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CONTROL STRATEGIES APPLIED TO A STOCHASTIC DISEASE MODEL WITH TERM-TIME FORCING

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ABSTRACT. A SIR model with time-varying contact rate and stochastic perturbations is studied. The contact rate is modelled as a piecewise constant by assuming that it is a switching parameter. Extrinsic noise is considered by assuming that there are white noise perturbations in the transmission of the disease. Both time-constant control and impulsive control are applied to the SIR model and some threshold conditions are established which guarantee the disease is eradicated in mean square.

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1. Introduction

Mathematical epidemiology is useful for attempting to understand how a disease spreads in a population and, moreover, for estimating vaccination levels necessary to eradicate a disease [18]. In modeling the spread of a disease, it is physically reasonable to suppose there are external, environmental forces which partially drive the spread of a disease, and hence affect a model's parameters. Indeed, since real life is full of randomness and stochasticity, there are real benefits to be gained in using a stochastic model formulation for the spread of an infectious disease [8, 32]. This is particularly true when modeling biological phenomena such as internal HIV viral dynamics [8]. Further, it is true that noise can induce non-trivial effects in physical and biological systems by modifying the behaviour of the corresponding deterministic evolution of the system [29].

There has been some work done in the literature on stochastic epidemic models with white noise perturbations. For example, Tornatore et. al [29] investigated the stability of the disease-free equilibrium of a stochastic SIR model with distributed time delay. Beretta et. al [5] analyzed white noise stochastic perturbations around positive endemic equilibria of epidemic models with time delays influenced by probability. In [32], a two-group SIR model with white noise stochastic perturbations around its positive endemic equilibrium is introduced and studied. Carletti [6] investigated the stability properties of a stochastic model for phage-bacteria interaction in open marine environment. Dalal et. al [7, 8] studied a model of AIDS and HIV by analyzing

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condom use in a stochastic epidemic model with parameter perturbations, motivated by the inherent randomness associated with HIV and AIDS.

Traditionally, mathematical infectious disease models assume that the contact rate (the average number of contacts between individuals that are sufficient for transmission of the disease) is a constant in the model. However, studies have shown that the contact rate varies seasonally, as it is higher during the winter months (when individuals group together more often) compared to the summer months [19]. Periodicity in disease transmission is apparent in the spread of many diseases, such as measles, chickenpox, mumps, rubella, poliomyelitis, pertussis, and influenza [17]. There are many causes of seasonality in the spread of a disease, for example, changes in the abundance of vectors, changes in host behaviour, and changes in host immunity [9, 10, 14]. Seasonality is usually incorporated into epidemic models by assuming that the contact rate is a smoothly-varying parameter, for example, see [2, 3, 4, 13, 14, 19, 22, 24, 25, 27] and the references therein. An alternative approach, first proposed by Schenzle in [26], and since studied in, for example, [11, 20, 21], is to assume that the contact rate experiences abrupt changes in time, which is referred to as term-time forcing. This is reasonable, for example, for a childhood disease spreading among students who experience holiday breaks and has been shown to more accurately match the transmission data in some cases [11].

The objective of this paper is to study infectious disease models with time-varying contact rates and stochasticity. Stochasticity is added to epidemic models by considering multiplicative white noise, motivated by randomness in the transmission of the disease due to external forces. Time-varying contact rates are introduced into infectious disease models by assuming that the contact rate is a switching parameter, which is piecewise constant. Based on these assumptions, an SIR model with term-time forcing and stochastic perturbations is considered. A constant treatment and pulse treatment strategy are applied to the model and analyzed. For the pulse treatment scheme, the inter-pulse period does not necessarily have to equal the period of the seasonal changes in the model. Some threshold criteria guaranteeing eradication of the disease in the mean square are given. To the best of the authors' knowledge, there have been no studies on stochastic epidemic models with switching parameters and control schemes. In this paper, some switched systems techniques found in [15, 16, 21] are used to prove the threshold conditions found.

This paper is organized as follows: in Section 2, a constant treatment scheme is applied to an SIR model with term-time forcing and stochastic perturbations. A pulse treatment scheme is applied to the same SIR model in Section 3. Some threshold conditions are established for both models which guarantee that the number of infected converges to zero with probability one. Simulations are given in Section 4

to illustrate the theorems established in this paper. Finally, some conclusions are drawn and future directions are given in Section 5.

2. **Constant Treatment Model**

Split the population into three distinct groups: the susceptible, S, who are healthy and able to contract the disease; the infected, I, who are infected and able to transmit the disease; and the recovered, R, who have recovered from the disease (either naturally or by treatment) and gained permanent immunity. Assume that the birth rate $\mu > 0$ is constant (which is equal to the natural death rate) and all individuals are born healthy. Assume that individuals recover from the disease naturally at a rate $g > 0$. Assume that the incubation period of the disease is negligible and that individuals in the population mix homogeneously.

