it is costly, so public health policy makers seek to minimize the infection with a minimum cost. Therefore, in the next section, we focus on finding the successful intervention strategy which leads to decrease the number of infections with a minimum cost.

3.2 Optimal Control Analysis

Let's recall the controlled system :

$$\begin{cases} \frac{dS}{dt} &= \Lambda - \left(\beta - \frac{\pi I(t)}{1+I(t)}\right) S(t)I(t) - (1+u_1(t))\phi S(t) - \mu S(t) \\ \frac{dI}{dt} &= \left(\beta - \frac{\pi I(t)}{1+I(t)}\right) S(t)I(t) - (1+u_2(t))\gamma I(t) - \delta I(t) - \mu I(t) \\ \frac{dR}{dt} &= (1+u_1(t))\phi S(t) + (1+u_2(t))\gamma I(t) - \mu R(t) \end{cases} \tag{3.15}$$

The objective functional is

$$J(u_1, u_2) = \int_0^T \left(A_1 I(t) + \frac{A_2 u_1^2(t)}{2} + \frac{A_3 u_2^2(t)}{2} \right) dt, \tag{3.16}$$

Where set of controls is defined as follows

$$U = \{u_i : 0 \leq u_i \leq u_{imax} \leq 1,\ i = 1, 2; u_i \text{ is Lebesgue measurable}\}. \tag{3.17}$$

$$J(u_1^*, u_2^*) = \inf\{J(u_1, u_2) | u_1, u_2 \in U\} \tag{3.18}$$

Where A_1 is the weight of infection, A_2 and A_3 are the relative weights assigned to the cost of vaccination and treatment respectively. The control u_1 represent the **strengthening effort made on the vaccination program per unit time** while the control u_2 represent the **controlling effort that alters infection cases per unit time**. Thus, u_1 and u_2 lie between 0 and 1 whereas u_{1max} and u_{2max} will depend on the amount of resources available to implement each of the control measures.

The weights A_2 and A_3 will depend on the relative importance of each of the control measures in mitigating the spread of the disease. Thus, the terms $A_2 u_1^2$ and $A_3 u_2^2$ describe the costs associated with vaccination and treatment respectively. The vaccination cost could include the cost of the vaccine, the vaccine storage cost, other related overheads, etc. The treatment cost could include the cost of the medical tests and diagnosis, drug cost, hospitalization cost, etc[16] [47].

Our goal is to characterize an optimal control $(u_1^*, u_2^*) \in U$ which minimizes the cost of the vaccination and the cost of the treatment over the specified time interval.

3.2.1 Existence of optimal control

Theorem 3.2.1 *[4] There exists an optimal control $u^* = (u_1^*, u_2^*) \in U$ such that subject to the control system 3.15 with initial condition at $t = 0$:*

$$J(u_1^*, u_2^*) = \min_{(u_1, u_2) \in U} J(u_1, u_2). \tag{3.19}$$

Proof: (i) Convexity and closer of control set U : We have $U = [0,1]^2$ is closed by definition, further for two arbitrary points $y = (y_1, y_2)$ and $z = (z_1, z_2) \in U$ it is following by definition of convex set

$$(\lambda y + (1-\lambda)z) \in U, \ \forall \lambda \in [0,1]$$

Then U is convex.

(ii) Boundedness of the state system by a linear function in the state and control variables:

$$\|b(t,x,v)\| \leq \|g(t,x)\| + \|h(t,x)\|\|v\| \leq a_1 + a_2\|v\|$$

Let

$$v = (u_1, u_2)$$

and

$$x = (S(t), I(t), R(t))$$

we have :

$$\mathbf{b}(t,\mathbf{x},v) = \begin{bmatrix} \Lambda - \left(\beta - \frac{\pi I(t)}{1+I(t)}\right) S(t)I(t) - ((1+u_1(t))\phi + \mu)\, S(t) \\ \left(\beta - \frac{\pi I(t)}{1+I(t)}\right) S(t)I(t) - ((1+u_2(t))\gamma + \delta + \mu)\, I(t) \\ (1+u_1(t))\phi S(t) - (1+u_2(t))\gamma I(t) - \mu R(t) \end{bmatrix}$$

$$b(t,x,v) = g(t,x) + h(t,x)v$$

where

$$g(t,x) = \begin{bmatrix} \Lambda - \left(\beta - \frac{\pi I(t)}{1+I(t)}\right) S(t)I(t) - \mu S(t) - \phi S(t) \\ \left(\beta - \frac{\pi I(t)}{1+I(t)}\right) S(t)I(t) - (\gamma + \delta + \mu)I(t) \\ \phi S(t) - \gamma I(t) - \mu R(t) \end{bmatrix}$$

$$h(t,x) = \begin{bmatrix} -\phi S & 0 \\ 0 & -\gamma I(t) \\ \phi S & \gamma I(t) \end{bmatrix}$$

$$v = \begin{bmatrix} u_1(t) \\ u_2(t) \end{bmatrix}$$

Compute $\|g(t,x)\|$:

$$\|g(t,x)\| = \sqrt{g_1^2 + g_2^2 + g_3^2}$$

where:

$$g_1 = \Lambda - \left(\beta - \frac{\pi I(t)}{1 + I(t)}\right) S(t)I(t) - \mu S(t) - \phi S(t)$$

$$g_2 = \left(\beta - \frac{\pi I(t)}{1 + I(t)}\right) S(t)I(t) - (\gamma + \delta + \mu)I(t)$$

$$g_3 = \phi S(t) - \gamma I(t) - \mu R(t)$$

Thus,

$$a_1 = \|g(t, x)\|$$

Compute $\|h(t, x)\|$

$$h(t, x) = \begin{bmatrix} -\phi S(t) & 0 \\ 0 & -\gamma I(t) \\ \phi S & \gamma I(t) \end{bmatrix}$$

Approximating the Frobenius norm:

$$\|h(t, x)\| = \sqrt{(-\phi S(t))^2 + (-\gamma I(t))^2 + (\phi S(t))^2 + (\gamma I(t))^2}$$
$$= \sqrt{2\phi^2 S(t)^2 + 2\gamma^2 I(t)^2}$$

$$= \sqrt{2}\sqrt{\phi^2 S(t)^2 + \gamma^2 I(t)^2}$$

Compute $\|v\|$

$$v = \begin{bmatrix} u_1(t) \\ u_2(t) \end{bmatrix}$$

$$\|v\| = \sqrt{u_1^2 + u_2^2}$$

Compute Final Bound

$$\|b(t, x, v)\| \leq \|g(t, x)\| + \|h(t, x)\|\|v\|$$

Let

$$a_2 = \sqrt{2}\sqrt{\phi^2 S^2 + \gamma^2 I(t)^2}$$

Replacing each state variable by its upper bound $S = I = R = \frac{\Lambda}{\mu}$. So:

$$a_2 = \sqrt{2}\frac{\Lambda}{\mu}\sqrt{\phi^2 + \gamma^2}.$$

Then

$$\|b(t,x,v)\| \leq a_1 + a_2\|v\|.$$

(iii) Convexity of the integrand of the cost functional with respect to the control: Let $f(I,u_1,u_2) = A_1I + A_2\frac{u_1^2(t)}{2} + A_3\frac{u_2^2(t)}{2}$. It is clear that it is convex on the control set U.

