Research Article

Dynamics of HSV-2 in the Presence of Optimal Counseling and Education among Prisoners

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Received 31 March 2021; Revised 13 June 2021; Accepted 21 June 2021; Published 30 June 2021

Academic Editor: Xiaohua Ding

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Herpes simplex virus type 2 (HSV-2) is one of the major sexually transmitted infections in sub-Saharan prisons and the major driver of HIV. In this paper, we propose and analyze a basic mathematical model for the spread of HSV-2 in prisons. We compute the basic reproduction number and demonstrate that it is a sharp threshold for disease dynamics. The impacts of counseling and education of HSV-2-infected individuals are examined. Equilibrium states of the model are determined and their stability is investigated. The basic model is then extended to incorporate a time-dependent intervention strategy. The aim of the control is tied to reducing the number of infectious individuals through early detection, education, and counseling of the infected. The Pontryagin’s Maximum Principle is used to characterize the optimal level of the control, and the resulting optimality system is solved numerically.

1. Introduction

Herpes simplex virus type 2 (HSV-2), a double-stranded deoxyribonucleic acid (DNA) virus, is a sexually transmitted pathogen that almost exclusively infects the genital region and has been recognized as the most common cause of genital ulcer disease [1]. HSV-2 is characterized by some symptoms, which begin to show (1-2) weeks after sexual exposure to the virus. The first signs are a tingling sensation in the affected areas (such as genitalia, buttocks, and thighs) and some clusters of small red bumps that develop into blisters. It differs from other STIs, such as syphilis, human papillomavirus, gonorrhea, chlamydia, and trichomoniasis; infection with HSV-2 is lifelong, and once infected, there is currently no treatment to remove it [2, 3]. Current intervention alternatives for controlling HSV-2 are limited to the promotion of condom use, patient education, and treatment with oral medication [3]. Effective vaccines to prevent HSV-2 infection are not yet available but are currently being developed [1]. Annually, HSV-2 causes an estimated 23 million new infections worldwide and infects over 500 million people [4]. HSV-2 seroprevalence ranges from 16% among 14–49 year olds in the United States and to around 40–60% in areas of sub-Saharan Africa [5, 6]. Among the 15–19-year-old females in sub-Saharan Africa, there are 1.5 million new HSV-2 infections every year [4]. It was once recognized as the third most costly STI after HIV and HPV [7]. HSV-2 has now reached epidemic proportions worldwide; patients and physicians in a similar way face major predicaments in the prevention of further spread and the management of the disease. Furthermore, HSV-2 has received special attention in recent years because it can increase the risk of HIV infection by up to three/four times [1, 8, 9]; hence, a control on HSV-2 implies a reduction in HIV prevalence. It is worth noting that the risk of getting HIV infection is not affected by the absence of genital herpes symptoms, such as blisters [10]. The effort to control STIs, including HSV-2, in developing countries is hindered by shortages of specialist STI clinics and lack of suitable diagnostic methods and drugs for treatment. Prison inmates are reported to have high rates of HSV-2, and this is mostly due to low socioeconomic status and sexual health risk behaviors [11, 12].
On a global scale, the prison population is growing rapidly, with high incarceration rates leading to overcrowding, which largely stems from national law and criminal justice policies. In sub-Saharan countries, overcrowding and poor physical conditions prevail [13, 14]. This phenomenon poses significant health concerns with regard to the control of infectious diseases [13, 14]. Sub-Saharan prison populations are predominantly male and most prisons are male-only institutions, including the prison staff. In such a gender-exclusive environment, male-to-male sexual activity, prisoner-to-prisoner, is frequent [15]. The actual number of instances is likely to be much higher than what is reported, mainly due to continual denial, fear of being exposed, or the criminalization of sodomy and homosexuality. As much as homosexuality is gaining more and more space worldwide, in sub-Saharan Africa, where HSV-2 is at its highest, homosexuality is still being criminalized in most of those countries [16–19]. Countries in sub-Saharan Africa, like Zimbabwe, stated that they do not distribute condoms to inmates in the country’s prisons as there was no policy in place to guide such a program [20, 21]. Thus, inmates engage in same-sex without using any protection. Since the prison inmates return to the general population upon having freedom, this may likely expose them to HSV-2 infection. Thus, early detection, counseling, and treatment are needed for prisoners [22]. Until a vaccine is developed, it has been suggested that education may be the most effective way of reducing the further spread of HSV-2 [23].

Mathematical epidemiological models have been demonstrated to be vital tools that can help understand this disease and provide solutions to phenomena that are complex to measure in the field. The literature on epidemic models on the spread of infectious diseases is quite substantial and it also contains some recent research works (see [24–28]). In [24], a new definition of a fractional derivative with nonsingular kernel in the sense of Caputo was proposed, which generalized various forms existing in the literature. Furthermore, the research studied numerically the impact of the order on the dynamical behavior of the biological model. A delayed SIR epidemic model with a generalized incidence rate was studied in [24], with the time delay representing the incubation period. These studies, indeed, produced useful results and we utilize some of the results to develop and analyze our framework. Several mathematical epidemiological models have been presented and analyzed to probe the HSV-2 transmission dynamics and their control (see [29–37]). Podder and Gumel [29] formulated and analyzed a deterministic mathematical epidemiological model for HSV-2, which considered disease transmission by the infected individuals in the dormant state and an imperfect vaccine for HSV-2. In their model, they assumed that the infectious individuals revealing clinical symptoms are the ones who have more impact on the prevalence of HSV-2. A mathematical model was proposed in [30] to derive three summary measures for the population impact of imperfect HSV-2 vaccines as a function of their efficacies in reducing infectivity during shedding, susceptibility, and genital shedding. Mhlanga et al. [31] proposed and qualitatively analyzed a deterministic mathematical model to investigate the impact of poor treatment adherence towards HSV-2 antiviral treatment, which was later improved to consider HSV-2 treatment adherence and gender [32]. Feng et al. [33] developed and analyzed a mathematical model to explore the epidemiological synergy between HSV-2 and HIV by investigating the contribution of HSV-2 to HIV prevalence and evaluating the potential population-level impact of HSV-2 therapy on HIV control. Safi and DarAssi [34] proposed and carried out a comprehensive analysis of a simple age-structured HSV-2 model. In [35], a mathematical model was proposed and analyzed to try and investigate the impact of sexting on the transmission dynamics of HSV-2. Spicknall et al. [36], in their review, identified published literature of dynamic mathematical models assessing the impact of either prophylactic or therapeutic HSV-2 vaccination at the population level and compared each study’s model structure and assumptions and predicted vaccination impact. A case study on HSV-2 was carried out, with the major aim of understanding the transmission dynamics of an infectious disease. An SVIRI multigroup epidemic model for HSV-2 was then developed and analyzed, which considered infection relapse, waning vaccine immunity, and the effects of vaccination [37].

