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A Discrete Study on Stochastic Epidemic Models with the Optimal Control Policies and Its Analysis

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Abstract: The article aims to assess the stochastic model's control variant and study the effect of time on the epidemic's behavior. A single population must have strong immunity as it recovers from the epidemic. Lower the infected and susceptible individuals and maximize the absolute amount of recovered individuals by using possible minimal control variables. We have demonstrated whether we should implement a treatment policy to minimize the number of death cases by examining three different examples from various perspectives. We have also proved that the best policy in the event of a fatality is to avoid it. The epidemic model will be a bang policy with only one switch if the cost function depicted in the illustration is utilized. Furthermore, a switch can only be activated if criteria are satisfied. As a result, we investigate the approaches for preventing the HBV model. Finally, we will develop a more realistic model-building strategy that integrates the emergence of treatment effect on the infectives during their incubation period for a fatal epidemic. A fatal epidemic is expected to be more severe than a general epidemic, and more realistic model-building approaches are developed. The same mathematical methods and conclusions may be used nearly immediately to a wide range of spreading processes, which should be stressed.

Keywords: discrete study, stochastic epidemic model, optimal control.

具有最优控制策略的随机流行病模型的离散研究及其分析

摘要:本文旨在评估随机模型的控制变量,并研究时间对流行病行为的影响。单一人群 在从流行病中恢复时必须具有很强的免疫力。通过使用可能的最小控制变量来降低感染和易 感个体并最大化恢复个体的绝对数量。我们通过从不同角度研究三个不同的例子,证明了我 们是否应该实施一项治疗政策,以尽量减少死亡病例的数量。我们还证明,在发生死亡事件 时最好的策略是避免它。如果使用插图中描述的成本函数,流行病模型将是一个只有一个开 关的爆炸策略。此外,只有在满足标准时才能激活开关。因此,我们研究了预防乙肝病毒模 型的方法。最后,我们将制定一个更现实的模型构建策略,该策略将在致命流行病的潜伏期 对感染者的治疗效果的出现进行整合。预计致命的流行病将比一般流行病更严重,并且开发 了更现实的模型构建方法。相同的数学方法和结论几乎可以立即用于广泛的传播过程,应该 强调这一点。

关键词:离散研究、随机流行病模型、最优控制。

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1. Introduction

Epidemic models summarize and explain data on communicable diseases. Furthermore, they help us better understand the biological and sociological mechanisms underlying the spread of disease so that public health authorities, medical practitioners, and the government can take preventative measures to manage epidemic. For researchers, developing the an acceptable and accurate mathematical epidemic model is critical. In addition, these mathematical models aid in translating a description of individual behavior to a description of the transmission of disease in a population. In most communicable diseases, we have a good idea of how the disease is transmitted and how a single infection can spread. However, epidemiology does not have the formal framework needed to examine the potential breadth of an infection chain that is at least partially triggered by chance interactions. That makes mathematical models necessary to determine the likely spread of an epidemic.

1.1. Analyzing Epidemics Using Modeling

When it comes to modeling spreading processes, there are infinite possibilities. Every epidemic model is based on the existence of "compartments" in which people of a community are separated. Epidemic models often have two compartments: "Susceptible" and "Infected" (or simply "Susceptible"). An initial sample of a particular population is partitioned into these two divisions in models with only these two options. There are healthy but sensitive to infection in the "Susceptible" compartment (S). "Infected" persons are kept in the "Infected" compartment (I). From here, the population's interactions can be modeled in many ways.

Numerous alternative epidemic models attempt to capture different real-world disease transmission and dissemination aspects. For example, people who are no longer susceptible to the virus might be added to the "Removed" (R) compartment, commonly added to the database. A deceased, vaccinated, or immune person might be the subject of this statement. Other compartments have been proposed in the literature to explore the effect of, for example, an incubation time or partial immunity, or quarantine.

1.2. Control of Epidemics

Stochastic network models should be controlled as fast as possible to stop the spread of disease. However, we must first discuss our effective 'control levers' for addressing an epidemic before going into the specifics. The heterogeneous SIS dynamics will be enough for our purposes here.

$$p_i = -\delta_I p_i + \sum_{j=1}^{M} \beta_{ij} p_j (1 - p_i)$$
⁽¹⁾

M subpopulations in a metapopulation model, to put it another way, each node I represents a subpopulation (like a town) within a larger population (like a country)

of N individuals. The recovery rates I and infection rates ij, which define the interactions between diverse subpopulations, are the factors we must experiment with.

It is generally desired to enhance the recovery rate I and lower the infection rate is in order to reduce epidemic impacts. Better medical care for sick people can improve their chances of recovery in a certain population segment. If more resources were allocated, this subpopulation could benefit from additional doctors or more effective treatment modalities. There are several methods for reducing infection rates. First, the infection rate can be reduced by restricting movement between different populations. Since a subpopulation I can no longer affect other subpopulations, quarantining it is the same as setting *ji* = 0 for all *j*. For strategies to reduce the infection rate, there are more subtle tactics like giving masks to a population and raising knowledge about a disease so that individuals are less likely to get it.

Naturally, the disease would die fast if we had unlimited resources and treatment capability and kept everyone in confinement; however, this is not a practical option. Thus, given a limited budget, it is necessary to establish which factors are most critical tominimize the disease's consequences as much as feasible. We will formulate these issues and assess where the technology stands right now as a further step.

1.3. What Is the Significance of Epidemic Models?

We can describe the infectious individual's part in spreading the disease before describing how it spreads throughout a community using an epidemic model. Therefore, a model that depicts disease transmission within a community leads to crucial insights, such as the epidemic threshold theorem, which has a wellunderstood interpretation for its parameters.