(iv) We can easily see that there exist a constant $\eta > 1$ and positive numbers θ_1, θ_2 such that

$$f(I,u_1,u_2) = A_1I + A_2\frac{u_1^2(t)}{2} + A_3\frac{u_2^2(t)}{2} \geq \frac{1}{2}(A_2u_1^2(t) + A_3u_2^2(t))$$

$$\geq \theta_1(u_1^2(t) + u_2^2(t))^{\frac{\eta}{2}} - \theta_2$$

where

$$\theta_1 = \frac{1}{2}\min\{A_2, A_3\}, \theta_2 > 0, \quad \text{and} \quad \eta = 2.$$

Characterization of the optimal control :

Our goal is to minimize the cost function J :

$$J(u_1,u_2) = \int_0^T \left(A_1I(t) + \frac{A_2u_1^2(t)}{2} + \frac{A_3u_2^2(t)}{2}\right)dt,$$

To do that we have to find the optimal solution of ordinary differential equations for the adjoint variables by using the Pontryagin's Maximum Principle(PMP) under boundary conditions and characterization of an optimal control with respect to the controls $u_1(t), u_2(t)$ [4], [30], [5], [4]. To do this, we define the Hamiltonian for the control problem as follows:

$H = A_1I(t) + A_2\frac{u_1^2(t)}{2} + A_3\frac{u_2^2(t)}{2} + \lambda_1\dot{S} + \lambda_2\dot{I} + \lambda_3\dot{R}$

$= \left(A_1I(t) + \frac{A_2u_1^2(t)}{2} + \frac{A_3u_2^2(t)}{2}\right) + \lambda_1\left(\Lambda - \left(\beta - \frac{\pi I(t)}{1+I(t)}\right)S(t)I(t) - ((1+u_1(t))\phi + \mu)\,S(t)\right)$

$+\lambda_2\left(\left(\beta - \frac{\pi I(t)}{1+I(t)}\right)S(t)I(t) - (1+u_2(t))\gamma I(t) - \delta I(t) - \mu I(t)\right)$

$+\lambda_3\left((1+u_1(t))\phi S(t) + (1+u_2(t))\gamma I(t) - \mu R(t)\right)$

Let (S^*, I^*, R^*) be the optimal solutions of states with associated optimal variables (u_1^*, u_2^*) for the optimal control problem. Then there exist adjoint variables $\lambda = (\lambda_1, \lambda_2, \lambda_3)$ satisfying :

$$\frac{d\lambda}{dt} = \dot{\lambda} = -\frac{\partial H(t,u,\lambda)}{\partial x}$$

$$\dot{\lambda_1} = -\frac{dH}{dS} = -\left(-\lambda_1\left(\beta - \frac{\pi I(t)}{1+I(t)}\right)I(t) - \lambda_1((1+u_1(t))\phi + \mu) + \lambda_3(1+u_1(t))\phi + \lambda_2\left(\beta - \frac{\pi I(t)}{1+I(t)}\right)I(t)\right)$$

$$= \lambda_1\left(\beta - \frac{\pi I(t)}{1+I(t)}\right)I(t) + \lambda_1((1+u_1(t))\phi + \mu) - \lambda_3(1+u_1(t))\phi - \lambda_2\left(\beta - \frac{\pi I(t)}{1+I(t)}\right)I(t)$$

$\dot{\lambda_1} = (\lambda_1 - \lambda_2)\left(\beta - \frac{\pi I(t)}{1+I(t)}\right) I(t) + (\lambda_1 - \lambda_3)(1 + u_1(t))\phi + \lambda_1\mu$
$\dot{\lambda_2} = -\frac{dH}{dI} = -\left(A_1 - \lambda_1\left(\beta S(t) - \frac{\pi I(t)S(t)(2+I(t))}{(1+I(t))^2}\right)\right) + \lambda_2\left(\beta S(t) - \frac{\pi I(t)S(2+I(t))}{(1+I(t))^2}\right) - \lambda_2((1+u_2(t))\gamma + \delta + \mu) + \lambda_3(1 + u_2(t))\gamma$

$$\dot{\lambda_2} = -A_1 + (\lambda_1 - \lambda_2)\left(\beta S(t) - \frac{\pi I(t)S(2 + I(t))}{(1 + I(t))^2}\right) + (\lambda_2 - \lambda_3)(1 + u_2(t))\gamma + \lambda_2(\delta + \mu)$$

$$\dot{\lambda_3} = -\frac{dH}{dR} = (-\lambda_3\mu) = \lambda_3\mu$$

Then the adjoint system is

$$\begin{cases} \dot{\lambda_1} = (\lambda_1 - \lambda_2)\left(\beta - \frac{\pi I(t)}{1+I(t)}\right) I(t) + (\lambda_1 - \lambda_3)(1 + u_1(t))\phi + \lambda_1\mu \\ \dot{\lambda_2} = -A_1 + (\lambda_1 - \lambda_2)\left(\beta S(t) - \frac{\pi I(t)S(t)(2+I(t))}{(1+I(t))^2}\right) + (\lambda_2 - \lambda_3)(1 + u_2(t))\gamma + \lambda_2(\delta + \mu) \\ \dot{\lambda_3} = \lambda_3\mu \end{cases}$$

Minimizing H with respect u_1 and u_2

$$\frac{\partial H}{\partial u_1} = A_2 u_1(t) - \lambda_1\phi S(t) - \lambda_3\phi S(t) = 0$$

$$\frac{\partial H}{\partial u_2} = A_3 u_2(t) - \lambda_2\gamma I(t) + \lambda_3\gamma I(t) = 0$$

Then

$$u_1^*(t) = \frac{(\lambda_1 - \lambda_3)\phi S(t)}{A_2}$$
$$u_2^*(t) = \frac{(\lambda_2 - \lambda_3)\gamma I(t)}{A_3}$$

Where (λ_1, λ_2, λ_3) are marginal costs or multipliers from the state and adjoint equations. If $\lambda_1 > \lambda_3$, vaccinating susceptibles is economically valuable by reduces future infections. And if $\lambda_2 > \lambda_3$, treating infections is economically valuable by reduces deaths and hospitalizations. [16][45].

The transversality conditions (terminal condition) are $\lambda_i(T) = 0, i = 1, 2, 3$ because the cost function J is independent on the state at the final time. So we concluded:

$$u_1^*(t) = max\{0, min\{1, \frac{(\lambda_1 - \lambda_3)\phi S(t)}{A_2}\}\}$$

$$u_2^*(t) = max\{0, min\{1, \frac{(\lambda_2 - \lambda_3)\gamma I(t)}{A_3}\}\}$$

To find out the optimal controls and states, it is solved numerically using forward backward sweep method (see Chapter 4).

3.3 Stochastic Model Formulation

We are designed the stochastic model by perturbation of parameter β and performed optimal control analysis using stochastic maximum principle to study stochastic optimal control problem of SIR type epidemic model with media awareness programs under controls u_1 and u_2 [4][5]. It is assumed that fluctuations in the environment which manifest themselves as fluctuation in the parameters β,

$$\beta \to \beta + \epsilon\eta(t)$$

where, $\eta(t) \sim N(0,1)$ represents Gaussian white noise, and ϵ is the intensity of the white noise and $dW(t) = \eta(t)dt$, where $W(t)$ is standard Brownian motion with $W(0) = 0$, and with intensity of white noise $\epsilon^2 > 0$. The stochastic version corresponding deterministic model is described by the following set of stochastic differential equations :

$$\begin{cases} dS = \left[\Lambda - \left(\beta - \frac{\pi I(t)}{1+I(t)}\right) S(t)I(t) - (\phi+\mu)S(t)\right] dt - \epsilon S(t)I(t)dW(t) \\ dI = \left[\left(\beta - \frac{\pi I(t)}{1+I(t)}\right) S(t)I(t) - (\gamma+\delta+\mu)I(t)\right] dt + \epsilon S(t)I(t)dW(t) \\ dR = \left[\phi S(t) + \gamma I(t) - \mu R(t)\right] dt \end{cases} \tag{3.20}$$

3.3.1 The effect of media awareness program

In order to show the effect of media awareness program in the stochastic version of SIR model without controls, we use Monte Carlo method to solve the system of $SDEs$ (3.20) where the number of Monte Carlo paths is $N = 1000$.