From the presented models, it is worth noting that most of these models aimed at answering questions on the possible impact of imperfect HSV-2 vaccines. In this manuscript, we seek to develop a mathematical model to assess the impact of educating and counseling of prison inmates on the transmission dynamics of HSV-2 in the presence of HSV-2 early detection, treatment, and relapse. We will consider a male-only prison, where male-to-male sexual activity is frequent. Heterosexual transmission of HSV-2 is the principal mode of infection in Africa in general and sub-Saharan Africa in particular, where HSV-2 is at its highest. It is worth noting that, in most parts of sub-Saharan Africa, homosexuality is still being regarded as illegal, but it is rife in their prisons [16, 19, 38, 39]. Our manuscript focuses on sub-Saharan prisons.

The structure of the article is as follows. Section 2 constitutes model formulation and analytic results are presented in Section 3. Sensitivity analyses of the reproduction number are reported in Section 4, and the optimal control is discussed in Section 5. The article concludes with a discussion in Section 6.

2. Model Description

The model subdivides the total male prisoner population $N(t)$ into the following subpopulations: the susceptible $S(t)$, the infectious who are not on treatment $I(t)$, the infectious on treatment and no longer having sex with other men $R(t)$, and the infectious on treatment and still having sex with other men, but have reduced the number of partners $D(t)$. Thus, $N(t) = S(t) + I(t) + D(t) + R(t)$. The infected individuals in the $I(t)$ class have not been detected for HSV-2, and they do not know that they are infectious. It was shown that asymptomatic shedding is frequent and is the most common mechanism of transmission to sex partners; moreover, some individuals transmit the disease...
unknowingly [40]. Male prisoners are recruited at a constant rate \( \tau \), and we assume that every individual is recruited into the prison as susceptible. There is a constant natural death rate \( \mu \) in each class. Furthermore, individuals are released from the prison at rate \( \nu \), which is also assumed constant in each class. The force of infection associated with HSV-2 infection denoted by \( \lambda \) is given by

\[
\lambda = (1 - \alpha)\beta(I + (1 - \eta)D),
\]

where \( \beta \) is the infection rate and \( \eta \in (0, 1) \) is a modification parameter accounting for the assumed reduced likelihood of HSV-2 infection by individuals in class \( D \) compared to those in class \( I \), and the acute infection spontaneously clears with proportion \( \alpha \). Upon being detected, the infected individuals seek treatment at rate \( \theta \). Upon commencing treatment, proportion \( \rho \) of infectious individuals no longer engages in sex with other men and the complementary \( (1 - \rho) \) fails to totally stop having sex with other men. We assume that individuals in class \( R \) would be provided with all the needs that they may need so that they do not engage in sex with other men. Furthermore, individuals under treatment are also given counseling and education in relation to unprotected sex and other health-related risks. Hence, individuals in class \( D \) would reduce the number of their partners and they will also be careful when engaging in sex with other men. Due to the need for favors and lack of entertainment in prisons, some of the individuals in class \( R \) are assumed to relapse at rate \( \kappa \) and start having sex with other men again. In the context of sub-Saharan prisons where sex with other men (homosexuality) is criminalized, in our model, we assume that the prisoners are not given any condoms. Thus, the word sex refers to unprotected sex throughout the manuscript. This implies the following system of nonlinear differential equations to model the disease dynamics of HSV-2 in a male-only prison:

\[
\begin{align*}
S' &= \tau - \lambda S - (\mu + \nu)S, \\
I' &= \lambda S - (\mu + \nu + \theta)I, \\
R' &= \rho \theta I - (\mu + k + \nu)R, \\
D' &= (1 - \rho)\theta I + kR - (\mu + \nu)D.
\end{align*}
\]

**3. Model Analysis**

**3.1. Model Basic Properties.** In this section, we investigate the basic properties of the solutions of model system (2).

**Theorem 1.** Given the initial data as \( S(0) \geq 0, I(0) \geq 0, R(0) \geq 0, D(0) \geq 0 \), then the solutions \((S, I, D, R)\) of model system (2) are nonnegative for all \( t > 0 \).

**Proof.** Let \( \tilde{t} = \sup \{t > 0: S > 0, I > 0, R > 0, D > 0 \} \in [0, t] \). Hence, \( \tilde{t} > 0 \). Then, it follows from the first equation of model system (2) that

\[
\frac{dS}{dt} + (\lambda + \mu + \nu)S = \tau,
\]

which can also be expressed as

\[
\frac{d}{dt} \left( S(t) \exp \left( (\mu + \nu) t + \int_{0}^{t} \lambda(i) di \right) \right) = \tau \exp \left( (\mu + \nu) t + \int_{0}^{t} \lambda(i) di \right).
\]

Thus,

\[
S(\tilde{t}) \exp \left( (\mu + \nu) \tilde{t} + \int_{0}^{\tilde{t}} \lambda(i) di \right) - S(0) = \int_{0}^{\tilde{t}} \tau \exp \left( (\mu + \nu) y + \int_{0}^{y} \lambda(i) di \right) dy,
\]

so that

\[
S(\tilde{t}) = S(0) \exp \left[ -(\mu + \nu) \tilde{t} - \int_{0}^{\tilde{t}} \lambda(i) di \right] + \left( \exp \left[ -(\mu + \nu) \tilde{t} - \int_{0}^{\tilde{t}} \lambda(i) di \right] \right)
\times \left( \int_{0}^{\tilde{t}} \tau \exp \left( (\mu + \nu) y + \int_{0}^{y} \lambda(i) di \right) dy \right) > 0.
\]

In a similar way, it can be shown that \( I > 0, R > 0, \) and \( D > 0 \) for all time \( t > 0 \). \( \square \)

**Theorem 2.** The region \( \mathcal{G} = \{ (S(t), I(t), R(t), D(t): N(t) \leq (\tau/(\mu + \nu)) \} \) is positively invariant and attracting with respect to model system (2).