Most developed countries have used some type of time-constant (also called cohort) immunization program with varying degrees of success [1]. For example, many areas in the Western world recommend vaccination doses at 15 months and six years of age to combat measles [28]. Consider the strategy of time-constant control, where a portion $0 \leq \theta \leq 1$ of the infected population is being treated continuously in time. Note that $\theta = 0$ is uninteresting and, due to treatment failure, $\theta = 1$ is physically unrealistic. Assume that once treated, an infected individual moves to the recovered class with permanent immunity.

Motivated by the discussion in Section 1 on seasonal variations, assume that the contact rate is a switching parameter $\beta_{i_k} > 0$, where the index $i_k \in \aleph = \{1, 2, \ldots, m\}$ changes values according to the deterministic switching rule $\sigma(t) : [t_{k-1}, t_k) \to \aleph$, where $k = 1, 2, \ldots$, which is a piecewise constant function. Under this construction, β_{i_k} takes on a certain value in each time interval $(t_{k-1}, t_k]$. The switching times satisfy $t_0 = 0 < t_1 < \cdots < t_k < \cdots$ with $t_k \to$ as $k \to \infty$. Denote the class of switching rules which satisfy these properties by $\mathcal{S}$. Assume that the incidence rate of the disease, defined as the average number of new cases per unit time, is proportional to the number of infected and susceptible present and takes the form $\beta_{i_k} SI/N$, where the total population $N = S + I + R$. The derivation comes from a physical argument: the incidence rate is the number of average contacts multiplied by the total number of susceptibles and the probability the contact is with an infected.

Assume that, due to external forces in the model causing stochastic perturbations in the transmission of the disease, the contact rate is perturbed to $\beta_{i_k} + cn(t)$, where $c > 0$ is a constant, and $n(t)$ is Gaussian white noise. Then the disease is transmitted with the incidence rate $[\beta_{i_k} + cn(t)]SI/N$. The motivation is that, from a practical perspective, the contact rate is estimated as an average value plus some error which is assumed to follow a normal distribution. This is the technique of parameter perturbation, which is standard in stochastic population modeling [7, 8].

Taking these assumptions into account, the switched SIR model with stochastic perturbations and constant treatment is given by,

$$\begin{cases} dS = \left[\mu N - \beta_{i_k}\dfrac{SI}{N} - \mu S\right]dt - c\dfrac{SI}{N}dB, \\ dI = \left[\beta_{i_k}\dfrac{SI}{N} - gI - \theta I - \mu I\right]dt + c\dfrac{SI}{N}dB, \\ dR = [gI + \theta I - \mu R]dt, \end{cases} \tag{2.1}$$

for $t \in (t_{k-1}, t_k]$. Here $i_k \in \aleph$ is governed by a switching rule σ, dB is a Wiener process (or Brownian motion), under the Itô interpretation, defined on a complete probability space $(\Omega, \mathcal{F}, \mathbb{P})$ with a filtration $\{\mathcal{F}_t\}_{t\geq 0}$ satisfying the usual conditions [23]. The initial conditions are $S(0) = S_0 > 0$, $I(0) = I_0 > 0$ and $R(0) = R_0$ such that $S_0 + I_0 + R_0 = 1$. The positivity conditions are to make the problem physically interesting. System (2.1) has a single disease-free equilibrium point $\bar{\mathbf{Q}} = (\bar{S}, \bar{I}, \bar{R}) = (1, 0, 0)$, where there are no infected individuals present in the population.

Following the work of Dalal et. al [7, 8], where the authors have proved existence and non-negativity of solutions of stochastic epidemic models, the following theorem on positivity and existence of solutions may be presented.

Theorem 2.1. *For any initial conditions (S_0, I_0, R_0) with $S_0 > 0$, $I_0 > 0$, and $S_0 + I_0 + R_0 = 1$, there exists a unique solution to (2.1) on $t \geq 0$ and the solution remains in $\Omega_{SIR} = \{(S, I, R) \in \mathbb{R}^3_+ |\ S + I + R = 1\}$ with probability one for all $t \geq 0$ almost surely.*

Proof. Note that it is apparent from integration the equations in (2.1) and adding them together that $S+I+R = 1$ almost surely. Then the proof can be broken into two parts: first we prove global existence of solutions and then we prove non-negativity of each variable almost surely.