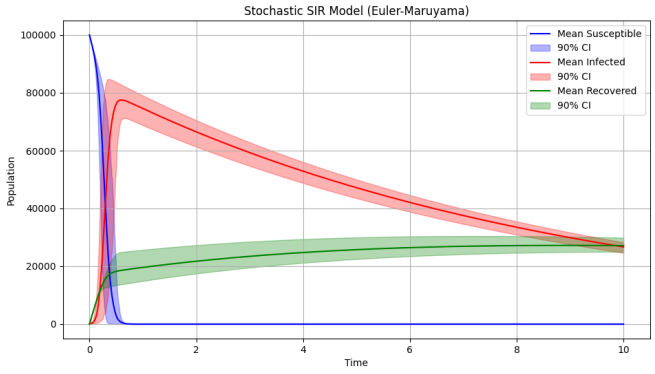


Figure 3.4: The stochastic SIR model without media awareness program($\pi = 0$) (without controls)

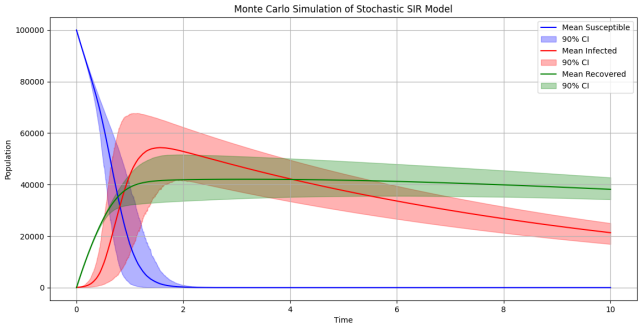


Figure 3.5: The Stochastic SIR model with media awareness program (without controls)

Interpretation :

The figure 3.4 shows the results of the stochastic SIR model without media awareness and without controls. Unlike the deterministic case, this model captures the randomness in disease transmission. The shaded regions represent the 95% confidence intervals (CI) for the susceptible S, infected I, and recovered R populations, reflecting the possible variation in the disease trajectory. This uncertainty highlights the unpredictable nature of real epidemics, where small random fluctuations can significantly impact the outbreak's dynamics.
From the figure 3.5 Initially, the number of susceptible individuals is high, but it decreases rapidly as the epidemic spreads the number of infected individuals rises rapidly at the beginning, creating an **epidemic peak** moreover the model reaches a **steady-state equilibrium**. As deterministic case the number of infected get drop down due to the effect of media awareness program. However it is not enough to eliminate epidemic. More efficient strategies could be applied.

3.3.2 Existence and uniqueness of positive solutions

Now we want to show that the solution of system (3.20) is positive and global by using Lyapunov analysis method [3], [45],[18], [32].

Theorem 3.3.1 *There is a unique solution* $S(t), I(t), R(t)$ *of system* (3.20) *on* $t \geq 0$ *for any initial value* $(S(0), I(0), R(0)) \in \mathbb{R}^3_+$, *and the solution will remain in* $\mathbb{R}^3_+$ *with probability* 1, *namely,* $(S(t), I(t), R(t)) \in \mathbb{R}^3_+$ *for all* $t \geq 0$ *almost surely.*

proof. Since the coefficients of the equation are locally Lipschitz continuous for any given initial value $(S(0), I(0), R(0)) \in \mathbb{R}^3_+$, there is a unique local solution $S(t), I(t), R(t)$ on $t \in [0, \tau_e)$, where

τ_e is the explosion time. To show that this solution is global, we need to show that $\tau_e = \infty$ a.s. Let $k_0 > 0$ be sufficiently large so that $S(0), I(0)$ and $R(0)$ all lie within the interval $[1/k_0, k_0]$. For each integer $k > k_0$, define the stopping time

$$\tau_k = \inf\left\{t \in [0, \tau_e) : \min(S(t), I(t), R(t)) \leq \frac{1}{k} \text{ or } \max(S(t), I(t), R(t)) \geq k\right\},$$

where, we set $\inf \emptyset = \infty$ (as usual $\emptyset$ denotes the empty set). According to the definition, τ_k is increasing as $k \to \infty$. Set $\tau_\infty = \lim_{k\to\infty} \tau_k$. We whence see $\tau_\infty \leq \tau_e$. If we can show that $\tau_\infty = \infty$ a.s., then $\tau_e = \infty$ and $(S(t), I(t), R(t)) \in \mathbb{R}^3_+$ a.s. for all $t > 0$.

In other words, to complete the proof all we need to show is that $\tau_\infty = \infty$ a.s. If this statement is false, then there exist a pair of constants $T > 0$ and $\epsilon_1 \in (0, 1)$ such that $\mathbb{P}(\tau_\infty \leq T) > \epsilon_1$. Hence there is an integer $k_1 > k_0$ such that

$$\mathbb{P}(\tau_k \leq T) \geq \epsilon_1, \quad \text{for all } k > k_1. \tag{3.21}$$

For $t \leq \tau_k$, it is observed for each k,

$$dN(t) = [\Lambda - \mu N(t) - \delta I]dt$$

$$\leq [\Lambda - \mu N(t)]dt.$$

Therefore,

$$N(0) = S(0) + I(0) + R(0)$$

$$(N(t)) \leq \begin{cases} \frac{\Lambda}{\mu}, & \text{if } N(0) \leq \Lambda/\mu, \\ N(0) & \text{if } N(0) > \Lambda/\mu \end{cases}$$

$$:= M$$

Define a C^2-function $V : \mathbb{R}^3_+ \to \mathbb{R}_+$ by

$$V(S, I, R) = (S - 1 - \log S) + (I - 1 - \log I) + (R - 1 - \log R)$$

$$dV(S, I, R) = \left(1 - \frac{1}{S}\right) dS + \frac{1}{2S^2}(dS)^2 + \left(1 - \frac{1}{I}\right) dI + \frac{1}{2I^2}(dI)^2 + \left(1 - \frac{1}{R}\right) dR + \frac{1}{2R^2}(dR)^2$$

$$= \left(1 - \tfrac{1}{S}\right)\left[(\Lambda - \left(\beta - \tfrac{\pi I}{(1+I)}\right) SI - (\phi + \mu)S)dt - \epsilon SI dW(t)\right]$$

$$+ (1 - \tfrac{1}{I})\left[(\beta - \tfrac{\pi I}{(1+I)})SI - (\gamma + \delta + \mu)I)dt + \epsilon SI dW(t)\right] + \left(1 - \tfrac{1}{R}\right)(\phi S + \gamma I - \mu R)dt + \tfrac{\epsilon^2 S^2 I^2}{2I^2}dt + \tfrac{\epsilon^2 S^2 I^2}{2S^2}dt$$

$$= \{\left(1 - \tfrac{1}{S}\right)(\Lambda - \left(\beta - \tfrac{\pi I}{(1+I)}\right) SI - (\phi + \mu)S) + (1 - \tfrac{1}{I})\left[(\beta - \tfrac{\pi I}{(1+I)})SI - (\gamma + \delta + \mu)I\right] + \left(1 - \tfrac{1}{R}\right)(\phi S + \gamma I - \mu R) + \tfrac{\epsilon^2 S^2}{2} + \tfrac{\epsilon^2 I^2}{2}\}dt + \epsilon(I - S)dW$$

$$= LV dt + \epsilon(I - S)dW(t).$$

Therefore, $LV = \left(1-\frac{1}{S}\right)(\Lambda - \left(\beta - \frac{\pi I}{(1+I)}\right)SI - (\phi+\mu)S) + (1-\frac{1}{I})\left[(\beta - \frac{\pi I}{(1+I)})SI - (\gamma+\delta+\mu)I\right] + \left(1-\frac{1}{R}\right)(\phi S + \gamma I - \mu R) + \frac{\epsilon^2 S^2}{2} + \frac{\epsilon^2 I^2}{2}$
$= \Lambda - \mu S - \mu R - \mu I - \frac{\Lambda}{S} + \left(\beta - \frac{\pi I}{(1+I)}\right)I + (\phi+\mu) - \left(\beta - \frac{\pi I}{(1+I)}\right)S + (\gamma+\delta+\mu) - \frac{\phi}{R}S - \frac{\phi}{R}I + \mu + \frac{1}{2}\epsilon^2(I^2+S^2) \leq \Lambda + 3\mu + \phi + \gamma + \delta + \beta M + \epsilon^2 M^2 := \tilde{K}$

$$E\left[W\{S(\tau_k \wedge \tau), I(\tau_k \wedge \tau), R(\tau_k \wedge \tau)\}\right]$$

$$\leq W\{S(0), I(0), R(0)\} + E\left[\int_0^{\tau_k \wedge \tau} \tilde{K} dt\right]$$

$$\leq W\{S(0), I(0), R(0)\} + \tilde{K}_\tau. \tag{3.22}$$

Set $\Omega_k = (\tau_k \wedge \tau)$, note that for every $\omega \in \Omega_k$, there is at least one of $S(\tau_k, \omega), I(\tau_k, \omega), R(\tau_k, \omega)$ that equals k or $1/k$ and hence