**Proof.** Adding all the equations of model system (2), we have

\[
\frac{dN}{dt} = (\mu + \nu) \frac{\tau}{\mu + \nu} - N = N(0) - N(t), \quad N(0) = N_0,
\]

with \( N_0 = S(0) + I(0) + R(0) + D(0) \). Since \( (dN/dt) \leq \tau - (\mu + \nu)N \), it follows that

\[
\frac{dN}{dt} \leq 0, \quad \text{if } N > \frac{\tau}{\mu + \nu}.
\]

Making use of the standard comparison theorem [41], it follows that

\[
N(t) = N_0 \exp\left[ - (\mu + \nu) \right] + \left( \frac{\tau}{\mu + \nu} \right) (1 - \exp\left[ - (\mu + \nu) \right]).
\]

Particularly, \( N(t) \leq (\tau/(\mu + \nu)) \) if \( N_0 \leq (\tau/(\mu + \nu)) \). Therefore, model system (2) will be analyzed in a suitable region \( \mathcal{G} \subset \mathbb{R}^4_+ \). The region

\[
\mathcal{G} = \left\{ (S(t), I(t), R(t), D(t)): N(t) \leq \frac{\tau}{\mu + \nu} \right\}
\]

is positively invariant and attracting. Existence, uniqueness, and continuation results for model system (2) hold in this region. \( \square \)
3.2. The Disease-Free Equilibrium and Reproduction Number.

It can be established through direct computations that model system (2) has a disease-free equilibrium given by

\[ \mathcal{E}^0 = (S^0, I^0, R^0, D^0) = \left( \frac{\tau}{\mu + \nu}, 0, 0, 0 \right). \]  \hspace{1cm} (11)

We denote the reproduction number by \( R_0 \), which is defined as the average number of secondary infections generated by a single infectious case in a completely naive population during its average infectious period [42]. It is frequently considered as a threshold quantity for the disease dynamics, vital in predicting the transmission and spread of the disease. By making use of the next-generation matrix notations in [42], we have the nonnegative matrix \( F \), which represents the generation of new infections, and the nonsingular matrix \( V \), representing the disease transfer among compartments. Thus, at the DFE, \( F \) and \( V \) are, respectively, given by

\[
R_0 = \rho(FV^{-1}) = \frac{\tau \beta (1 - \alpha) [\mu + \nu + \theta (1 - \eta)]}{(\mu + \nu)^2 (\mu + \nu + k)} \left( \mu + \nu \right). \hspace{1cm} (13)
\]

Through the application of Theorem 2 in van den Driessche and Watmough [42], the following result is established.

**Theorem 3.** If \( R_0 < 1 \), then \( \mathcal{E}^0 \) is locally asymptotically stable and unstable for \( R_0 > 1 \).

Additionally, we can obtain a stronger result regarding the global dynamics of the DFE. We will utilize the comparison theorem [41, 43] approach in analyzing the global asymptotic stability.

**Theorem 4.** The disease-free equilibrium point \( \mathcal{E}^0 \) is globally asymptotically stable (GAS) if \( R_0 \leq 1 \) and unstable when \( R_0 > 1 \).

**Proof.** The proof is based on using a comparison theorem [41, 43]. Note that the equations of the infected components in model system (2) can be written as

\[
\begin{pmatrix} I' \\ R' \\ D' \end{pmatrix} = (F - V) \begin{pmatrix} I \\ R \\ D \end{pmatrix} - \beta \left( \frac{\tau}{\mu + \nu} - S \right),
\]

where matrices \( F \) and \( V \) are as defined in equation (12). Utilizing the fact that the eigenvalues of the \( F - V \) matrix all have negative real parts, it then follows that the linearized differential inequality system (15) is stable whenever \( R_0 < 1 \).

\[
\begin{pmatrix} I' \\ R' \\ D' \end{pmatrix} \leq (F - V) \begin{pmatrix} I \\ R \\ D \end{pmatrix},
\]

In consequence, \( (I, R, D) \to (0, 0, 0) \) as \( t \to \infty \). Hence, by the comparison theorem [41, 43], \( (I, R, D) \to (0, 0, 0) \) as \( t \to \infty \), and evaluating system (2) at \( I = R = D = 0 \) gives \( S \to S^0 \) for \( R_0 \leq 1 \). Thus, the DFE \( (\mathcal{E}^0) \) is GAS for \( R_0 \leq 1 \).

It is worth noting that \( R_0 = 1 \) is a sharp threshold for disease dynamics as portrayed by the result established in Theorem 4: the disease dies out when \( R_0 \leq 1 \); conversely, the disease persists when \( R_0 > 1 \). In biological terms, a uniform persistent system depicts that the disease persists for a long period. Next, we examine the uniform persistence, and we claim the following result. \qed

**Theorem 5.** If \( R_0 > 1 \), model system (2) is uniformly persistent; namely, there exists a constant \( \gamma > 0 \) such that

\[
\lim_{t \to \infty} \inf S(t) > \gamma, \quad \lim_{t \to \infty} \inf I(t) > \gamma, \quad \lim_{t \to \infty} \inf R(t) > \gamma, \quad \lim_{t \to \infty} \inf D(t) > \gamma,
\]

for any initial conditions satisfying
S(0) ≥ 0,  
I(0) ≥ 0,  
R(0) ≥ 0,  
D(0) ≥ 0.

Proof. Let \( \Delta = \{(S, I, R, D) \in \mathbb{R}^4_+ : I = R = D = 0\} \). Thus, \( \Delta \) denotes the set of all the disease-free states of model system (2) and it can be established that \( \Delta \) is positively invariant. Let \( M = \Delta \cap \mathcal{F} \), which then implies that \( M \) is positively invariant since \( \Delta \) is positively invariant and \( \mathcal{F} \) is also positively invariant. In addition, note that \( \mathcal{F}^0 \in M \) and \( \mathcal{F}^0 \) attracts all solutions in \( \mathcal{F} \). Accordingly, \( \Omega(M) = \{\mathcal{F}^0\} \), where \( \Omega(M) \) denotes a union of periodic orbits. The equations for the infected components of model system (2) can be represented as

\[
x'(t) = Y(x)x(t),
\]

where \( x(t) = (I(t), R(t), D(t))^T \) and \( Y(x) = [\Gamma - V] \), with

\[
\Gamma = \begin{bmatrix}
(1 - \alpha)\beta S & 0 & (1 - \alpha)(1 - \eta)\beta S \\
0 & 0 & 0 \\
0 & 0 & 0
\end{bmatrix}.
\]

We can clearly see that \( Y(\mathcal{F}^0) = F - V \). Moreover, it can be easily checked that \( Y(\mathcal{F}^0) \) is irreducible. Applying Lemma A.4 from Ackleh et al. [44], we can show that \( M \) is a uniform weak repeller, which has been stated below for elucidation.