To prove non-negativity, we follow the procedure of [7, 8]. Note that the coefficients of (2.1) are piecewise Lipschitz continuous on any interval $(t_{k-1}, t_k]$, and hence there exists a unique solution for all $(S_0, I_0, R_0) \in \mathbb{R}^3_+$ for $t \in [0, \tau_e)$ where τ_e is the explosion time (see [12]). Then it is sufficient to show $\tau_e = \infty$ almost surely in order to guarantee global existence of the solution. Let $N_0 \geq 0$ be sufficiently large so that $1/N_0 \leq S_0 \leq N_0$, and $1/N_0 \leq I_0 \leq N_0$. For each integer $N \geq N_0$, define the stopping time

$$\tau_k = \inf\{t \in [0, \tau_e) : S(t) \notin (1/N, N) \text{ or } I(t) \notin (1/N, N) \text{ or } R(t) \notin (1/N, N)\}$$

and note that τ_k is increasing as $k \to \infty$. Set $\tau_\infty = \lim_{k\to\infty} \tau_k$ and note that $\tau_\infty \leq \tau_e$ almost surely. If $\tau_\infty = \infty$ almost surely then $\tau_e = \infty$ almost surely and hence $(S, I, R) \in \mathcal{R}^3_+$ almost surely for all $t \geq 0$.

We prove the claim $\tau_\infty = \infty$ by contradiction: if not, then there exists $T > 0$ and $0 < \epsilon < 1$ such that $\mathbb{P}[\tau_\infty \leq T] > \epsilon$. Thus there exists $N_1 \geq N_0$ such that

$$\mathbb{P}[\tau_k \leq T] \geq \epsilon, \quad \forall N \geq N_1. \tag{2.2}$$

Consider the auxiliary function $V = S + 1 - \ln S + I + 1 - \ln I + R + 1 - \ln I$, which is always positive. By Itô's formula,

$$\begin{aligned} dV = &[4\mu - \mu S - \mu I - \mu/S + \beta_{i_k} I - \beta_{i_k} S + g + \theta]dt \\ &[-\mu R - (g+\theta)I/R + c^2(S^2 + I^2 + R^2)/2]dt + c(I - S)dB. \end{aligned}$$

For $t^* \leq T$, define $\eta = \min(t^*, \tau_k)$. Then for $t \in [0, \eta]$,

$$\begin{aligned} dV &\leq [(\beta_{i_k} + g + \theta)I + 4\mu + g + \theta + c^2(S + I + R)^2/2]dt + c(I - S)dB, \\ &= [(\beta_{i_k} + g + \theta)I + 4\mu + g + \theta + c^2/2]dt + c(S - I)dB. \end{aligned}$$

Use

$$I \leq 2(I + 1 - \ln I) - (4 - 2\ln 2) \leq 2(S + 1 - \ln S + I + 1 - \ln I) = 2V.$$

Then,

$$\begin{aligned} dV &\leq [2(\beta_{i_k} + g + \theta)V + 4\mu + g + \theta + c^2/2]dt + c(S - I)dB, \\ &\leq [\lambda(1 + V)]dt + c(S - I)dB, \end{aligned}$$

where $\lambda = \max\{2(\beta_1 + g + \theta), \ldots, 2(\beta_m + g + \theta), c^2/2 + 4\mu + g + \theta\}$. Thus,

$$\begin{aligned} \int_0^\eta dV(S(t), I(t), R(t)) \leq &\int_0^\eta \lambda[1 + V(S(t), I(t), R(t))]dt \\ &+ \mathbb{E}\left[\int_0^\eta \lambda[1 + V(S(t), I(t), R(t))]dt\right], \end{aligned}$$

which implies,

$$\begin{aligned} \mathbb{E}\left[V(S(\eta), I(\eta), R(\eta))\right] &\leq V_0 + \mathbb{E}\left[\int_0^\eta \lambda[1 + V(S(t), I(t), R(t))]dt\right], \\ &\leq V_0 + \lambda t^* + \lambda \mathbb{E}\left[\int_0^\eta V(S(t), I(t), R(t))dt\right], \\ &\leq V_0 + \lambda T + \lambda \mathbb{E}\left[\int_0^{t^*} V(S(\eta), I(\eta), R(\eta))dt\right], \\ &= V_0 + \lambda T + \lambda \int_0^{t^*} \mathbb{E}[V(S(\eta), I(\eta), R(\eta))]dt, \end{aligned}$$

where $V_0 = V(S_0, I_0, R_0)$. By Gronwall's inequality,

$$\mathbb{E}[V(S(\eta), I(\eta), R(\eta))] \leq [V(S_0, I_0, R_0) + \lambda T]\exp(c_2 T). \tag{2.3}$$

Set $\Omega_N = \{\tau_N \leq T\}$ for $N \geq N_1$. Then $\mathbb{P}(\Omega_N) \geq \epsilon$ by equation (2.2). Note that for all $B \in \Omega_N$: $S(\tau_N, B) = N$ or $1/N$; or $I(\tau_N, B) = N$ or $1/N$; or $R(\tau_N, B) = N$ or