$W\{S(\tau_k), I(\tau_k), R(\tau_k)\}$ is no less than $k - 1 - \log k$ or $1/k - 1 - \log k$ consequently.

$$W\{S(\tau_k), I(\tau_k), R(\tau_k)\} \geq k - 1 - \log k \wedge 1/k - 1 - \log k.$$

It is then follows (3.21) and (3.22) that

$$W\{S(0), I(0), R(0)\} + \tilde{K}\tau \geq E\left[\mathbf{1}_{\Omega_k}(\omega) W\{S(\tau_k), I(\tau_k), R(\tau_k)\}\right]$$

$$\geq \varepsilon\left[k - 1 - \log k \wedge 1/k - 1 - \log k\right]$$

where $\mathbf{1}_{\Omega_k}(\omega)$ is the indicator function of Ω_k. Let $k \to \infty$ leads to the contradiction

$$\infty > W\{S(0), I(0), R(0)\} + \tilde{K}\tau = \infty.$$

So we must therefore have $\tau_\infty = \infty$, and Hence the proof is achieved.

Remark 3.3.1: *From theorem 3.3.1, for any initial value* $(S(0), I(0), R(0)) \in \mathbb{R}^3_+$, *there is a unique global solution* $(S(t), I(t), R(t)) \in \mathbb{R}^3_+$ *almost surely. Hence*

$$dN(t) \leq [\mu N(t)]\, dt, \text{ and } N(t) \leq \frac{\Lambda}{\mu} + e^{-\mu t} N(0).$$

So

$$\Omega^* = \{(S, I, R) \in \mathbb{R}^3_+ : 0 < S, 0 \leq I, 0 \leq R, S + I + R \leq \frac{\Lambda}{\mu}\}$$

is a positively invariant set of system (3.20), *which is similar to* Ω *of deterministic system. From now , we always assume that* $(S(t), I(t), R(t)) \in \Omega^*$.

3.4 Stochastic Optimal Control Analysis

The stochastic version corresponding to the deterministic controlled model is described by the following set of stochastic differential equations:

$$\begin{cases} dS = \left[\Lambda - \left(\beta - \frac{\pi I(t)}{1+I(t))}\right) S(t)I(t) - (\phi(1+u_1(t)) + \mu)S(t)\right] dt - \epsilon S(t)I(t)dW(t) \\ dI = \left[\left(\beta - \frac{\pi I(t)}{1+I(t)}\right) S(t)I(t) - ((1+u_2(t))\gamma + \delta + \mu)I(t)\right] dt + \epsilon S(t)I(t)dW(t) \\ dR = [\phi(1+u_1(t))S(t) + \gamma(1+u_2(t))I(t) - \mu R(t)]\, dt \end{cases} \tag{3.23}$$

Where $S(0) > 0$, $I(0) > 0$, $R(0) \geq 0$. We define the vectors:

$$x(t) = [S(t), I(t), R(t)] \text{ and } u(t) = [u_1(t), u_2(t)],$$

for $n \in \mathbb{N}$, $x_0 \in \mathbb{R}^n$, and an n-dimensional Brownian motions $W(t)$. Consider the general n-dimensional stochastic differential equation:

$$dx(t) = b(x(t), u(t))dt + \sigma(x(t))dW(t),$$

with the initial conditions

$$x(0) = [S(0), I(0), R(0)] = x_0,$$

where b and σ are vectors with components such that

$$b_1(x(t), u(t)) = \Lambda - \left(\beta - \frac{\pi I}{1+I)}\right) SI - (\phi(1+u_1) + \mu)S$$

$$b_2(x(t), u(t)) = \left(\beta - \frac{\pi I}{1+I}\right) SI - ((1+u_2)\gamma + \delta + \mu)I,$$

$$b_3(x(t), u(t)) = \phi(1+u_1)S + \gamma(1+u_2)I - \mu R,$$

$$\sigma_1(x(t), t) = -\epsilon SI,$$

$$\sigma_2(x(t), t) = \epsilon SI,$$

$$\sigma_3(x(t), t) = 0.$$

The cost functional is given as follows :

$$J(u) = \mathbb{E}\left\{\int_0^T (A_1 I + \frac{1}{2}A_2 u_1^2(t) + \frac{1}{2}A_3 u_2^2(t))dt\right\}$$

where A_1, A_2, A_3 are positive constants. Our goal is to find an optimal control $u^* = [u_1^*, u_2^*]$ such that

$$J(u^*) \leq J(u), \forall u_1, u_2 \in U,$$

where U is an admissible control set defined by

$$U = \{u_1, u_2 \mid 0 \leq u_1 \leq u_{\max}, 0 \leq u_2 \leq u_{\max}\},$$

The aim is to characterize an optimal control $(u_1^*, u_2^*) \in U$ which minimizes the cost of the vaccination and the cost of the treatment over the specified time interval and also minimizes the number of infectives at terminal time. In order to use the stochastic maximum principle, we define the Hamiltonian $H(x, u, p, q)$ by

$$\begin{aligned} H = &\left(A_1 I(t) + A_2\frac{u_1^2(t)}{2} + A_3\frac{u_2^2(t)}{2}\right) + p_1\left[\Lambda - \left(\beta - \frac{\pi I(t)}{1+I(t)}\right) I(t)S(t) - ((1+u_1(t))\phi + \mu)S(t)\right] \\ &+ p_2\left[\left(\beta - \frac{\pi I(t)}{1+I(t)}\right) I(t)S(t) - ((1+u_2(t))\gamma + \delta + \mu_2)\, I(t)\right] \\ &+ p_3\left[(1+u_1(t))\phi S(t) + (1+u_2(t))\gamma I(t) - \mu R(t)\right] \\ &- q_1\epsilon S(t)I(t) + q_2\epsilon S(t)I(t) \end{aligned} \tag{3.24}$$

It follows from the stochastic maximum principle that, the adjoint equations are:

$$dp(t) = -\frac{\partial H(x(t), u, p, q)}{\partial x}dt + q(t)dW(t)$$

$$\begin{cases} dp_1 = \left[p_1\left(\left(\beta - \frac{I(t)\pi}{1+I(t)}\right) I(t) + ((1+u_1(t))\phi + \mu)\right) - p_2\left(\beta - \frac{I(t)\pi}{1+I(t)}\right) I(t) - p_3(1+u_1(t))\phi + q_1\epsilon I(t)\right] dt \\ -q_2\epsilon I(t)dt + q_1 dw \\ dp_2 = \left[p_1\left(\beta S(t) - \frac{2\pi I(t)S(t) + \pi S(t)I^2(t)}{(1+I(t))^2}\right) - p_2\left(\beta S(t) - \frac{2\pi I(t)S(t) + \pi S(t)I(t)^2}{(1+I(t))^2} - ((1+u_2(t))\gamma + \delta + \mu)\right)\right] dt \\ + \left[-p_3(1+u_2(t))\gamma - A_1 + q_1 S(t)\epsilon - q_2\epsilon S(t)\right] dt + q_2 dw \\ dp_3 = p_3\mu dt \end{cases}$$