Lemma 1. Assume that \( \Omega(M) \) is a union of periodic orbits and the following hold:

1. \( \forall \mathcal{P} \subseteq \Omega(M) \) a periodic orbit of period \( T \), \( \exists x \in \mathcal{P} \) such that \( P(T, x) \) is primitive

2. \( r(P) > 1 \), for each periodic orbit \( \mathcal{P} \subseteq \Omega(M) \)

Then, \( M \) is a uniformly weak repeller.

Since the spectral radius of \( Y(\mathcal{F}^0) = \mathcal{R}_0 > 1 \), the spectral radius of \( e^{\Gamma(\mathcal{F}^0)} > 1 \). Hence, condition 2 of Lemma A.4 is satisfied. Taking \( x = \mathcal{F}^0 \), we have \( P(T, \mathcal{F}^0) = e^{\Gamma(\mathcal{F}^0)} \), which is a primitive matrix, because \( Y(\mathcal{F}^0) \) is irreducible, as outlined in Theorem A.12(i) [45]. This satisfies condition 1 of Lemma A.4. Therefore, \( M \) is a uniform weak repeller and the disease is weakly persistent. \( M \) is compact since it is trivially closed and bounded relative to \( \mathcal{F} \). Consequently, by Theorem 1.3 [46], we have established that \( M \) is a uniform strong repeller and the disease is uniformly persistent.

3.3. Endemic Equilibrium and Its Stability Analysis. We now investigate the stability of the endemic equilibrium point (EEP). Expressed in terms of the force of infection, the EEP for model system (2) is given by \( \mathcal{E}^* = (S^*, I^*, R^*, D^*) \), with

\[
\begin{align*}
S^* &= \frac{\tau}{\lambda^* + \mu + v} \\
I^* &= \frac{\tau \lambda^*}{(\lambda^* + \mu + v)(\mu + v + \theta)} \\
R^* &= \frac{\lambda^* \tau \rho \theta}{(\mu + v + k)(\lambda^* + \mu + v)(\mu + v + \theta)} \\
D^* &= \frac{\lambda^* \tau \theta (k + (1 - \rho)\mu + v)}{(\mu + v)(\mu + v + k)(\lambda^* + \mu + v)(\mu + v + \theta)}.
\end{align*}
\]

On substituting (20) into the force of infection \( \lambda^* \), which is equation (2), we have

\[
\lambda^* g(\lambda^*) = \lambda^* (Y_1 \lambda^* + Y_2) = 0,
\]

where \( \lambda^* = 0 \) corresponds to the disease-free equilibrium and \( g(\lambda^*) = 0 \) corresponding to the endemic equilibrium point existence, where

\[
\begin{align*}
Y_1 &= \frac{1}{\mu + v} \\
Y_2 &= \frac{\mathcal{R}_0 - 1}{Y_1}.
\end{align*}
\]

\( Y_1 \) is always positive and \( Y_2 \) is positive or negative on condition that \( \mathcal{R}_0 < 1 \) or \( \mathcal{R}_0 > 1 \), respectively.

Theorem 6. The endemic equilibrium point exists whenever \( \mathcal{R}_0 > 1 \).

Proof. By analyzing the linear equation \( Y_1 \lambda^* + Y_2 = 0 \), it follows that

\[
\lambda^* = \frac{Y_2}{Y_1} = \frac{\mathcal{R}_0 - 1}{Y_1}.
\]

It is important to state that the disease is endemic when the force of infection \( \lambda^* > 0 \), which implies that \( \mathcal{R}_0 > 1 \). Therefore, the endemic equilibrium point \( \mathcal{E}^* \) exists whenever \( \mathcal{R}_0 > 1 \).

To examine the local stability of the endemic equilibrium point, we will make use of the Centre Manifold Theory [47], and we establish the following result.

Theorem 7. The endemic equilibrium point \( \mathcal{E}^* \) is locally asymptotically stable for \( \mathcal{R}_0 > 1 \) and sufficiently close to 1.

The proof for Theorem 7 is presented in Appendix A.

Theorem 8. If \( \mathcal{R}_0 > 1 \), the endemic equilibrium point \( \mathcal{E}^* \) is globally asymptotically stable.

The proof for Theorem 8 is presented in Appendix B.

4. Sensitivity Analysis

To further examine the results of our foregoing analysis, we simulated model system (2), making use of the parameters in
Table 1. Regrettably, the scarcity of the data on HSV-2 with a focus on sub-Saharan prisons limits our ability to calibrate; however, some of the parameter values are assumed within the realistic range for illustrative purposes. These miserly assumptions reflect the shortage of information currently available on HSV-2 within prisons, as also observed in [55]. Reliable data on STIs within prisons would enhance our understanding and be of great assistance in the possible intervention strategies to be executed. Before we present our numerical simulations, we shall first explore the sensitivity indices of \( R_0 \) based on the perturbation of fixed point estimates.

Sensitivity analysis is very important since it tells us how important each parameter is to disease transmission. Such information is important not only for experimental design but also to other various aspects, such as data assimilation and reduction of complex nonlinear models. Sensitivity analysis is commonly used to determine the robustness of model predictions to parameter values since there are usually errors in data collection and presumed parameter changes. It is used to discover parameters that have a high impact on the reproduction number and should be targeted by intervention strategies.

In countless models of epidemiology, the size of the reproductive number is related to the level of infection. The same applies with model system (2) since we are dealing with HSV-2. Sensitivity analysis examines the quantity and type of change inherent in the model as captured by the terms that define the reproductive number (\( R_0 \)). \( R_0 \) is exceedingly sensitive to a certain parameter, then a perturbation of the states that connects the dynamics to such may prove useful in identifying policies or intervention strategies that reduce epidemic prevalence. When the respective variable is a differentiable function of the parameter, the sensitivity index may also be defined using partial derivatives.