Comparisons are frequently used to help researchers better understand how diseases spread. As a result, we can look at outbreaks of the same disease in various locales and periods and outbreaks of distinct diseases. In many ways, building epidemic models that correctly represent diverse epidemic outbreaks and then comparing the models is the best way to make such comparisons. That is a common statistical analysis approach. In addition, epidemic models play a significant role in evaluating the effectiveness of disease control measures. However, it is common in most scientific fields for researchers to examine the effects of changes by comparing the findings of multiple trials. As a result, it is not always possible to conduct trials involving epidemic outbreaks of disease. For this challenge, it is natural to try to develop a model that adequately captures the basic properties of epidemics in the community. Then these models are used to anticipate the specific alterations. For example, the objective is that changing the model to reflect the proposed campaign will appropriately represent the basic aspects of epidemics in a population that has only been partially vaccinated by using a model like this to evaluate a vaccine campaign.

2. Review of Related Studies

Research of Tran, Ky & Yin, George [1] focuses on stochastic SIS epidemic models under regime change. Assuming that a decision-maker can either alter the infectivity period or reduce the projected discounted cost of disease, medical treatment, and the negative impact on society, we hope to minimize those estimated discounted costs. In addition, a vaccine developed. Markov model has been chain approximation methods are used to build numerical approaches for estimating continuous-time dynamics. The approximation strategies converge to the best strategy as the mesh size decreases. To demonstrate our findings, we present numerical examples.

A non-linear stochastic deterministic SIS model with vaccination is the subject of [2]. Two timedependent control factors are used in the research to discover the most effective methods of limiting the spread of disease using deterministic and stochastic optimal control analyses. We numerically apply an estimate based on the deterministic model's answer to solve the stochastic optimal control problem. The numerical simulations are used to show and compare the results. Following the intensity of noise, the likelihood of an illness and the expense of nonpharmaceutical therapies and vaccinations rises.

Boutayeb et al. [3] help better understand how information spreads in online ecosystems like Facebook, WhatsApp, and Twitter. We have developed a new discrete-time model. An additional compartment is added to the model to study the effect of sharing on the amount of information. We consider the possible interactions between persons and information on the Internet, such as posts, photographs, and videos. With the help of a theoretical framework, we can demonstrate how our optimal control technique reduces the amount of shared data and the number of people who share it, thus lowering annoyance and instability in society. We conduct numerical simulations to examine various scenarios before and after implementing our control approach. Simulated and discussed sensitivity analysis of parameter information is also included.

Kovacevic et al. [4] investigate infectious disease transmission dynamics using a continuous-time stochastic SIS epidemiological model. Previously afflicted people might re-enter the transmission chain after healing. Individuals who have been infected with a decision-maker concerned with minimizing costs connected with illness and pharmacological therapy can dynamically regulate disease. This stochastic control problem makes two alternative assumptions about the available information available. First, the Hamilton-Jacobi-Bellman (HJB) equation can be used to predict the best degree of control at any time using a complete and accurate count of infected individuals. If no state measurement is available, that can be recast as an optimal control problem for the Kolmogorov forward equation. Because of the degeneracy of the HJB and Kolmogorov equations, unusual reasoning is required to establish optimality requirements in both circumstances. Information patterns have been studied quantitatively based on the theory that has been established so far.

According to Lorch et al. [5], SIS epidemic processes can be modeled and controlled by Stochastic optimum control of stochastic differential equations with leaps from the standpoint of marked temporal point processes and stochastic optimal control of stochastic differential equations with jumps. For the first time in the history of disease outbreak research, we have a new perspective that can help us transcend the limitations of current control tactics. To keep the number of people infected as low as possible, we use treatment intensities to choose who and when to treat. Preliminary tests show that our control technique regularly beats several options with simulated data. Our method may make future developments of realistic data-driven control tactics for epidemic processes possible.

Bolzoni et al. [6] in SIR (Susceptible-Infected-Recovered) epidemic models investigate the optimal control problem, focusing on alternate control approaches such as vaccination, isolation, culling, and transmission reduction. Using the Minimum Principle of Pontryagin (PMP), we prove that only bang-bang controls with a maximum of one switch are allowed for all the policies considered. There are times when it makes sense to postpone control actions and subsequently apply them at their greatest rate for the rest of the outbreak. A recent study found that the ideal technique for lowering the total infectious burden across an outbreak is to utilize the maximum control possible for the whole epidemic. The most important implication of our findings is that it may be difficult to reduce the total infectious burden while also shortening an epidemic's duration and vice versa.

Furthermore, numerical simulations revealed that optimal control could be delayed even when the control reproduction number is less than one. The transition from no control to maximum control can occur even after the infection has peaked. Our findings are especially relevant to livestock infections because sanitary restrictions imposed on farms during ongoing outbreaks, such as animal movements and export prohibitions, need shorter outbreak duration.

According to Nasir et al. [7], SIR pandemic infection may be modeled using a discrete stochastic model, and the optimal control policy can be calculated using this model. A Markov Decision Process (MDP) model is offered as an alternative to state-space modeling. State-specific actions and probability are included in the proposed model. For calculating the optimal control policy, an optimality criterion is discussed. Case studies and graphical representations explain the optimal policy's behavior and the tradeoffs involved in selecting the optimality criterion. Scaling population size is also introduced to deal with largescale issues.