Then the stochastic adjoint system is

$$\begin{cases} \dot{p_1} = \left[(p_1 - p_2)\left(\beta - \frac{\pi I(t)}{1+I(t)}\right) I(t) + (p_1 - p_3)(1 + u_1(t))\phi + p_1\mu + (q_1 - q_2)\epsilon I(t)\right] dt + q_1 dw \\ \dot{p_2} = \left[-A_1 + (p_1 - p_2)\left(\beta S(t) - \frac{\pi I(t)S(t)(2+I(t))}{(1+I(t))^2}\right) + (p_2 - p_3)(1 + u_2(t))\gamma + p_2(\delta + \mu) + (q_1 - q_2)\epsilon S(t)\right] dt \\ +q_2 dw \\ \dot{p_3} = p_3\mu \end{cases} \tag{3.25}$$

With the initial and terminal conditions

$$S(t_0) = S(0), \quad I(t_0) = I(0), \quad R(t_0) = R(0), \tag{3.26}$$

$$p_1(T) = 0, p_2(T) = 0, p_3(T) = 0 \tag{3.27}$$

By differentiating Hamiltonian equation with respect to u_1 and u_2, we get the optimal controls u_1^* and u_2^*:

$$u_1^*(t) = \max\left[\min\left(\frac{S(t)\phi(p_1 - p_3)}{A_2}, 1\right), 0\right] \tag{3.28}$$

$$u_2^*(t) = \max\left[\min\left(\frac{I(t)\gamma(p_2 - p_3)}{A_3}, 1\right), 0\right] \tag{3.29}$$

where p_1, p_2 and p_3 are marginal costs. If $p_1 > p_3$, vaccinating susceptibles is economically valuable by reduces future infections. If $p_2 > p_3$, treating infections is economically valuable by reduces deaths and hospitalizations [53][16].

The numerical simulation of results of deterministic and stochastic optimal control problems are discussed in the next chapter 4.

Chapter 4

Numerical analysis of optimal control for Influenza with media awareness programs under treatment and vaccination

In this chapter we want to present the numerical results and discussion of deterministic and Stochastic optimal control analysis of Influenza. The feasibility of analysis regarding deterministic and stochastic optimality conditions are simulated numerically over time. All parameter values in the computations are the same in both scenarios [4][45][5]. They are summarized in the Table 3.1. For simulations we use Python programming language.

4.1 Numerical analysis of deterministic optimal control problem for Influenza

In this section, the numerical simulations of the deterministic optimal control problem is discussed.

An iterative scheme of fourth order Runge-Kutta method is used for solving the deterministic optimality system. The algorithm is the forward-backward scheme; starting with an initial guess for the optimal controls, the state variables are then solved forward in time from the dynamics of system using a Runge-Kutta method of the fourth order. Then, those state variables and initial guess for the controls are used to solve the adjoint equations backward in time with

given final conditions, again employing a fourth order Runge-Kutta method. The controls are updated and used to solve the state and then the adjoint system. This iterative process terminates when current state, adjoint, and control values converge ([1] [4] [45][46]). This method can be summarized in the following algorithm.

Algorithm 5: The forward-backward scheme method

1 **Input :** $x(0) = [S(0), I(0), R(0)]$,$u^* = [u_1^*(0),\ u_2^*(0)]$, $\lambda_i(T) = 0$.

2 **Output :** u_1^*, u_2^*, $S^*(t)$, $I^*(t)$, $R^*(t)$

3 **Solve Forward the State Equations using a numerical method (e.g., RK4):**

4

$$\dot{x} = b(x, u), \quad x(0) = x_0.$$

5 **Using the terminal conditions** $\lambda_i(T) = 0$ **and the values for** u_1 ,u_2 **and** $S(t)$, $I(t)$, $R(t)$, **solve the Costate (Adjoint) Equations backward in time :**

6

$$\dot{\lambda}_i = -\frac{\partial H}{\partial x}, \quad \lambda_i(T) = 0.$$

7 **Update** u_1 **and** u_2 **by entering the new** S^*, I^*, R^* **and** λ_i **values into the characterization of the optimal control.**

8 **Iterate until convergence.**

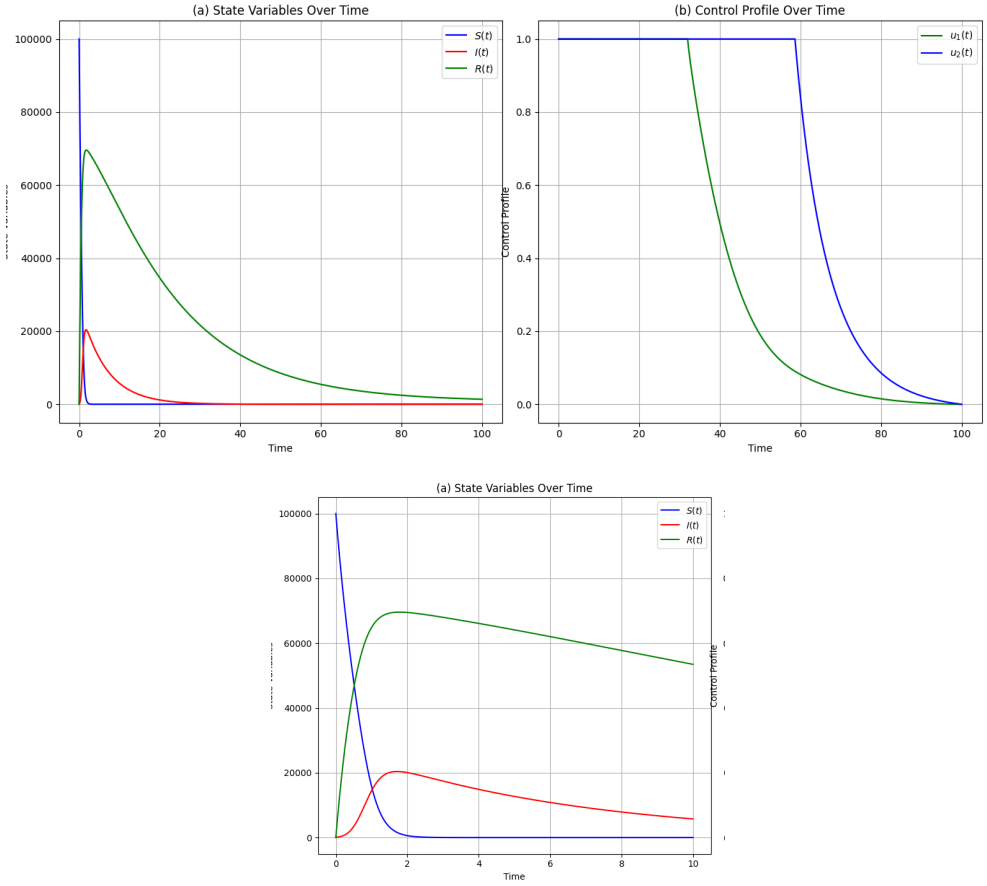


Figure 4.1: Simulation of deterministic model solution (top left). Control profile $u(t)$ (top right). Zoom of the solution for the 10 first days (bottom).

Note: *The program execution took 35 seconds to complete the full simulation.*

Interpretations

Figure 4.1 presents the simulation of deterministic model solution and control profile $u(t)$. The results indicate an increase in the number of recovered individuals and a reduction in infections. The vaccination effort, represented by u_1, is maximized for up to 34 days before gradually decreasing to its lower bound over the next 100 days. Meanwhile, the control u_2, which regulates the treatment of infected individuals, achieves optimal results when maintained at its maximum level from $t = 0$ to $t = 58$ days.