**Definition 1.** The normalized forward sensitivity index of variable \( \rho \) that depends on a parameter \( \tau \) is defined by

\[
\gamma^\tau = \frac{\partial \rho}{\partial \tau} \cdot \frac{\tau}{\rho}.
\]  

(24)

Results from Table 2 suggest that whenever the infection rate and the relapse rate are increased, the reproduction number would also increase. An increase in the number of prisoners relapsing by 10% results in the increase of the reproduction number by 6.82%. Counseling and education of prisoners on treatment can be helpful in the reduction of the reproduction number by reducing the rate of relapse. With sub-Saharan prisons not allowing same-sex engagements and ignoring the issue as if it is not happening within their prisons, methods such as giving the prisoners condoms or allowing them to meet with their respective spouses can be helpful. An increase in the ratio of the prisoners seizing to have sex with other men is vital in reducing the reproduction number. An increase in the ratio of men seizing having sex with other men in prison by 10% reduces the reproduction number by 6.1%. Furthermore, we note that an increase in the treatment rate is also important in the reduction of the reproduction number, as expected. It is important to state that African governments must now start accepting that homosexuality is rife within their prisons; hence, protection must be made available for prisoners.

Figure 1 depicts the impact of varying the infection rate \( \beta \) on the reproduction number under various scenarios:

(i) Scenario 1: \( R_s \) refers to the reproduction number when everyone is having sex in prison, and it is achieved by setting \( \rho = 0 \) and \( k = 0 \) on our reproduction number equation (13).

(ii) Scenario 2: \( R_n \) refers to our reproduction number as defined and given in equation (13).

(iii) Scenario 3: \( R_{nr} \) refers to the scenario where there are no prisoners who are relapsing back into having sex with other men, and it is achieved by setting \( k = 0 \) on our reproduction number in equation (13).

(iv) Scenario 4: lastly, \( R_{nt} \) refers to the reproduction number where there are no prisoners having sex with other men and it is achieved by setting \( \rho = 1 \) and \( k = 0 \) on the reproduction number equation (13).

From Figure 1, we can clearly see that \( R_s > R_n > R_{nr} > R_{nt} \). Thus, in the absence of men having sex with other men, the reproduction number would be the lowest. That is, the reproduction number can be greatly reduced if the prisoners are given protection against STIs. Furthermore, we note that our results from Table 2 are in agreement with our result from Figure 1 in that a reduction in the number of individuals relapsing is vital in the reduction of the reproduction number. Thus, methods which encourage prisoners not to have unprotected sex with other men and which minimizes the rate of relapse would be vital in curtailting the spread of HSV-2 within the prison.

Next, we present some numerical simulations to assess the impact of relapse on the prison population.

Our simulations show the effect of increasing the relapse rate on HSV-2 transmission dynamics over a 20-year period. Figure 2(a) suggests an interesting result in that increasing the relapse rate has no much effect on the infectious individuals who have not yet been detected for HSV-2 (I). Figure 2(b) suggests that an increase in the rate of relapse results in the reduction of the individuals on treatment who are no longer having sex with other men (R). Also, an increase in the rate of relapse has the effect of increasing the cases of individuals who are on treatment but still having sex with other men (D) as depicted in Figure 2(c). Thus, on top of treatment in trying to arrest the rate of relapse, education and counseling of the detected individuals would be crucial in controlling HSV-2 in prisons. From Table 2, we established that increasing the relapse rate causes the reproduction number to also increase. Therefore, from Figure 2 and Table 2, we can see that it is important to make sure the rate of relapse is low.


Table 1: Model parameters and their baseline values.

| Definition                              | Symbol | Baseline values (range) | Source |
|-----------------------------------------|--------|-------------------------|--------|
| Recruitment rate                        | \( \tau \) | 123 per 100 000         | [48]   |
| Infection rate                          | \( \beta \) | 0.5 (0.1–0.6)           | [49, 50] |
| Natural death rate                      | \( \mu \) | 0.0142 (0.01–0.02)      | [51]   |
| Rate of infections spontaneously clear  | \( \alpha \) | 0.2 (0.125–0.33)        | [52]   |
| Relapse rate                            | \( k \) | 0.1 (0.01–0.2)          | Assumed |
| Treatment rate                          | \( \theta \) | 0.1 (0–0.2)             | [33]   |
| Ratio of seizing having sex with other men | \( \rho \) | 0.2 (0.125–0.33)        | [51]   |
| Modification parameter                  | \( \eta \) | (0, 1)                  | Assumed |

The time unit is in years.

Table 2: Sensitivity indices of model parameters to \( R_0 \).

| Symbol | Sensitivity index |
|--------|-------------------|
| \( \rho \) | -0.60672212 |
| \( \alpha \) | -0.428571 |
| \( \beta \) | 1 |
| \( \eta \) | -0.483801 |
| \( \theta \) | -0.534468 |
| \( k \) | 0.681731 |
| \( \mu \) | -0.107538 |
| \( \nu \) | -0.96885 |

5. Optimal Control Problem

From the conclusion we obtained under the sensitivity analysis and the time series plots presented in Table 2, we now introduce into the model system (2) a time-dependent control \( u \). The time-dependent control \( u \) represents the effort on counseling and education of HSV-2 patients who have been detected and are on treatment, in turn reducing the total number of HSV-2 infections. Counseling would be crucial since it would deliver important information, advice, and assistance to help inmates with their medications and to ensure they take them properly (with proper adherence). While education would make sure the inmates are well-informed about their health and the dangers of unprotected sex or any other risky behaviors. It is beneficial to note that when counseling and education are implemented, the infection will be reduced. Simultaneously, we hope to minimize the costs of achieving this. Thus, the model system (2) with the time-dependent control is then given by

\[
\begin{align*}
S' &= \tau - (1 - a)\beta (I + (1 - u)(1 - \eta)D)S - (\mu + \nu)S, \\
I' &= (1 - a)\beta (I + (1 - u)(1 - \eta)D)S - (\mu + \nu + \theta)I, \\
R' &= \rho \theta I - (\mu + \nu + k)R, \\
D' &= (1 - \rho)\theta I + kR - (\mu + \nu)D.
\end{align*}
\]