$1/N$. Hence $V(S(\tau_N, B), I(\tau_N, B), R(\tau_N, B)) \geq N + 1 - \ln N$ or $(1/N) + 1 - \ln(1/N)$. That is,

$$V(S(\tau_N, B), I(\tau_N, B), R(\tau_N, B)) \geq \min\{N + 1 - \ln N, 1/N + 1 + \ln N\}.$$

Then equations (2.2) and (2.3) imply

$$(V(S_0, I_0, R_0) + \lambda T) \exp(\lambda T) \geq E[1_{\Omega_N}(B) V(S(\tau_N, B), I(\tau_N, B), R(\tau_N, B)), \tag{2.4}$$

$$\geq \epsilon(\min\{N + 1 - \ln N, 1/N + 1 + \ln N\}), \tag{2.5}$$

where 1_{Ω_N} is the indicator function of Ω_N. Let $N \to \infty$ then $c_3 = (V(S_0, I_0, R_0) + \lambda T) \exp(\lambda T)$ satisfies $\infty > c_3 = \infty$ which is a contradiction, hence $\tau_\infty = \infty$.

To show that solutions remain positive: assume that $S_0 > 0$ and $I_0 > 0$, which implies that $S(\Delta t) > 0$ and $I(\Delta t) > 0$ almost surely for Δt small positive. By Itô's formula,

$$d \ln I = [\beta_{i_k} S - \mu - g - \theta - c^2 (SI)^2/2] dt + cSI dB.$$

Hence

$$I = I_0 \exp\left\{ \int_0^t [\beta_{\sigma(u)} S(u) - \mu - g - \theta - c^2 S^2(u) I^2(u)/2] du + \int_0^t cS(u) I(u) dB \right\}.$$

Similarly,

$$d \ln S = [\mu/S - \beta_{i_k} I - \mu - c^2 (SI)^2/2] dt + cSI dB.$$

Hence

$$S = S_0 \exp[\int_0^t [\mu/S(u) - \beta_{\sigma(u)} I(u) - \mu - c^2 S^2(u) I^2(u)/2] du + \int_0^t cS(u) I(u) dB].$$

Then, by changing the time origin to Δt and since solutions exist for all $t \geq 0$ almost surely, it follows, without loss of generality, that $I > 0$ and $S > 0$ for all $t \geq 0$ almost surely. Finally, since $R' = (g + \theta) I - \mu R$ and $I > 0$ almost surely, then it follows that $R \geq 0$ almost surely. □

Since there have been observed seasonal variations in the contact rate, consider a special switching rule that is periodic [15]: Assume that the switching rule σ satisfies $t_k - t_{k-1} = \tau_k$ with $\tau_{k+m} = \tau_k$. Assume that $\beta_i = \beta_k$ for $t \in (t_{k-1}, t_k]$ and that $\beta_{k+m} = \beta_k$, where $\omega = \tau_1 + \tau_2 + \cdots + \tau_m$ is one period of the switching rule. Denote the set of switching rules that satisfy this property by $\mathcal{S}_{\text{periodic}} \subset \mathcal{S}$.

Define

$$\mathcal{R}_\theta = \frac{\frac{1}{\omega} \int_0^\omega \beta_{\sigma(t)} dt + \frac{1}{2} c^2}{\mu + g + \theta}. \tag{2.6}$$

If $\sigma \in \mathcal{S}_{\text{periodic}}$ and $c = 0$ (no stochastic perturbations), then $\mathcal{R}_\theta$ is the basic reproduction number of the model, which follows from the epidemic literature (for example, see [3, 31]). The basic reproduction number has an important physical interpretation:

it represents the average number of secondary infections produced by one infected individual, during their infectious period, in a wholly susceptible population.

In order to investigate the eradication of the disease, the following definition is required.

Definition 2.2 ([23])**.** If x_k $(k \geq 1)$ and x are random variables belonging to L^p (p^{th} moment has finite value) and $\mathbb{E}[|x_k - x|^p] \to 0$, then x_k is said to converge to x in p^{th} moment.

The following theorem may now be proven using the basic reproduction number as a threshold.

Theorem 2.3. *If $\sigma \in \mathcal{S}_{periodic}$ and $\mathcal{R}_\theta < 1$ then the solution of system* (2.1) *converges in the second moment to the disease-free equilibrium $\bar{\mathbf{Q}}$.*

Proof. From equation (2.1), it follows from Itô's formula that

$$dI^2 = [I^2(\beta_{i_k} S - \mu - g - \theta) + \frac{1}{2} c^2 S^2 I^2]dt + 2cSI^2 dB.$$