4.2 Numerical analysis of stochastic optimal control

We use Least-squares regression based method, where the number of Monte Carlo paths is 1000 and only 200 are plotted. The state variables are solved forward in time from the dynamics system (3.23) using a forward Euler-Maruyama method, then Least-squares regression based method is applied to solve the adjoint equations (3.25) backward in time with given final conditions where the polynomial basis functions up to degree 2 is used for the least-squares regression. Then the controls are updated and used to solve the state and the adjoint systems.

Note: *The program execution took 135.90 seconds to complete the full simulation.*

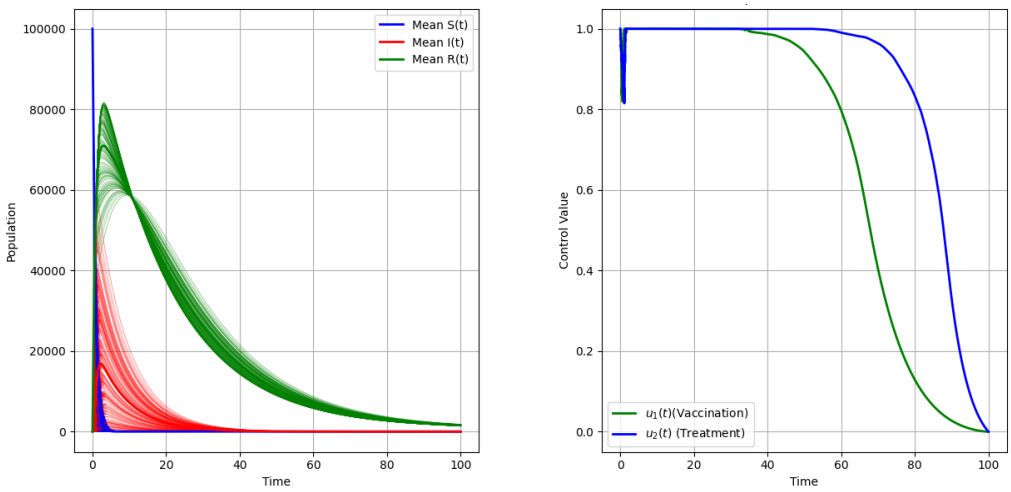


Figure 4.2: Simulation of stochastic model solution (Solution of FSDEs) and control profile u_1 (vaccination), u_2 (treatment).

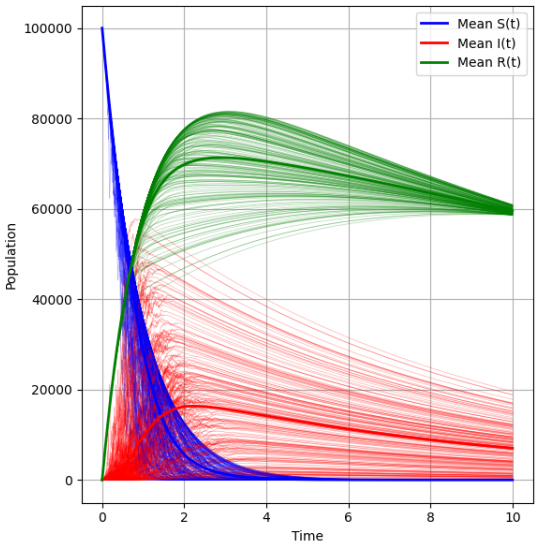


Figure 4.3: Zoom of the stochastic model solution for the 10 first days.

Interpretations

As control is applied, the infected population gets drop down, and the susceptible population is reduced quickly while the recovered population gets drop up over time meaning that the intervention is effective.

This result suggests that an aggressive control strategy is initially required, where maximum intervention is applied at the start and then relaxed as the epidemic is being reduced under controls. The sudden drop at the end may indicate that the infection has diminished to a level where further intervention is no longer necessary.

Moreover, the relatively high weight for vaccination ($A2 = 150$) means the cost of vaccination is significantly higher than the cost of treatment, encouraging the system to reduce vaccination efforts earlier to minimize costs that's why the vaccination effort drops down after 34 days. This indicates that as the weight of control (vaccination efforts) increases, the disease can be controlled in a minimum time. Also the treatment control also starts near 1 but decreases after 58 days. The weight of treatment ($A3 = 100$) makes it a more cost-effective intervention over a longer period compared to vaccination.

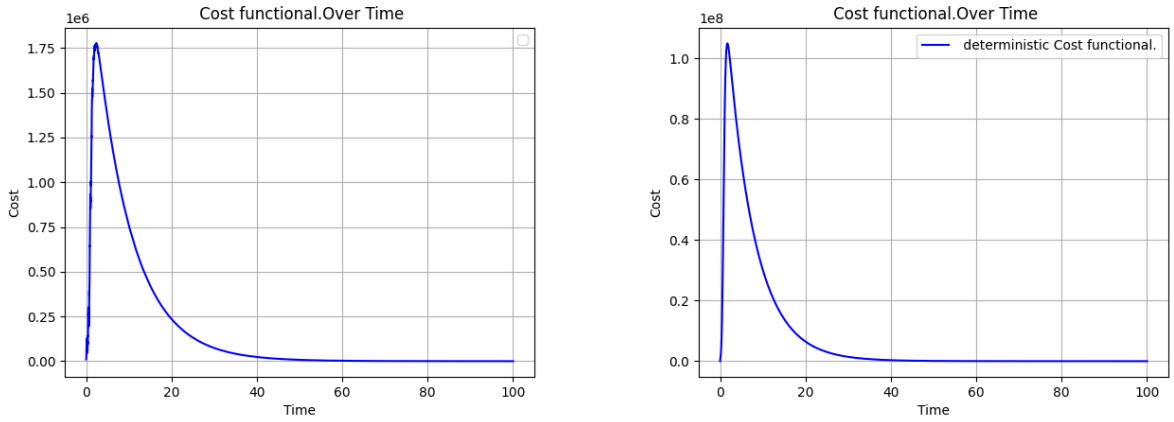


Figure 4.4: Simulation of deterministic and stochastic cost functional

Figure 4.4 represent the evolution of the deterministic and stochastic cost functional over time. From the figure the stochastic model demonstrates that incorporating noise yields more precise results compared to the deterministic model because the cost associated with the stochastic problem is less than that of the deterministic problem. We conclude that the deterministic model provides a baseline prediction, but the stochastic model is more effective to minimize infection with minimum costs.