Thus, we want to determine the optimal value \( u^* \) that minimizes the cost objective functional \( J(u) \), with

\[
J(u) = \int_0^T \left[ ad + bu(D + R) + cu^2 \right] dt,
\]

where \( T \) denotes the final time. The coefficient \( a \) represents the weight constant for class \( D \). The costs associated with counseling and education of HSV-2 patients under treatment are represented by the terms with coefficient \( b \). The objective functional in (26) also comprises a quadratic term with coefficient \( c \) to point out potential nonlinearities in the costs. It is worth noting that the control effort is assumed to be nonlinear due to a number of advantages associated with a nonlinear function on the control. One of the advantages is that a nonlinear control allows the Hamiltonian to attain its minimum over the control set at a unique point. We assume that the population of the infectious individuals on treatment and no longer having sex with other men \( R \) is indistinguishable from the population of infectious individuals on treatment and having partial sex with other men \( D \) concerning counseling and education, thus incurring costs. The control \( u \) is bounded, Lebesgue integrable function [56, 57]. The goal is to find the optimal control \( u^* \) such that

\[
J(u^*) = \min_{u \in \mathcal{U}} J(u),
\]

where the control set is given by

\[
\mathcal{U} = \{ u(t), \quad u: [0, T] \rightarrow [0, 1], \text{ is Lebesgue measurable} \}.
\]

5.1. Characterization of Optimal Control. The necessary conditions that an optimal control must satisfy come from
Pontryagin’s Maximum Principle [58]. Equations (25) and (26) are converted into a problem of minimizing pointwise the Hamiltonian $J$, with respect to the control $u$, through the application of the Pontryagin’s Maximum Principle. First, we formulate the Hamiltonian from the cost functional equation (26) and the governing dynamics equation (25) to obtain the optimality conditions:

$$H = aD + bu(D + R) + cu^2 + \lambda_1 [b(1 - \alpha)\beta(I + (1 - \mu)(1 - \eta)D) - (\mu + v)S] + \lambda_2 [(1 - \alpha)\beta(I + (1 - \mu)(1 - \eta)D) - (\mu + v + \theta)I] + \lambda_3 [\rho \theta I - (\mu + v + k)R] + \lambda_4 [(1 - \rho)\theta I + kR - (\mu + v)D].$$

Figure 2: Time series plots showing the effect of increasing relapse rate $k$ on (a) infectious population who are not on treatment $I$, (b) infectious on treatment but still having sex with other men, and (c) infectious on treatment and no longer having sex with other men, over a period of 20 years. The rest of the parameters are as presented in Table 1, and the following assumed initial conditions were used: $S = 2000, I = 400, R = 300, D = 200$. 

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In addition, \( \lambda_1, \lambda_2, \lambda_3, \lambda_4 \) are the respective adjoints for the states \( S, I, R, D \). The system of adjoint equations is obtained by taking the appropriate partial derivatives of the Hamiltonian given in equation (29) concerning the respective state and control variables.

**Theorem 9.** Given an optimal control \( u^* \) and solutions \( S^*, I^*, R^*, D^* \) of corresponding state system (25) that minimizes \( J(u^*) \) over \( \mathcal{U} \), then there exist adjoint variables \( \lambda_1, \lambda_2, \lambda_3, \lambda_4 \) satisfy

\[
\begin{align*}
\frac{d\lambda_1}{dt} &= \frac{\partial \mathcal{H}}{\partial u} = (1-\alpha)\beta(1-u)(1-\eta)(1-D)\left[\lambda_1 - \lambda_2\right] + (\mu + \nu)\lambda_1, \\
\frac{d\lambda_2}{dt} &= \frac{\partial \mathcal{H}}{\partial S} = (1-\alpha)\beta S\left[\lambda_1 - \lambda_2\right] + \theta\left[\lambda_2 - \lambda_4\right] + (\mu + \nu)\lambda_2 + \rho\theta\left[\lambda_4 - \lambda_3\right], \\
\frac{d\lambda_3}{dt} &= \frac{\partial \mathcal{H}}{\partial R} = k\left[\lambda_3 - \lambda_4\right] + (\mu + \nu)\lambda_3 - bu, \\
\frac{d\lambda_4}{dt} &= \frac{\partial \mathcal{H}}{\partial D} = (1-\alpha)\beta DS\left[\lambda_1 - \lambda_2\right] + (\mu + \nu)\lambda_4 - a - bu,
\end{align*}
\]

with transversality conditions

\[ \lambda_i(T) = 0, \quad \text{where } i = 1, 2, 3, 4. \]  

Additionally, the control \( u^* \) is given as

\[
u^* = \min \left\{ \max \left\{ 0, \frac{(1-a)(1-\eta)\beta DS[\lambda_1-\lambda_2] - b(D+R)}{2c} \right\} \right\}.
\]  

**Proof.** By making use of the result by Fleming and Rishel [59], the existence of optimal control is guaranteed. As a result, we obtain the differential equations governing the adjoint variables through the differentiation of the Hamiltonian function, evaluated at the optimal controls. Therefore, the adjoint system can be written as

\[
\frac{d\lambda_1}{dt} = \frac{\partial \mathcal{H}}{\partial u} = (1-\alpha)\beta(1-u)(1-\eta)(1-D)\left[\lambda_1 - \lambda_2\right] + (\mu + \nu)\lambda_1, \\
\frac{d\lambda_2}{dt} = \frac{\partial \mathcal{H}}{\partial S} = (1-\alpha)\beta S\left[\lambda_1 - \lambda_2\right] + \theta\left[\lambda_2 - \lambda_4\right] + (\mu + \nu)\lambda_2 + \rho\theta\left[\lambda_4 - \lambda_3\right], \\
\frac{d\lambda_3}{dt} = \frac{\partial \mathcal{H}}{\partial R} = k\left[\lambda_3 - \lambda_4\right] + (\mu + \nu)\lambda_3 - bu, \\
\frac{d\lambda_4}{dt} = \frac{\partial \mathcal{H}}{\partial D} = (1-\alpha)\beta DS\left[\lambda_1 - \lambda_2\right] + (\mu + \nu)\lambda_4 - a - bu,
\]

evaluated at the optimal control and corresponding state variables and results in the stated adjoint systems (30) and (31). Additionally, on differentiating the Hamiltonian function with respect to the control variables in the interior of the control set \( \mathcal{U} \), where \( 0 < u < 1 \), we have

\[
\frac{\partial \mathcal{H}}{\partial u} = 0.
\]