Integrating gives,

$$\begin{aligned} I^2(t) &= I_0^2 + \int_0^t [(\beta_{\sigma(u)} S(u) - \mu - g - \theta) I^2(u) + \frac{1}{2} c^2 S^2(u) I^2(u)]du \\ &\quad + \int_0^t 2cS(u) I^2(u) dB(u), \\ &\leq I_0^2 + \int_0^t \lambda_{\sigma(u)} I^2(u) du + \int_0^t 2cS(u) I^2(u) dB(u), \end{aligned}$$

where $\lambda_i := \beta_i - \mu - g - \theta + \frac{1}{2} c^2$. Taking expected values, $\mathbb{E}[I^2(t)] \leq I_0^2 + \int_0^t \lambda_{\sigma(u)} \mathbb{E}[I^2(u)]du$, since $\mathbb{E}[\int_0^t 2cS(u) I^2(u) dB(u)] = 0$. It follows that

$$\mathbb{E}[I^2(t)] \leq I_0^2 \exp\left[\int_0^t \lambda_{\sigma(u)} du\right]. \tag{2.7}$$

Hence,

$$\mathbb{E}[I^2(\omega)] \leq I_0^2 \exp\left[\lambda_1 \tau_1 + \cdots + \lambda_m \tau_m\right] = I_0^2 \exp\left[\omega(\mu + g + \theta)(\mathcal{R}_\theta - 1)\right]. \tag{2.8}$$

Define $\eta := \exp\left[\omega(\mu + g + \theta)(\mathcal{R}_\theta - 1)\right]$, which satisfies $\eta < 1$ since $\mathcal{R}_\theta < 1$. Hence $\mathbb{E}[I^2(\omega)] \leq \eta I_0^2 < I_0^2$, and, similarly, $\mathbb{E}[I^2(h\omega)] \leq \eta \mathbb{E}[I^2((h-1)\omega)]$ for any integer $h = 1, 2, \ldots$ Hence,

$$\mathbb{E}[I^2(h\omega)] \leq \eta \mathbb{E}[I^2((h-1)\omega)] \leq \eta(\eta \mathbb{E}[I^2((h-2)\omega)]) \leq \cdots \leq \eta^h I_0^2.$$

Therefore the sequence $\{\mathbb{E}[I^2(h\omega)]\}_{h=0}^{\infty}$ converges to zero as $h \to \infty$. Further, it is apparent that $\mathbb{E}[I^2(t)]$ is bounded on each interval $((h-1)\omega, h\omega]$, and so it follows

that I^2 converges to zero in probability. From the equation for R, it is true that $R^2 = R_0^2 + \int_0^t 2R(u)[(\theta+g)I(u) - \mu R(u)]du$, then by Holdër's inequality,

$$\begin{aligned}\mathbb{E}[R^2] &= R_0^2 + \int_0^t [2(\theta+g)\mathbb{E}[I(u)R(u)] - 2\mu\mathbb{E}[R^2(u)]]dt,\\ &\leq R_0^2 + \int_0^t [2(\theta+g)\sqrt{\mathbb{E}[I^2(u)]\mathbb{E}[R^2(u)]} - 2\mu\mathbb{E}[R^2(u)]]dt.\end{aligned}$$

Since $\mathbb{E}[I^2] \to 0$, it follows that $\mathbb{E}[R^2]$ converges to zero. Finally, since $S = 1 - I - R$, it follows that $\mathbb{E}[S^2] = 1 + \mathbb{E}[I^2] + \mathbb{E}[R^2] - 2\mathbb{E}[I] - 2\mathbb{E}[R] + 2\mathbb{E}[IR]$. Again using Holdër's inequality it follows that $\mathbb{E}[S^2]$ converges to one. Thus the solution of system (2.1) converges in the second moment to the disease-free equilibrium $\bar{\mathbf{Q}}$. □

Note that the condition $\mathcal{R}_\theta < 1$ defines a critical control rate θ_{crit} such that $\theta > \theta_{\text{crit}}$ guarantees disease eradication in the mean square, where

$$\theta_{\text{crit}} = \frac{1}{\omega}\int_0^\omega \beta_{\sigma(t)}dt + \frac{1}{2}c^2 - \mu - g. \tag{2.9}$$

It is possible to prove a threshold theorem in the non-periodic case. Define the time-weighted average

$$\langle \mathcal{R}_\theta \rangle = \sup_{t\geq 0} \frac{\frac{1}{t}\int_0^t \beta_{\sigma(u)}du + \frac{1}{2}c^2}{\mu + g + \theta}. \tag{2.10}$$

Theorem 2.4. *If $\sigma \in \mathcal{S}$ and $\langle \mathcal{R}_\theta \rangle < 1$ then the solution of system* (2.1) *converges in the second moment to the disease-free equilibrium $\bar{\mathbf{Q}}$.*