Halawar et al. [8] investigate a non-linear dynamical system of linear quadratic control. First, the distinction between stochastic and deterministic control systems is demonstrated, and the incidence of symmetry breaking as a noise function is included in the stochastic model's definition. Then, the Pontryagins Maximum Principle is used to solve the Deterministic optimum control issue and show its existence. Next, the stochastic optimal control problem is examined with the Maximum Stochastic Principle and numerical simulations. Finally, we numerically apply an estimate based on the deterministic model's answer to solve the stochastic optimal control problem.

Sharom and Malik [9] analyzed that Combinations of isolation, quarantine, vaccination, and therapy are frequently required to eliminate most infectious diseases, according to mathematical modeling of infectious diseases. However, disease elimination will be tough if they are not provided at the right moment and in enough volume. Against the spread of infectious diseases, optimum control theory can be used to devise the best disease intervention tactics. This strategy can reduce both the expense of the infection and of administering the control. Mathematical models that use optimal control theory to determine the best tactics for limiting the spread of an infectious disease are reviewed in this study.

Lee et al. [10] analyzed Stochastic approaches for calculating influenza transmission models presented in this research. Stochastic modeling for deterministic SEIR-type epidemiological models is first revisited. The primary goal of our research is to demonstrate the computational methodologies for stochastic epidemic models. First, some influenza models are constructed using the moment closure method (MCM) and compared to findings obtained using the traditional stochastic simulation approach (SSA). Even though both methods produce related peak and end epidemic size results, the MCM has drastically shortened calculation time and expenses. Afterward, the MCM was used to predict the spread of the 2009 H1N1 flu virus in South Korea. Next, the usual deterministic strategy and the stochastic approach are contrasted for influenza outcomes (MCM). Our findings reveal that stochastic and deterministic models have a significant disparity when only many infected individuals are present. That is followed by looking at vaccine and antiviral treatment's effectiveness in various settings.

Ding & Lenhart [11] emphasized disease models.

This paper introduces the theory of optimal control applied to discrete-time models. Such optimum control problems can be solved by following simple steps and discussing a few preconditions. This example illustrates how to apply Pontrya-Maximum gin's principle to characterize the ideal level of control. Numerical data are presented to demonstrate certain examples.

3. Research Methods

3.1. Optimal Control Analysis for a Deterministic System

3.1.1. Model 1

The deterministic version of the stochastic system in equation (1) is as follows:

$$\frac{dA(t)}{dt} = \frac{\theta S(t)A(t)}{N} - \left(\mu_0 + \sigma + \gamma_1\right)A\left(t\right)$$

$$\frac{dS(t)}{dt} = \Lambda - \frac{\theta S(t)A(t)}{N} - \left(\mu_0 + \delta\right)S\left(t\right)$$

$$\frac{dB(t)}{dt} = \sigma A\left(t\right) - \left(\mu_0 + d_1 + \gamma_2\right)B\left(t\right)$$

$$\frac{dR(t)}{dt} = \gamma_1 A\left(t\right) + \gamma_2 B\left(t\right) + \delta S\left(t\right) - \mu_0 R\left(t\right)$$
(2)

3.1.2. Model 2

An effective weapon in the fight against infectious diseases is mathematical modeling's optimization programming.

Those who have been infected may be able to understand and prepare for the immunization. u1(t) for S(t) and u2(t) for A(t), B(t) are added to the equation to apply the vaccine (2). Our goal is to reduce infection; we follow these guidelines.

$$\mathcal{F}\left(u_{1}, u_{2}\right) = \int_{0}^{T} \left[\left[\xi_{1}S\left(t\right) + \xi_{2}A\left(t\right) + \xi_{3}B\left(t\right) + \frac{1}{2}\left(\xi_{4}u_{1}^{2}(t) + \xi_{5}u_{2}^{2}(t)\right) \right] dt,$$
(3)

Subject to

$$\begin{aligned} \frac{dd}{dt}S\left(t\right) &= \Lambda - \frac{\theta S(t)A(t)}{N} - (\mu_0 + \delta + u_1(t))S\left(t\right) \\ \frac{d}{dt}A\left(t\right) &= \frac{\theta S(t)A(t)}{N} - (\mu_0 + \sigma + \gamma_1)A\left(t\right) - u_2\left(t\right)A\left(t\right) \\ \frac{d}{dt}B\left(t\right) &= \sigma A\left(t\right) - (\mu_0 + d_1 + \gamma_2)B\left(t\right) - u_2\left(t\right)B\left(t\right) \end{aligned}$$
(4)
$$\begin{aligned} \frac{d}{dt}R\left(t\right) &= \gamma_1 A\left(t\right) + \gamma_2 B\left(t\right) + (u_1\left(t\right) + \delta\right)S\left(t\right) - \mu_0 R\left(t\right) + u_2\left(t\right)A\left(t\right) + u_2\left(t\right)B\left(t\right) \end{aligned}$$

S(t), A(t), and B(t) must be in proportion to each other, and these little positive constants, $\xi 1$, $\xi 2$, and $\xi 3$, ensure that they are.

 $S0 > 0, A(0) = A0 \ge 0, B(0) = B0 \ge 0, R0 > 0.$ (5) Infected populations' values or weights are referred to as $\xi 1$, $\xi 1$, and $\xi 3$ in (3), which might be unchanged, positive, or zero. As a result, the cost of cure and vaccine will be represented by (3) $\xi 4$ and $\xi 5$, both positive constants. The expense of lowering both types of infection will be made clear in our goal. We will not be able to reduce the number of susceptible, but our goal is to train operators $(u_1^*, u_{2,i}^*)$ such as

$$J(u_2^*, u_1^*) = \min\{J(u_2, u_1), u_2, u_1 \in U\},$$
(6)

Subjected to the control system (4) and for (5), where U is

 $U := \{(u2, u1) | 0 \le u2, u1 \le 1, u2, u1 \ @s``Lebesgue measurable "on[0, T] i = 1, 2\}$ (7)

First, we determined the basis for our control strategy's existence.