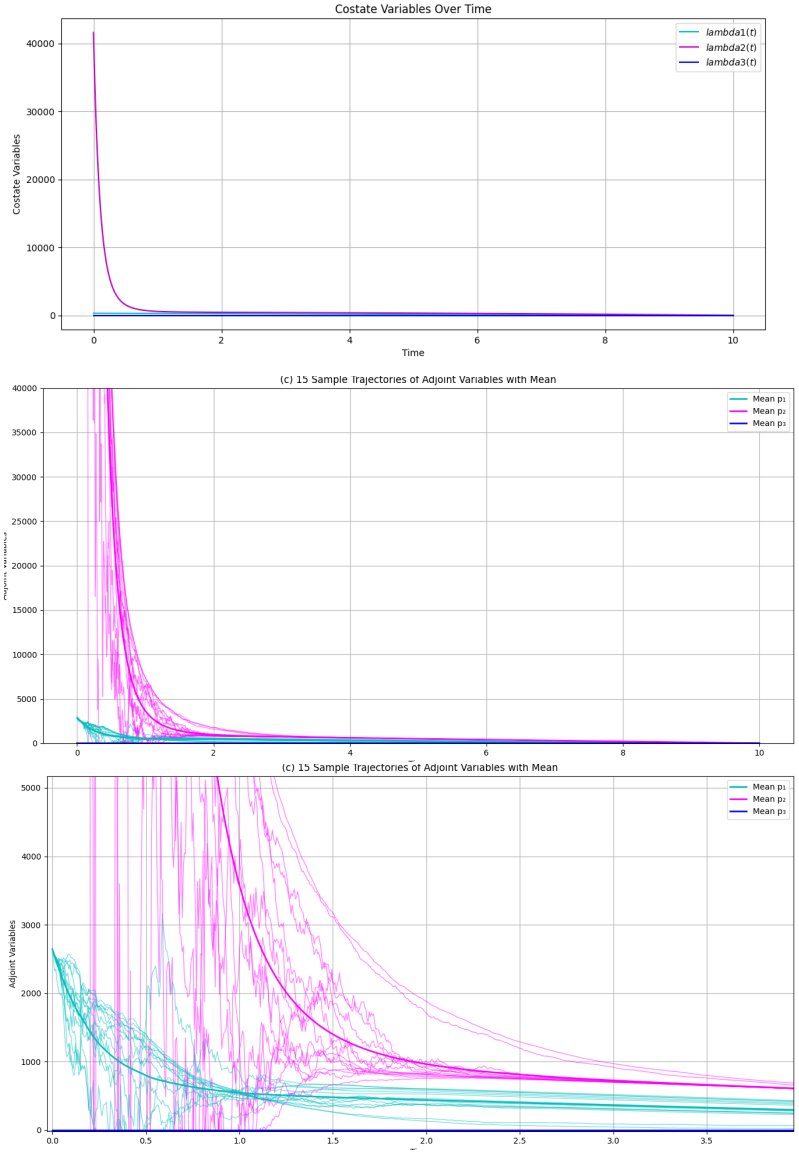


Figure 4.5: Simulation of deterministic and stochastic adjoint trajectories .

Note: *The program execution took 67.09 seconds to complete the full simulation. It is also worth noting that we take N=1000 Monte Carlo paths, and only 15 are plotted to save time.*

Figure 4.5 shows the simulation of deterministic and stochastic adjoint (Costate) trajectories over time, it is clear from the figure $p_1 > p_3$ and $p_2 > p_3$ so vaccinating susceptibles and treating infections are economically valuable to reduce future infections, deaths and hospitalizations.

Remark 4.2.1: *We can solve stochastic optimal control problem by using a so-called "proxy method", as mentioned in [4, 42].*

4.3 Deterministic and stochastic analysis for different Scenarios

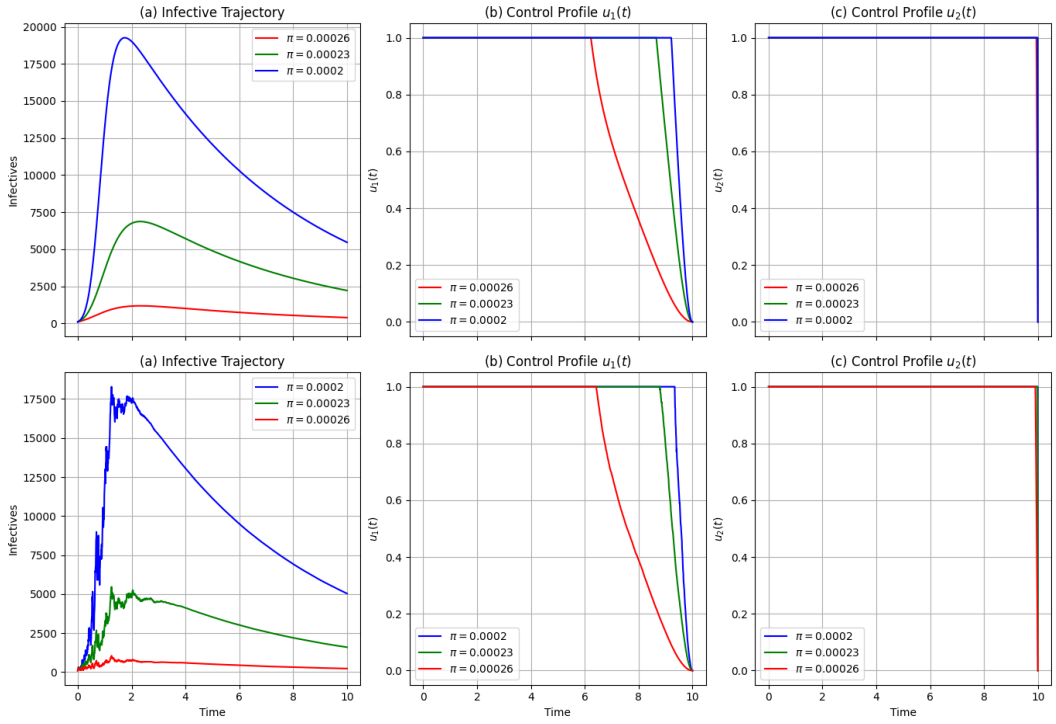


Figure 4.6: Simulation of deterministic and stochastic infective trajectory, and control profile u_1 and u_2 for varying rate of awareness π.

Note: *In order to save time, the simulation time horizon was limited to 10 units of time. It is important to note that this reduction does not affect the accuracy or reliability of the results.*

Figure 4.6 illustrates the impact of varying the media awareness rate on the infective trajectory and the optimal control strategies for vaccination (u_1) and treatment (u_2). As the value of π increases from 0.0002 to 0.00026, the number of infective individuals decreases over time, this confirms that as awareness grows infection decreases. For vaccination effort (u_1), greater values of awareness, reduce the vaccination effort over time. In contrast, the treatment effort (u_2) remains at its maximum across all awareness levels, highlighting the continued necessity of treatment efforts to avoid a new waves of the epidemic because some individuals become infected despite awareness.

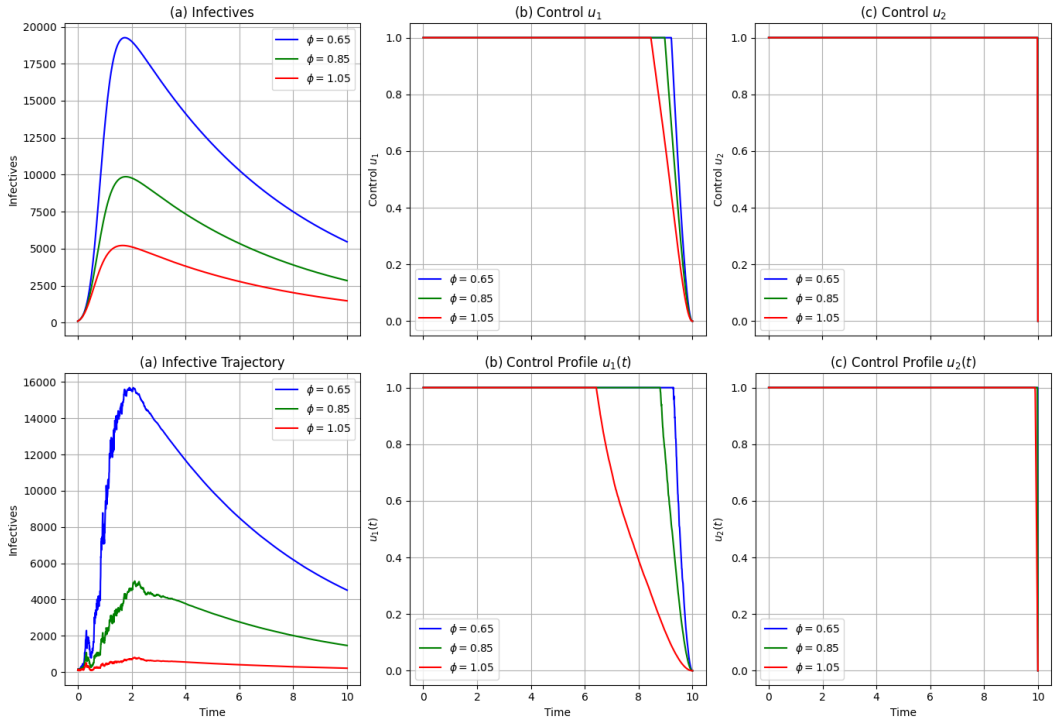


Figure 4.7: Simulation of deterministic and stochastic infective trajectory, and control profile $u_1(t)$ and $u_2(t)$ for varying rate of vaccination parameter ϕ.