Thus, solving for \( u^* \), the optimal control results in the optimality condition given as

\[
u^* = \frac{(1-a)(1-\eta)\beta DS[\lambda_1-\lambda_2]-b(D+R)}{2c}.
\]

Characterization equation (32) can be derived through the use of the bounds on the controls. \( \square \)

**Remark 1.** Due to the a priori boundedness of the state and adjoint functions and the ensuing Lipschitz structure of the ordinary differential equations, the uniqueness of the optimal control for small-time \( (T) \) was attained. The uniqueness of the optimality system results in the uniqueness of the optimal control, which consists of equations (25), (30), and (31) with characterization equation (32). The restriction on the length of the time interval is to guarantee the uniqueness of the optimality system; the smallness in the length of time is due to the opposite time orientations of equations (25), (30), and (31); the state problem has initial values and the adjoint problem has final values. This restriction is frequently used in control problems (see [56, 60–65]).

Next, we consider the numerical solutions of the optimality system, the corresponding optimal control, and the interpretations.

### 5.2. Numerical Simulations

We will apply an iterative method with Runge–Kutta fourth-order scheme in solving the optimality system. Starting with a guess for the adjoint variables, the state equations are solved forward in time. After that, those state values are employed to solve the adjoint equations backward in time, and the iterations carry on until convergence. The simulations were executed using parameter values in Table 1 and the following values \( a = 10000, b = 10, c = 100 \). The initial conditions for the
differential equations are \( S(0) = 0.75, I(0) = 0.15, D(0) = 0.05, R(0) = 0.05 \).

Figure 3(a) depicts that optimal counseling and education have a positive effect on susceptible individuals. We note that in the presence of the control, there are more susceptible prisoners as compared to the absence of control. Figure 3(b) illustrates the effect of the control on the individuals who are infected and not on treatment \( I \). We note that in the presence of the control, the population of the infected prisoners who are not on treatment increases sharply in the first 10 years and begins to drop smoothly for the remainder of the period under study. In the presence of the control, we see that the population reduces steadily for the whole period under review. Thus, optimal counseling and education are vital in reducing the number of infected prisoners who are not on treatment.

Figures 3(c) and 3(d) depict that optimal counseling and education have an impact on reducing the number of prisoners infected with HSV-2. We note that for both populations, \( R \) and \( D \), the number of the individuals under optimal control is far lesser than the individuals in the absence of controls.

Figure 4 represents control \( u^* \). It is worth noting that the control is feasible for the rest of the period under study. Thus, the control is important in curtailed the spread of HSV-2 within the prison settings.

Motivated by the article and techniques in [66], we now determine the total number of new infections in prison by the following formula:

\[
I_w = \int_0^T \beta(1 - \alpha)(I + (1 - \alpha)D)dt,
\]

and recalling that, the total cost associated with the infected individuals is given by equation (26). Using our baseline values from Table 1, the total cost of executing the control strategy will be \( \mathcal{J} = 2.0268 \times 10^4 \). In Table 3, we vary the relapse rate \( k \) and see how it affects the number of new infections \( I_w \) and the total cost of executing strategy \( \mathcal{J} \).

Table 2 shows the effects of increasing the rate of relapse within its possible range of \((0.01 - 0.3)\) on the number of new infections \( I_w \) and the total cost of executing strategy \( \mathcal{J} \). We can see that as the rate of relapse increases, the total costs also increase and the number of new infections also increases. The biggest increase in the number of new infections takes place when the rate of relapse is increased from 0.0 to 0.1, with an increase of around 20%. The smallest increase takes place from 0.2 to 0.3, with an increase of around 2%. Thus, we incur more costs when the rate of relapse is increasing from the lower bound than approaching the upper bound. Consequently, in the absence of relapse, we have fewer new infections and the cost of managing the disease is lower.

6. Discussion

Sexually transmitted diseases (STDs) have been identified as the major public health problem worldwide, especially in developing countries, where the resources for their management are scarce. This has caused so many problems within the sub-Saharan prisons. With most countries in sub-Saharan Africa criminalizing homosexuality, it is known to some inmates continue to practice it in most of those countries. Sub-Saharan countries that do criminalize homosexuality do not offer any condoms or protective measures against STDs in their prisons. Until an effective vaccine is developed, education remains the most effective way of reducing further the spread of HSV-2 in sub-Saharan prisons. In this study, a mathematical model to assess the impact of early detection, education, and counseling on the spread of HSV-2 within sub-Saharan prisons is developed and analyzed. Qualitative analysis of the model has shown that the model has a global asymptotically stable disease-free equilibrium whenever the reproduction number is less than unity. The geometric approach method was used to prove the global stability of the endemic equilibrium. Sensitivity analysis of the reproduction number suggests that an increase in the ratio of men having sex with other men by 10% has an impact of increasing the reproduction number by 6.1%. Thus, the introduction of condoms or any other necessary and sufficient methods would be beneficial in curtailing HSV-2. Also, sensitivity analyses suggest that early detection would be beneficial in the reduction of the reproduction number, whereas a relapse of the individuals who would have temporarily seized having sex with other men increases the reproduction number. Optimal control was then applied with the aim of counseling and educating the individuals on treatment who are still having sex with other men without protection. The technical tool used to determine the optimal strategy is Pontryagin’s Maximum Principle. The control represents counseling and education of the detected individuals who are on treatment. Our optimal control results suggest that to successfully control HSV-2 prevalence, we need to keep the cost of the control effort as low as possible, at a low relapse rate. The main findings hinged on that lowering the rate of relapse to less than 20% would be beneficial in reducing HSV-2 within the prisons. Additionally, optimal counseling and education on individuals coming for treatment have a major impact in reducing HSV-2.

Our study is in agreement with several studies that managed to also investigate the aspect of HSV-2 within prison inmates. It was established that there is a need for governments to make policy for screening and treating of HSV-2 of prison inmates, and this was in line with our study in that education and counseling of those detected are impactful [67, 68]. Our study is also in agreement with the research by [67] in that educational campaigns and counseling of inmates are important in the reduction of HSV-2 among inmates. Furthermore, the following studies concurred with our study in that HSV-2 is very high among men who have sex with other men, and they have to be targeted in the prisons, with early detection, education, and counseling [55, 69].