3.2. Existence of Solution

This section will cover a qualitative analysis of the solutions to (3)–(7). Because the control parameters are already non-negative and Lebesgue measurable, we need to verify that a solution exists that is bounded in positive existence. Take

$$\Theta t = X\Theta + C(\Theta), \tag{8}$$

There is a non-linear system with a fixed coefficient in Eq. (8): where Θ t indicates the derivative of Θ concerning time. We established the parameters.

$$\Theta = \begin{pmatrix} S(t) \\ A(t) \\ B(t) \\ R(t) \end{pmatrix}$$

$$C = \begin{pmatrix} -(\mu_0 + \delta + u_1(t)) & 0 & 0 & 0 \\ 0 & -(\mu_0 + \sigma + \gamma_1 + u_2(t)) & 0 & 0 \\ 0 & 0 & -(\mu_0 + d_1 + u_2(t) + \gamma_2) & 0 \\ u_1(t) & u_2(t) + \gamma_1 & u_2(t) + \gamma_2 & -\mu_0 \end{pmatrix}$$

$$X \left(\Theta\right) = \begin{pmatrix} \Lambda - \theta S(t)A(t) \\ \theta S(t)A(t) \\ 0 \\ 0 \end{pmatrix}$$

As Equation (8) bounded and non-linear, therefore set

$$G(\Theta) = C\Theta + \psi(\Theta),$$
 (9)
This satisfies:

$$\begin{split} \psi(\theta 1) - \psi(\theta 2) &| \leq n1|S1(t) - S2(t)| + l p 2| - A2(t) + A1(t)| + n3| - B2(t) + B1(t)| + n4|R1(t) - R2(t)|, \leq N(|S1(t) - S2(t)| + |A1(t) - A2(t)| + |B1(t) - B2(t)| + |R1(t) - R2(t)|), \end{split}$$
(10) where $N = max\{(n_1, n_2, n_3, n_4)\}$ is free of state parameters of system (2). For this we can also take

$$|G(\Theta_1) - G(\Theta_2)| \le |\Theta_1 - \Theta_2|M, \tag{11}$$

Here $< \infty > M = \max\{\|C\|, N\}$, infers that G is welldefined and meets Lipschitz's requirement of uniformly continuous evolution. We conclude that the model's solution exists (4) because the control and state parameters are non-negative. The next section will see how the control variable affects our function's end aim.

4. Result and Discussion

Theorem 1: Asu $* = (u_2^*, u_1^*) \in U$, there will be two optimal variables for managing the system (3)–(7).

Proof: To arrive at our conclusion, we must

demonstrate that the parameters outlined are sufficient.

The control and state variables have already been proven to be positive. There is an additional need for compactness: the set of (7) must be convex and near and bound. Convex functions may alternatively represent the control parameters of the goal function. Because the equation under consideration is correct, the optimal control (u_1^*, u_2^*) can be found.

4.1. Constraints on Optimality

Here, we need to describe the optimal control solution attribute of (3)–(7). The Hamiltonian and Lagrangian equations for our controlled issue must be formulated.

Let x = (S, A, B, R) and u = (u1, u2) in vector form represent the values of x and u, respectively. The Lagrangian describes the optimal solution to (3)–(7). We first need to define the Lagrangian and Hamiltonian for our control issue. Vectors x and u represent state and control variables, respectively, so let us denote them as x = (S, A, B, R) or u = (u1 and u2). This form will be the Lagrangian L.

$$L(S(t), A(t), B(t), u(t)) = \xi 1S(t) + \xi 2A(t) + \xi 3B(t) + \frac{1}{2} \left(\xi_4 u_1^2(t) + \xi_5 u_2^2(t) \right),$$
(12)

Functions change because of Hamiltonian H:

$$H(x, u, \lambda) = \lambda \cdot g(x, u) + L(x, u),$$
(13)
And $g(x, u) = (g_1(x, u) - g_2(x, u)), \quad \lambda = (\lambda)$

And $g(x, u) = (g_1(x, u), ..., g_4(x, u))$, $\lambda = (\lambda_1, ..., \lambda_4)$ and $u = (u_1(t), u_2(t))$ also $g_3(x, u) = \sigma A(t) - (\mu_0 + d_1 + \gamma_2)B(t) - u_2(t)B(t)$,

$$g_4(x,u) = \gamma_1 A(t) + \gamma_2 B(t) + (u_1(t) + \delta)S(t) - \mu_0 R + u_2(t)(A + B)(t)$$
(14)

In addition, the usual Pontryagin's Maximum Principle will be used for our control problem to ensure optimality conditions. Assuming (x^*, u^*) is an optimal solution of the investigated system in the form (3)-(7), then a "Hamiltonian" system is defined.

$$\begin{cases} \frac{dx(t)}{dt} = \frac{\partial H}{\partial \lambda} \left(x^{*}(t) \right), u^{*}(t), \lambda(t) \\ \frac{d\lambda(t)}{dt} = -\frac{\partial H}{\partial x} \left(\lambda(t), u^{*}(t), x^{*}(t) \right) \end{cases}$$
(15)