Proof. Note that $\langle \mathcal{R}_\theta \rangle < 1$ implies that there exists $\epsilon > 0$ such that $(\frac{1}{t}\int_0^t \beta_{\sigma(u)}du + \frac{1}{2}c^2)/(\mu+g+\theta) \leq 1-\epsilon$, so that $\int_0^t(\beta_{\sigma(u)} + \frac{1}{2}c^2 - g - \mu - \theta)du \leq -\epsilon(\mu+g+\theta)t$. Therefore,

$$\int_0^t \lambda_{\sigma(u)}du \leq -\epsilon(\mu+g+\theta)t, \tag{2.11}$$

where $\lambda_\sigma := \beta_\sigma - g - \mu - \theta + \frac{1}{2}c^2$. Assume that i_k follows the switching rule $\sigma \in \mathcal{S}$, then from equation (2.7) and (2.11), it follows that $\mathbb{E}[I^2(t)] \leq I_0^2 \exp(-\epsilon(\mu+g+\theta)t)$ for all $t \geq 0$. Showing convergence of $\mathbb{E}[S^2]$ and $\mathbb{E}[R^2]$ to one and zero, respectively, follows similarly as in the proof of Theorem 2.3. □

3. **Pulse Treatment Model**

A time-constant control scheme has the effect of reducing the basic reproduction number but does not antagonize the underlying mechanics which spread the disease [19]. On the other hand, pulse vaccination strategies have recently gained prominence for their successful application to poliomyelitis and measles outbreaks in Central and South America [28]. The most notable example of a successful application of a vaccination program was the World Health Organization's global initiative

against smallpox, beginning in 1967 and leading to global eradication by 1977 [18]. A pulse control scheme is based on the idea of impulsively treating or vaccinating a fraction of individuals at certain points in time. Theoretical results show that pulse control schemes can achieve disease eradication at lower vaccination levels compared to conventional cohort immunization programs [1].

Assume that at the times $t = kT$, $k = 1, 2, \ldots$, a portion $0 \leq p \leq 1$ of the infected population is impulsively treated, causing them to move immediately to the recovered class. It is assumed that the time scale of the dynamics of the disease is much larger than the time scale of the treatment process, and so the instantaneous movement to the recovered class is a reasonable assumption. The model is then,

$$\begin{cases} dS = [\mu - \beta_{i_k} SI - \mu S]dt - cSIdB, & t \in (t_{k-1}, t_k], \\ dI = [\beta_{i_k} SI - gI - \mu I]dt + cSIdB, \\ dR = [gI - \mu R]dt, \\ S(t^+) = S(t), & t = kT, \\ I(t^+) = (1-p)I(t), \\ R(t^+) = R(t) + pI(t), \end{cases} \tag{3.1}$$

where $I(t^+) = \lim_{h \to 0^+} I(t+h)$, $i_k \in \aleph$, and with initial conditions $S(0^+) = S_0 > 0$, $I(0^+) = I_0 > 0$ and $R(0^+) = R_0$. As in the constant treatment model (2.1), it can be shown that $S, I, R \geq 0$ up until at least the first impulsive time. Further, the impulsive equations do not take make the solution negative and hence the meaningful physical domain is Ω_{SIR} almost surely. System (3.1) has the disease-free equilibrium $\bar{\mathbf{Q}} = (\bar{S}, \bar{I}, \bar{R}) = (1, 0, 0)$.

Define the lowest common multiple of the inter-pulse period and seasonal period by $z := lcm(T, \omega)$ and define

$$\mathcal{R}_p = \frac{\frac{1}{\omega}\int_0^\omega \beta_{\sigma(t)} dt + \frac{1}{2}c^2}{\mu + g - \frac{1}{T}\ln(1-p)}. \tag{3.2}$$

If $\sigma \in \mathcal{S}_{\text{periodic}}$ and $c = 0$ then $\mathcal{R}_p$ is the basic reproduction number of the model, which follows from the epidemic literature [30].

Theorem 3.1. *If $\sigma \in \mathcal{S}_{periodic}$ and $\mathcal{R}_p < 1$ then the solution of system (3.1) converges to the disease-free equilibrium $\bar{\mathbf{Q}}$ in the second moment.*

Proof. From equation (3.1), it follows from Itô's formula that

$$dI^2 = [I^2(\beta_{i_k} S - \mu - g) + \frac{1}{2}c^2 S^2 I^2]dt + 2cSI^2 dB.$$