Figure 4.7 presents the simulation results for infective trajectories and control profiles under different values of the vaccination rate parameter. Figure 4.7(a) shows that as the vaccination rate parameter increases from 0.65 to 1.05, the number of infective individuals decrease. This confirms the direct role of effective vaccination in mitigating disease spread. From figure 4.7(b), for larger values, such as 1.05, the vaccination effort drops earlier and more gradual. On the other hand, figure 4.7 (c) shows that the treatment control remains at its maximum level across all vaccination rates, indicating that treatment continues to play a crucial role, regardless of how effective the vaccination is.

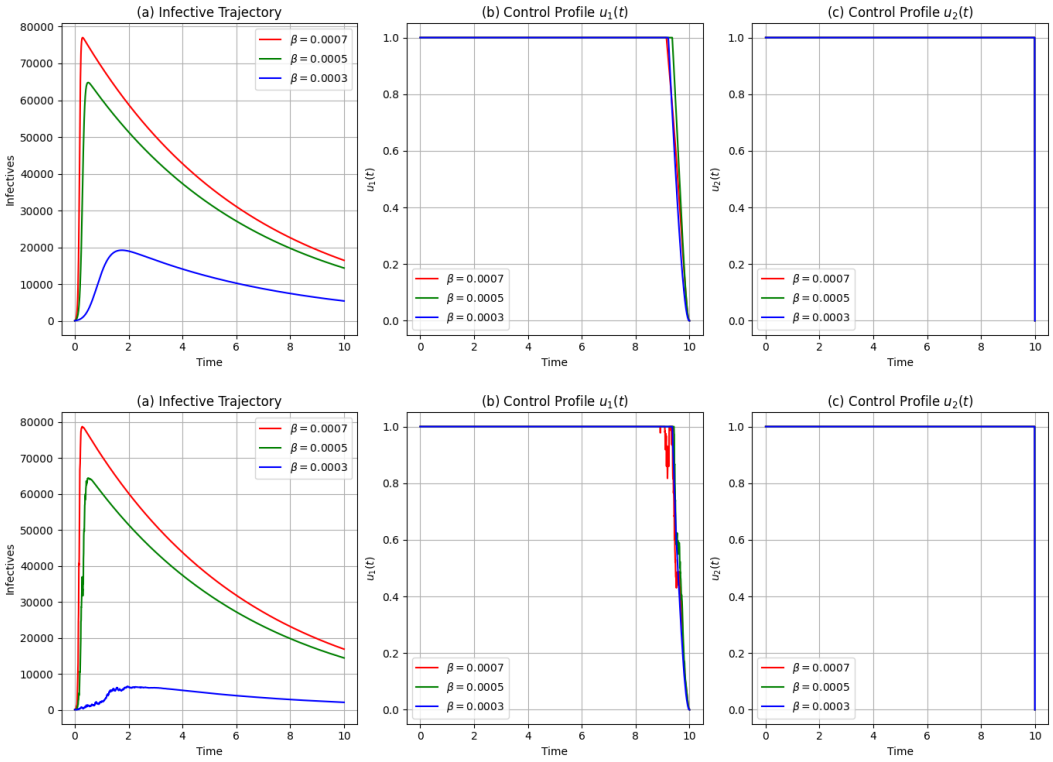


Figure 4.8: Simulation of deterministic and stochastic infective trajectory, and control profile $u_1(t)$ and $u_2(t)$ for varying Contact rate of susceptible with infectives β.

Figure 4.8 shows the simulation results for infective trajectories and optimal control profiles with respect to varying values of the contact rate β between susceptible and infective individuals. From figure 4.8 (a), the infective trajectory increases as the contact rate β rises. This highlights the strong influence of contact rate β. Figure 4.8 (b) illustrates the vaccination effort (u_1). The intensity and fluctuations in (u_1) are more pronounced for higher values, indicating a more reactive and sustained vaccination effort is needed as the disease spreads faster with higher contact rates. From figure 4.8(c), the treatment control remains at its maximum throughout the simulation across all contact rates, suggesting that treatment must be continuously applied at full capacity to manage the infectious population effectively.

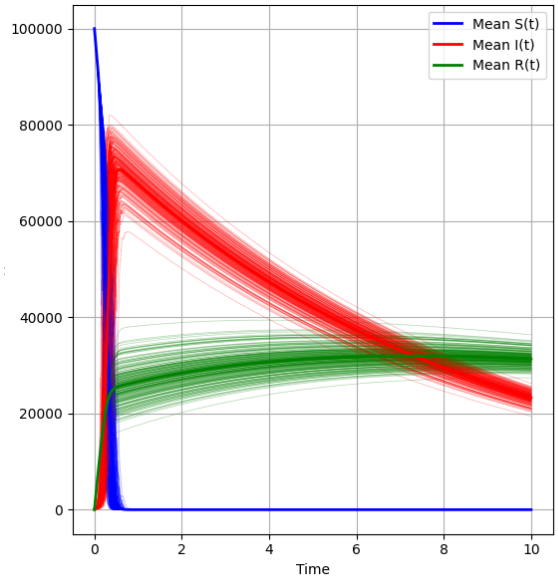


Figure 4.9: Simulation of state variables over time without media awareness and without vaccination with treatment.

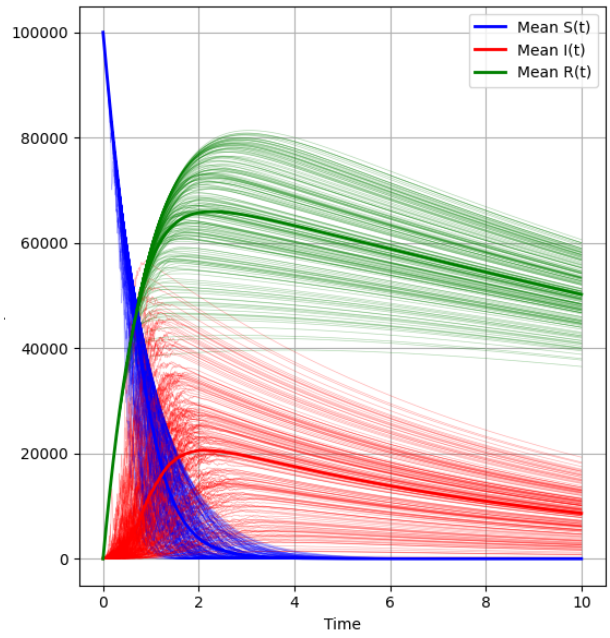


Figure 4.10: Simulation of state variables over time with media awareness and vaccination without treatment.

From figure 4.9 there is a small reduction in the number of infections in the presence of treatment,while a significant drop occurs in the presence of media awareness program and vaccination without treatment as shown in the figure 4.10 . This confirms the importance of prevention

strategies in mitigating epidemics. We conclude that the prevention is better than cure.

General Conclusion

The dissertation focuses on developing stochastic epidemic models that use stochastic differential equations for numerical simulation of the Susceptible-Infected-Recovered (SIR) framework for influenza. Our research demonstrates the implementation of Pontryagin's Maximum Principle along with the Stochastic Maximum Principle to solve both the deterministic and stochastic optimal control problems.

The comparison between deterministic and stochastic control strategies was conducted through numerical simulations to identify their respective effectiveness. The study results show the fundamental necessity of including uncertainty within epidemic control models and strategies. Stochastic optimal control methods deliver more authentic disease spread prevention techniques for uncertain conditions which makes them essential for practical implementation.

The work provides essential information to policy makers together with public health authorities about how stochastic components improve epidemic control strategies to create better adaptable interventions. In this work the most effective and essential control measures among all prevention strategies are media awareness programs together with vaccination. These results can be useful through further investigation of complex models. By using real world data and approximate stochastic control based on more sophisticated techniques such as Deep Learning, these findings could be of valuable degree to treat and interpret more complex epidemic models.

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