The maximality axiom:

$$H\left(x^{*}\left(t\right)\right), u^{*}\left(t\right), \lambda\left(t\right)\right) = \max_{u \in [0,1] \times [0,1]} H\left(x^{*}\left(t\right)\right), u^{*}\left(t\right), \lambda\left(t\right)\right)$$
(16)

And axiom of intersection is:

$$0 = \lambda(t_f) \tag{17}$$

Theorem 2: Let S*, A*, B*, and R* be the values of the state variables that correspond to the best controls. (u* 2, u* 1) for system (3)–(7). Then, \exists variables of adjoint λ i(t), i = 1,...,4, satisfying:

$$\dot{\lambda_{1}}(t) = -\xi_{1} + (\lambda_{1}(t) - \lambda_{2}(t)) \frac{\theta A^{*}(t)}{N} + (\mu_{0} + \delta + u_{1}^{*}(t))\lambda_{1}(t) - (u_{1}^{*}(t) + \delta)\lambda_{4}(t)$$

$$\dot{\lambda_{2}}(t) = -\xi_{2} + (\lambda_{1}(t) - \lambda_{2}(t)) \frac{\theta S^{*}(t)}{N} + \lambda_{2}(t)(\mu_{0} + \sigma + \gamma_{1}) - \lambda_{3}(t)\sigma - \lambda_{4}(t)\gamma_{1} + (\lambda_{2}(t) - \lambda_{4}(t))u_{2}^{*}(t), \qquad (18)$$

$$\begin{aligned} \lambda'_{3}(t) &= -\xi_{3} + (\lambda_{3}(t) - \lambda_{4}(t))u * 2(t) \\ &+ \lambda_{3}(t)(\mu_{0} + d_{1} + \gamma_{2}) - \gamma_{2}\lambda_{4}(t), \\ \lambda'_{4}(t) &= \mu_{0}\lambda_{4}(t) \end{aligned}$$

With intersection (terminal) axiom for i = 1,2,3,4 $\lambda_I(T) = 0,(19)$

Next, the optimal control parameters u_1^* (t) and u_2^* (t) are as follows

$$u_{1}^{*}\left(t\right) = \max\left\{\min\left\{\frac{(\lambda_{1}(t) - \lambda_{4}(t))S^{*}(t)}{\xi_{4}}, 1\right\}, 0\right\}$$
(20)
And:
$$u_{2}^{*}\left(t\right) = \max\left\{\min\left\{\frac{(\lambda_{2}(t) - \lambda_{4}(t))A^{*}(t) + (\lambda_{3}(t) - \lambda_{4}(t))B^{*}(t)}{\xi_{5}}, 1\right\}, 0\right\}$$
(21)

Proof (18) can be derived from (15) by taking the condition of intersection stated in (19) as a basis for the axiom (19) can take from the condition of intersection given in (17). Take the derivative of H and assess $\frac{\partial H}{\partial u_1} = 0$ and $\frac{\partial H}{\partial u_2} = 0$ to determine the best solution. In addition, we use the condition for maximal (16) to obtain (20)–(21), which established the integral theorem in our case.

The analytical evaluation showed that the optimal values for state and control variables could be calculated from the optimal system, which has state (4) and equation of the adjoint variables (18), along with boundary conditions (5) and (19), and which fulfills properties of optimal control parameters (u_1^*, u_2^*) , and this was confirmed experimentally (20) and (21).

4.2. Optimal Control Strategy for Stochastic System (Model 1)

Stochastic optimum control for systems focuses on this section (1). Stochastic control system based on model (1)'s identical two control parameters (u_1 and u_2) and stochastic perturbation

$$dS(t) = \left[A - \frac{\theta S(t)A(t)}{N} - (\mu_0 + \delta + u_1(t))S(t) \right] dt + \Phi_1 S(t) dW_1(t) dA(t) = \left[\frac{\theta S(t)A(t)}{N} - (\mu_0 + \sigma + \gamma_1)A(t) - u_2(t)A(t) \right] dt + \Phi_2 A(t) dW_2(t) (22)
$$dB(t) = \left[- (d_1 + \gamma_2 + \mu_0)B(t) + \sigma A(t) - B(t)u_2(t) \right] dt + \Phi_3 B(t) dW_3(t), dR(t) = \left[(\delta + u_1(t))S(t) - \mu_0 R(t) + \gamma_2 B(t) + u_2(t)B(t) + \gamma_1 A(t) + u_2(t)A(t) \right] dt + \Phi_4 R(t) dW_4(t),$$
With some of the initial approximation:$$

With some of the initial approximation:

$$R(0) > 0, B(0) \ge 0, A(0) \ge 0, S(0) > 0,$$
 (23)
To help our readers, we've included the vector
below:

$$u(t) = [u_{2}(t), u_{1}(t)]'x(t) = [x_{4}(t), x_{3}(t), x_{2}(t), x_{1}(t)]'$$

and: (24)

$$dx(t) = g(x(t))dw(t) + f(x(t), u(t))dt, \quad (25)$$

with initial approximation as:

 $x_0 = [x_4(0), x_3(0), x_2(0), x_1(0)]' = x(0),$ (26) where f and g are the vectors defined as:

$$f_1\left(x\left(t\right), u\left(t\right)\right) = \left[\Lambda - \frac{\theta S(t)A(t)}{N} - (\mu_0 + u_1(t))S\left(t\right)\right] dt + \Phi_1 S\left(t\right) dW_1\left(t\right)$$

$$f_2\left(x\left(t\right), u\left(t\right)\right) = \left[\frac{\theta S(t)A(t)}{N} - (\mu_0 + \sigma + \gamma_1)A\left(t\right) - u_2\left(t\right)A\left(t\right)\right] dt + \Phi_2 A\left(t\right) dW_2\left(t\right)$$
(27)

$$f_{3}(x(t), u(t)) = [\sigma A(t) - (\mu_{0} + d_{1} + \gamma_{2})B - u_{2}(t)B(t)]dt + \Phi_{3}B(t)dW_{3}(t), f_{4}(x(t), u(t)) = [u_{2}(t)B(t) + \gamma_{1}A(t) + u_{2}(t)A(t) + \gamma_{2}B(t) + u_{1}(\Box)S(t) - \mu_{0}R(t)]dt + \Phi_{4}R(t)dW_{4}(t),$$

 $g_1 = \Phi_1 S, g_2 = \Phi_2 A, g_3 = \Phi_3 B, g_4 = \Phi_4 R$ The following cost-quadratic-functional considerations are considered:

$$G\left(A,B,u_{1},u,u_{2}\right) = \frac{E}{2} \left\{ \int_{0}^{y_{1}} \left(X_{1}S + X_{2}A + X_{3}B + \frac{Y_{1}}{2}u_{1}^{2} + \frac{Y_{2}}{2}u_{2}^{2}\right) dt + \frac{k_{1}}{2}S^{2} + \frac{k_{2}}{2}A^{2} + \frac{k_{3}}{2}B^{2} + \frac{k_{4}}{2}R^{2} \right\},$$
(28)

Here X1, X2, X3, Y1, Y2, k4, k3, k2, and k1 are constants and greater than 0.

In this section, we will figure out what the optimal solution is $u^*(t) = (u_2^*(t), u_1^*(t))$

$$J(u) \ge G(u^*), \forall u \in U$$

$$U = U$$
(29)

U is the controlling admissible set given as:

$$\{u_i(t) : u_i(t) \in [0, u_i^{max}], \forall u_i \in L^2[0, t_f] t \in (0, t_f], i = 1, 2\} = U$$
(30)

where $u_i^{max} > 0$ at i = 1, 2 are not changeable. The next

step is to use the stochastic maximum rules, which we will do on the first try. For this, we take "Hamiltonian" $H_m(x, u, m, n)$ as:

$$H(x,u,m,n) = \langle f(x,u),m \rangle - l(x,u) + \langle g(x),n \rangle$$
(31)

where $\langle ... \rangle$ represent the inner product space due to "Euclidean" and m = [m₄, m₃, m₂, m₁] ' and n = [n₄, n₃, n₂, n₁] ' represent the respective adjoint vectors. The maximization principle is as follows:

$$dx^{*}\left(t\right) = \frac{\partial H(x^{*}(t), u^{*}(t), m, n)}{\partial m} dt + g(x^{*}(t)) dW\left(t\right)$$

$$dm^{*}\left(t\right) = -\frac{\partial H(x^{*}(t), u^{*}(t), m, n)}{\partial x} dt + n\left(t\right) dW\left(t\right)$$
(32)
(33)

 $Hm(x * \max_{0 \le x \le 1} xe^{-x^2}(t), u * (t), m, n) = \max_{U \in U} H_m u(x * (t), u * (t), m, n)$ (34)

 $x^*(t)$ is the path of x's optimality (t). The beginning and the end (BCs) conditions of Eqs. (32) and (33) are

$$x_0 = x^*(0), (35)$$

$$-\frac{\partial h(x^*(t_f))}{\partial x} = m(t_f)$$
(36)

respectively. As Eq. (34) shows that the optimality

variable $x^{*}(t)$ is an operator of q(t), p(t) and $x^{*}(t)$, this implies that

 $u * (t) = \phi(m, n, x *),$ (37)

 ϕ can be computed by Eq. (34). Thus, Eqs. (32) and (33) can be written as

$$dx * (t) = \frac{\partial H(x*(t),u*(t),m,n)}{\partial m} dt + g(x*(t)) dW(t)$$
(38)

$$dm(t) = -\frac{(\partial H(x^*(t),u^*(t),m,n))}{\partial x}dt + b(t)dW(t) \quad (39)$$

So, the "Hamiltonian" is:

 $H = \left(X_{1}S + X_{2}A + X_{3}B + u_{1}^{2}\frac{Y_{1}}{2} + u_{2}^{2}\frac{Y_{2}}{2} + S^{2}\frac{k_{1}}{2} + B^{2}\frac{k_{3}}{2} + A^{2}\frac{k_{2}}{2} + R^{2}\frac{k_{4}}{2}\right) + m_{1}\left(\Lambda - \frac{\theta S(t)A(t)}{N} - (\mu_{0} + \delta + u_{1}(t))S(t)\right) + m_{2}\left(\frac{\theta S(t)A(t)}{N} - (\mu_{0} + \sigma + \gamma_{1})A(t)\right) + u_{2}\left(t\right)A(t)\right) + m_{3}(\sigma A(t) - B(\mu_{0} + d_{1} + \gamma_{2})) + m_{4}\left(u_{2}\left(t\right)B(t) + \gamma_{1}A(t) + u_{2}\left(t\right)A(t) + \gamma_{2}B(t) + \left(u_{1}\left(t\right) + \delta\right)S(t) - R(t)\mu_{0}\right) + \Phi_{1}Sn_{1} + \Phi_{2}An_{2} + \Phi_{3}Bn_{3} + \Phi_{4}Rn_{4}.$ (40)