Before the first impulse is applied,

$$I^2(T) = I_0^2 + \int_0^T [(\beta_{\sigma(t)}S(t) - \mu - g)I^2(t) + \frac{1}{2}c^2S^2(t)I^2(t)]dt + \int_0^T 2cS(t)I^2(t)dB(t),$$
$$\leq I_0^2 + \int_0^T \lambda_{\sigma(t)}I^2(t)dt + \int_0^T 2cS(t)I^2(t)dB(t),$$

where $\lambda_i := \beta_i - \mu - g + \frac{1}{2}c^2$. Taking expected values $\mathbb{E}[I^2(T)] \leq I_0^2 + \int_0^T \lambda_{\sigma(t)}\mathbb{E}[I^2(t)]dt$, since $\mathbb{E}[\int_0^T 2cS(t)I^2(t)dB(t)] = 0$. It follows that $\mathbb{E}[I^2(T)] \leq I_0^2 \exp\left[\int_0^T \lambda_{\sigma(t)}dt\right]$. Hence, $\mathbb{E}[I^2(T^+)] \leq I_0^2(1-p)\exp[\tau_1\lambda_1 + \cdots + \tau_m\lambda_m]$. Similarly,

$$\mathbb{E}[I^2(z^+)] \leq I_0^2(1-p)^{z/T}\exp\left\{\frac{\omega}{T}[\tau_1\lambda_1 + \cdots + \tau_m\lambda_m]\right\},$$
$$= I_0^2\exp\left\{\frac{z}{T}\ln(1-p) + \frac{z}{\omega}(\tau_1\lambda_m + \cdots + \tau_m\lambda_m)\right\},$$
$$= I_0^2\exp\left\{z(\mu + g - \frac{1}{T}\ln(1-p))(\mathcal{R}_p - 1)\right\}.$$

Define $\eta := \exp\left\{z(\mu + g - \frac{1}{T}\ln(1-p))(\mathcal{R}_p - 1)\right\} < 1$ since $\mathcal{R}_p < 1$. Hence $\mathbb{E}[I^2(z^+)] \leq \eta I_0^2 < I_0^2$. Since $\sigma \in \mathcal{S}_{\text{periodic}}$, it can be similarly shown that $\mathbb{E}[I^2(hz^+)] \leq \eta\mathbb{E}[I^2((h-1)z^+)]$ for any integer $h = 1, 2, \ldots$, and therefore,

$$\mathbb{E}[I^2(hz^+)] \leq \eta\mathbb{E}[I^2((h-1)z^+)] \leq \eta(\eta\mathbb{E}[I^2((h-2)z)]) \leq \cdots \leq \eta^h I_0^2.$$

Therefore the sequence $\{\mathbb{E}[I^2(hz^+)]\}_{h=0}^{\infty}$ converges to zero as $h \to \infty$. Since $\mathbb{E}[I^2(t)]$ is bounded for $0 \leq t \leq z$ and on each interval $((h-1)z, hz]$ for $h = 1, 2, \ldots$, it follows that $\mathbb{E}[I^2]$ converges to zero as $h \to \infty$. Therefore, I^2 converges to zero in probability. Showing convergence of $\mathbb{E}[S^2]$ and $\mathbb{E}[R^2]$ to one and zero, respectively, follows similarly as in the proof of Theorem 2.3. □

Note that the condition $\mathcal{R}_p < 1$ defines a critical control rate p_{crit} such that $p > p_{\text{crit}}$ guarantees disease eradication, where

$$p_{\text{crit}} = 1 - \exp\left[T\left(\mu + g - \frac{1}{\omega}\int_0^\omega \beta_{\sigma(t)}dt - \frac{1}{2}c^2\right)\right]. \tag{3.3}$$

4. **Simulations**

Consider the constant treatment model (2.1) with initial conditions $S_0 = 0.2$, $I_0 = 0.8$, and $R_0 = 0$. Assume that the switching rule takes the periodic seasonal form:

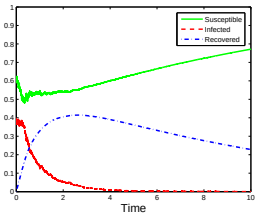
$$\sigma = \begin{cases} 1 & \text{if } t \in (k, k+0.25],\ k = 0, 1, 2, \ldots \\ 2 & \text{if } t \in (k+0.25, k+1], \end{cases} \tag{4.1}$$

which implies $\tau_1 = 0.25$, $\tau_2 = 0.75$, and $\omega = 1$. Take the parameters $\beta_1 = 2$ during the winter, $\beta_2 = 1$ during other seasons, $\mu = 0.1$, $g = 0.9$, and noise parameter $c = 0.5$. If $\theta = 0.4$ then $\mathcal{R}_\theta = 0.982$, and so, by Theorem 2.3, the solution converges

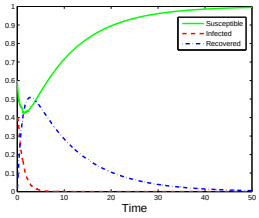
to the disease-free equilibrium of system (2.1) in the second moment. In this case, $\theta_{\text{crit}} = 0.375$ to guarantee disease eradication in mean square. See Figures 1a and 1b.

Consider the pulse treatment model (3.1) with the same initial conditions, periodic switching rule (4.1), and parameter values. Then treatment rate $p = 0.35$ with inter-pulse period $T = 2$ gives $\mathcal{R}_p = 0.961$, which implies the solution converges to the disease-free equilibrium of system (3.1) in the second moment, by Theorem 3.1. In this case, $p_{\text{crit}} = 0.313$ to guarantee disease eradication in mean square. See Figures 1c and 1d.