It follows from the stochastic maximum principle that

$$dm^{*}\left(t\right) = -\frac{\partial H(x^{*}, u^{*}, m, n)}{\partial x}dt + n\left(t\right)dW\left(t\right)$$
(41)

Variables are gathered. Piece-wise continuity and Lebesgue measurability will be examined for the

objective function's appropriate set of control variables. We will create an optimal control and state sequence that can be convergent in the feasible field for the boundedness of all feasible controls. Such a role identifies a controlling factor:

$$\dot{m_1(t)} = -X_1 + (m_1(t) - m_2(t)) \frac{\theta A^*(t)}{N} + m_1(t) (\mu_0 + \delta + u_1^*(t)) - m_4(t) (u_1^*(t) + \delta) + \Phi_1 n_1,$$

$$\dot{m_2(t)} = -X_2 + (m_1(t) - m_2(t)) \frac{\theta S^*(t)}{N} + m_2(t) (\mu_0 + \sigma + \gamma_1) - m_3(t)\sigma - m_4(t)\gamma_1, + (m_2(t) - m_4(t))u_2^*(t) + \Phi_2 n_2$$
(42)

$$m'_{3}(t) = -X_{3} + (m_{3}(t) - m_{4}(t))u * 2(t) + m_{3}(t)(\mu_{0} + d_{1} + \gamma_{2}) - \gamma_{2}m_{4}(t) + \Phi_{3}n_{3}, m'_{4}(t) = m_{4}(t)\mu_{0} + \Phi_{4}n_{4},$$

Along with the auxiliary starting and ending constrains as:

$$S^{*}(0) = \widehat{S}, \ A^{*}(0) = \widehat{A}, \ B^{*}(0) = \widehat{B}, \ R^{*}(0) = \widehat{R}, \ p(t_{f}) = -\frac{\partial h(x^{*}(t_{f}))}{\partial x},$$
(43)

and:

 $h(S, A, B, R,) = \frac{k_1}{2}S^2 + \frac{k_1}{2}A^2 + \frac{k_1}{2}B^2 + \frac{k_1}{2}R^2 \quad (44)$ where $m_1(t_f) = -k_1S, m_2(t_f) = -Ak_2, m_3(t_f) = -Bk_3, m_4(t_f) = -Rk_4.$ On derivative of

Hamiltonian equation with respect to u1, u2 we obtain the optimal control u_1^* and u_2^* as follows

$$u_{1}^{*} = max \left\{ \min \left\{ \frac{1}{Y_{1}} (m_{1} - m_{4})S^{*}, 1 \right\}, 0 \right\}$$

$$u_{2}^{*} = max \left\{ \min \left\{ 1, \frac{(m_{2} - m_{4})A^{*} + (m_{3} - m_{4})B^{*}}{Y_{2}} \right\}, 0 \right\} (45)$$

Plugging the control parameter into the dynamical system helps achieve the goal of optimality. A mathematical model is an equation or system of differential equations that may be controlled, as Eq (27). It is necessary to construct the goal function according to Eq. (28)'s instructions. It is important to keep the balance between low-cost and opposing variables intact until the end of the process. The choice of the objective function directly impacts the optimality; hence, attention must be paid when selecting it. Weight should be given the most important word if there are more than two regulating goal functions. Before we can apply the Pontryagin Maximum Rules, we must check the existence and compactness of optimal control that can maximize/minimize the objective function subject to some starting point and differential equations or systems of differential equations that optimize the "Hamiltonian" by some points. To begin, the Hamiltonian is defined as follows:

Hamiltonian

= (integrableobjectivefunctional)

+ (*RHSofDifferentialsystem*)(*adjoint*)

Control strategies that maximize the "Hamiltonian" for u at u* are a fundamental component of an optimal control method. Derivatives for a state variable can be applied to the adjoint equation to get the final condition.

4.3. Simulations Based on Numbers

Systems (1) and (2) must be approximated to validate our analytical results (2). Simulations can be drawn from qualitative aspects and the parameters chosen in an epidemiologically plausible manner. The computed model (1) is presented as follows using the stochastic Runge–Kutta Method (Table 1):

Table 1 Descriptions of peremeters applied in model

	Table 1 Descriptions of parameters applied in model
Λ	The rate of recruitment
μ_0	Natural death rate
θ	The rate of interaction between an infected and healthy
	population
γ_1	The rate at which acutely infected individuals are getting
	a recovery
γ_2	The recovery rate from chronically infected cases
d_1	The death rate due to disease
δ	The vaccination rate of disease
σ	Transferring rate of acute to chronically infected class
	• • •

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where $\zeta k, i(k = 1, 2, 3, 4)$, N(0,1) and time increment are four independent Gaussian random variables. For $\Delta t > 0$, see Table 2.

Table 2 Values of parameters							
Parameters	Value	Source	Parameters	Value	Source		
Λ	98.75	Estimated	Φ_2	0.400	Estimated		
μ_0	0.18	Estimated	Φ_3	0.250	Estimated		
θ	0.50	Estimated	Φ_4	0.65	Estimated		
γ_1	0.08	Estimated	u_1	0.4460	Estimated		
Y2	0.02	Estimated	u_2	0.2250	Estimated		
d ₁	0.25	Estimated	S(0)	49	Estimated		
δ	0.04	Estimated	A(0)	52	Estimated		
σ	0.19	Estimated	B(0)	22	Estimated		
Φ_1	0.77	Estimated	R(0)	0	Estimated		





Fig. 1 Plots of S(t), A(t), and B(t) populations with and without controls for the stochastic and deterministic model approaches: (a)
S(t) – with and without control in the deterministic model approach; (b) S(t) – with and without control in the stochastic model approach; (c) A(t) – with and without control in the deterministic model approach; (d) A(t) – with and without control in the stochastic model approach; (e) A(t) – with and without control in the stochastic model approach; (e) A(t) – with and without control in the stochastic model approach; (f) A(t) – with and without control in the stochastic model approach; (f) B(t) – with and without control in the deterministic model approach





Fig. 2 Plot of R(t) populations with and without controls for the stochastic and deterministic models: (a) R(t) – with and without control in the deterministic model approach; (b) R(t) – with and without control in the stochastic model approach



Fig. 3 Plots of u1(t) and u2(t) for the stochastic and deterministic model approaches(a) Plots of u(t)1 and u(t)2, in deterministic model approach; (b) Plots of u(t)1 and u(t)2 in the stochastic model approach

5. Conclusion

5.1. Main Findings of the Present Study

Fig. 2 has been used to develop a stochastic virus system. Controlled and uncontrolled R(t) populations are shown on the graph that is Fig. 3. In a stochastic approach, the deflections in environmental noise are referred to as Φ i, where II is one of the following values: 1, 2, 3, 4, 5. This deflection affects both the stability of the population and the reduction of the epidemic. A stochastic method was used to investigate the optimum control mechanism for avoiding and managing the DISEASE pandemic in this situation. The results were published in science. When used in

optimal control methods, the optimum control technique based on this approach gives a superior approximation between stochastic and deterministic systems. On the right are the calculated solutions for optimal control in techniques 1 and 2. On the left are the retrieved individuals; on the right are the first three containers of the appraisal. On the right are the premeditated solutions for optimal control in techniques 1 and 2.

Moreover, Fig. 3 depicts the behavior of the best stochastic control of the model (1). It is possible to notice the difference here as well. Alternatively, the number of vulnerable, acute, and chronic cases has dropped, while the number of recovery cases has grown.

5.2. Comparison with Other Studies

Even though epidemic models have been studied for a long time, control engineers have only just joined the picture. The construction and study of epidemic models have thus generated a large amount of effort, but considerably less has provided the required understanding and equipment on how to regulate these processes successfully. Several spreading processes on complex networks can be modeled using the same concepts and methods. For example, the transmission of a disease across a population, the acceptance of a new product in the marketplace, the possibility of a computer virus transmitting over the World Wide Web, etc. The word "individual" may refer to any one of these four types of entities. This paper serves as an introduction to the latter. Newcomers to the topic of spreading processes on complicated networks can benefit from a brief report we have put together. That, however, is patently false. It implies that the deterministic approximations we investigate for any given population with a stochastic model are approximations. As a result, it is natural to wonder about the precision with which they describe their stochastic counterparts. In this effort, a better approximation is accomplished between stochastic and deterministic systems in the optimum control method. To better understand the stochastic model, a numerical solution was done.

5.3. Implication and Explanation of Findings

The control form of the stochastic model was investigated in the current study. As with the deterministic technique, the inquiry was carried out in the presence of two control factors, denoted by the letters u_1 and u_2 . As a result, we investigate the strategies of HBV model prevention by employing the two control factors listed below: It is represented by the control variable $u_1(t)$, which is utilized to lower the vulnerable population through vaccination. $u_2(t)$ represents the control variable treatment of hepatitis B infected patients. This control aims to minimize the rate of hepatitis B transmission. This control variable Jamali et al. A Discrete Study on Stochastic Epidemic Models with the Optimal Control Policies and Its Analysis, Vol. 49 No. 4 April 2022 342

aims to lessen the number of infected persons in the population. In addition, we will reduce other variables that may be contributing to the increase in infections. In our plan, we will employ the "Hamilton–Jacobi– Bellman" techniques for stochastic approaches and the usual techniques for deterministic approaches, which are only used by a few researchers now. In addition, we were interested in constructing mathematical and statistical models and other types of models.

5.4. Strengths and Limitations

The numerical simulation of a stochastic optimum control issue allows for evaluating the feasibility of a certain control strategy. We believe that our method provides a feasible approximation alternative to a formal approach to numerical simulation of the stochastic optimum control issue, which is significantly more difficult and labor demanding. It is a disadvantage of the procedure described above that it does not consider the system's present condition. Because of this, nodes that do not have any diseased neighbors may have their cure rates increased. The great amount of research on the difficulties covered in this article has resulted in a large body of work. However, there are still many fascinating control problems to be addressed, particularly in networked dynamics. There is still more work to be done to fully harness the potential of these findings and have a meaningful impact on society; this is especially true in terms of knowing how to govern these processes on complicated networks successfully.

5.5. Conclusion, Recommendation, and Future Research

The optimal control stochastic model has been solved numerically for better understanding. The stochastic RK technique is used to support the analytical outcomes in this study. We used numerical simulations of the researched models to verify our theoretical findings. The other disease can be prevented and controlled using the same method we studied. For example, stochastic infectious models can be used to explore the effect of time on the epidemic's behavior. This article focused on illness and epidemics. However, it should be noted that the same mathematical methods and conclusions may be used for a wide range of other spreading processes, such as information propagation through social media, malware spreading on the World Wide Web, and viral marketing. As more real-world data become available, we will be able to further develop the model by including the safety, efficiency, and universality of the vaccine in daily life. That will allow us to broaden further the stochastic system proposed in this paper by including a vaccinated class in the stochastic system. Further considering the impacts of infectious illness treatment, immunization, media attention, and other controls on the linked optimum control issues is the next stage in the model's development.

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