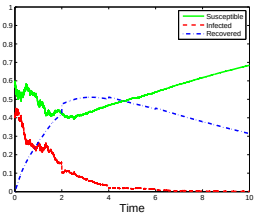
With the same parameter choices, the constant treatment scheme (2.1) requires a larger treatment rate compared to the pulse treatment scheme. This helps to contrast the constant scheme with the pulse scheme; it is often the case that a pulse scheme can achieve eradication using a lower value of p because of the existing trade-off between increasing the frequency of pulses and decreasing the pulse treatment rate requirement p. This is especially important when vaccine/treatment efficacy is considered, for example, the critical vaccination level for measles is approximately 94%, with vaccination efficacy approximately 95%. In order for a time-constant control program to be successful in this case, at least 99% of the population would need to be immunized, which is infeasible practically [18].



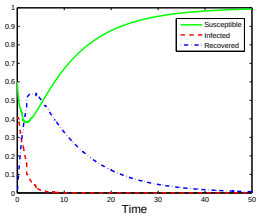
(a) Realization of (2.1) with $\theta = 0.4$.



(b) Ensemble average of (2.1).



(c) Realization of (3.1) with $p = 0.35$.

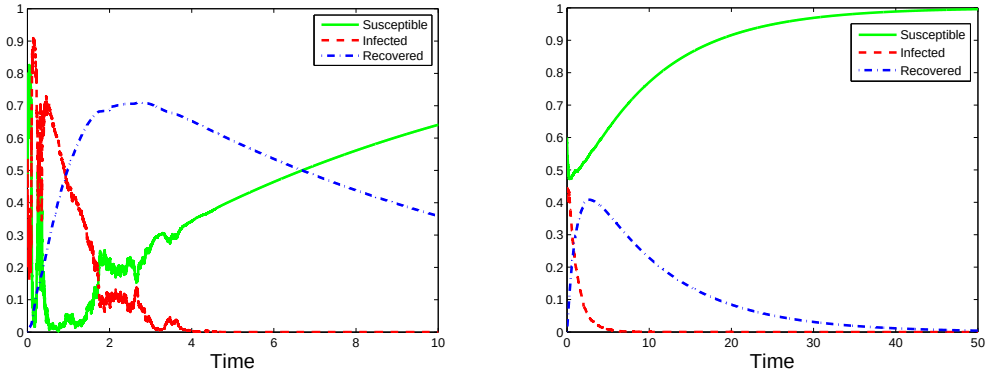


(d) Ensemble average of (3.1).

Figure 1

Finally, if we consider the pulse treatment model with $\beta_1 = 4$, treatment rate $p = 0.1$, other parameters the same as above except with noise parameter $c = 5$, then it is apparent from Figure 2 that the solutions converge to the disease-free equilibrium.

In this case $\mathcal{R}_p = 12.9$ and, because of the increase in the noise parameter, the critical treatment rate is $p_{\text{crit}} = 1$, which is physically infeasible. This raises an important point: because of the oscillatory nature with which solutions converges in the endemic case, there can be an extinction event if the noise parameter is large enough. When the number of infected oscillate close to zero, the infected population can reduce to zero from the relatively large stochastic perturbations. This illustrates the fact that the basic reproduction number $\mathcal{R}_p$ is too strict (and hence Theorem 3.1 is sufficient but not necessary); we conjecture the noise parameter c should be inversely proportional to the basic reproduction number. Based on simulations, this effect seems to also be present in the constant control scheme.



(a) Realization of (3.1) with $p = 0.1$ ($\mathcal{R}_p = 12.9$).

(b) Ensemble average of (3.1) with $c = 5$.

Figure 2

5. **Conclusions**

In this paper, a new type of epidemic model with time-varying contact rates and external noise is studied. In order to do this, the contact rate is modeled as a switching parameter and multiplicative Gaussian white noise is added to represent randomness in the model from external perturbations affecting the transmission of the disease. In particular, a constant treatment and pulse treatment scheme are applied to a new switched SIR model with stochastic perturbations. Under both control strategies, threshold criteria are established which guarantee disease eradication in the mean square, based on the reproduction number of the disease and the magnitude of stochasticity. Some simulations are given to illustrate the theorems in this paper.

For future work, one may consider establishing some results on permanence of the disease in the endemic case. Another possibility is to further investigate the possibility of an extinction event, mentioned at the end of Section 4, where the noise in

the model can lead to eradication in an otherwise endemic model. Another possibility is to assume that other parameters are time-varying or to look at more complex models with stochasticity, such as multi-group models, SEIR models, or age-structured models. Finally, one may consider constant vaccination or pulse vaccination schemes.

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