Our study has a few limitations. Limited data exist on HSV-2 with relation to sub-Saharan prisons, particularly...
modeling study on HSV-2 is low. Therefore, some of our numerical estimates remain uncertain. Thus, we had to base our numerical results on data that has been published in various literature. More datasets and experimental studies are needed to include more realistic biological processes in the models. It is worth noting that we assumed no one gets into prison infected with HSV-2; everyone is recruited as susceptible. We also assumed that if someone relapses or joins class $D$, they will never leave that class or stop having sex with other men while they are on treatment until they exit through death or freedom. This is shown in the model by the lack of a transition from class $D$ to class $R$. However, just like any other model, we cannot say the model is complete; it can be extended to include the aspect of gangs and age within the prison settings. Furthermore, when a disease spreads within the community, individuals acquire knowledge about the disease. Thus, it will be interesting to study the memory effect on the spread of HSV-2 using the new generalized fractional derivative outlined in [24].

Figure 3: Graphs of the numerical solutions of the optimality system, showing the propagation of (a) susceptible population $S$, (b) infectious who are not on treatment $I$, (c) infectious on treatment and no longer having sex with other men $R$, and (d) infectious on treatment and being a bit more careful when having sex $D$, over a period of 15 years.
Appendices

A

Proof of Theorem 8

Proof. To investigate the local stability of the EEP, we will make use of the Centre Manifold Theory as outlined in Theorem 4.1 of [47]. To employ the Centre Manifold Theory, it is vital to make the following changes first: $S = x_1, I = x_2, R = x_3, D = x_4$. We now apply the vector notation $X = (x_1, x_2, x_3, x_4)^T$. Therefore, model system (2) can be written in the form $(dX/dt) = F = (f_1, f_2, f_3, f_4)^T$, such that

\[\begin{align*}
x_1' &= f_1 = \tau - (1 - \alpha)\beta(x_2 + (1 - \eta)x_4)x_1 - (\mu + v)x_1, \\
x_2' &= f_2 = (1 - \alpha)\beta(x_2 + (1 - \eta)x_4)x_1 - (\mu + v + \theta)x_2, \\
x_3' &= f_3 = \rho \theta x_2 - (\mu + k + v)x_3, \\
x_4' &= f_4 = (1 - \rho)\theta x_2 + kx_3 - (\mu + v)x_4. \quad (A.1)
\end{align*}\]

This method requires us to evaluate the Jacobian of system (A.1) at $S^0$, with $S^0 = x_1^0, I^0 = x_2^0, R^0 = x_3^0$ and $D^0 = x_4^0$. Thus,

\[
f(S^0) = \begin{bmatrix} -(\mu + v) & -(1 - \alpha)\beta \tau & 0 & -\frac{(1 - \alpha)(1 - \eta)\beta \tau}{\mu + v} \\ 0 & \frac{(1 - \alpha)\beta \tau}{\mu + v} - (\mu + v + \theta) & 0 & \frac{(1 - \alpha)(1 - \eta)\beta \tau}{\mu + v} \\ 0 & \rho \theta & -(\mu + k + v) & 0 \\ 0 & (1 - \rho)\theta & k & -\mu + v \end{bmatrix}.
\]

| $k$ (relapse rate) | The total cost ($J$) | The number of new infections ($I_n$) |
|------------------|----------------------|----------------------------------|
| 0.0              | $1.5240 \times 10^4$ | 0.2288                           |
| 0.1              | $2.0268 \times 10^4$ | 0.2734                           |
| 0.2              | $2.1630 \times 10^4$ | 0.2861                           |
| 0.3              | $2.2214 \times 10^4$ | 0.2916                           |

Figure 4: Time series plot showing the effects of the optimal control $u$.

Table 3: The total number of newly infected individuals for 20 years and the total costs concerning the rate of relapse.

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for which it can be established that

\[
R_0 = \rho(FV^{-1}) = \frac{\tau(1 - \alpha)\beta[k(\mu + v + \theta(1 - \eta)) + (\mu + v)(\mu + v + \gamma)\theta]}{(\mu + v)^2(\mu + v + k)(\mu + \theta)}.
\] (A.3)

Taking \( \beta \) as the bifurcation parameter and solving for \( R_0 = 1 \), with \( \beta = \beta^* \), yields

\[
\beta = \beta^* = \frac{(\mu + v)^2(\mu + v + k)(\mu + \theta)}{\tau(1 - \alpha)[k(\mu + v + \theta(1 - \eta)) + (\mu + v)(\mu + v + \gamma)\theta]}.
\] (A.4)

It is worth noting that the linearized system (A.1) with the bifurcation point \( \beta^* \) has a simple zero eigenvalue. Hence, the author can apply the Centre Manifold Theory [47] as required in analyzing the dynamics of system (A.1) near \( \beta = \beta^* \). It can be established that the Jacobian of system (A.1) comprises the right eigenvector associated with the zero eigenvalue, given by \( u = (u_1, u_2, u_3, u_4)^T \), where

\[
u_1 = -\frac{(1 - \alpha)\beta T_{11} + (1 - \alpha)(1 - \eta)\beta T_{14}}{(\mu + v)^2},
\]
\[
u_2 = \frac{\mu + v + k}{\rho \theta} u_3,
\]
\[
u_3 > 0,
\]
\[
u_4 = \frac{\rho \theta k + (1 - \rho)(\mu + v + k)}{\rho \theta (\mu + v)} u_3,
\]

with \( u_1 > 0, u_2 > 0, u_3 > 0 \).

Also, the associated zero eigenvalue at \( \beta = \beta^* \) with respect to the left eigenvector of \( J (\beta^*) \) is given by

\[
v_1 = 0,
\]
\[
v_2 = \frac{(\mu + v)^2}{(1 - \alpha)(1 - \eta)\beta} v_4 > 0,
\]
\[
v_3 = \frac{k}{\mu + v + k} v_4 > 0,
\]
\[
v_4 > 0.
\]

Then, the author applied Theorem 4.1 from [47]. We will not state Theorem 4.1 because it has been used in many manuscripts and it is not important to be repeating it since we are only interested in applying it.

\[
\text{Determination of the Bifurcation Parameters } a \text{ and } b. \text{ The associated nonzero partial derivatives of } F \text{ for model system (A.1) at the disease-free equilibrium are given by}
\]

\[
\frac{\partial^2 f_1}{\partial x_1 \partial x_2} = \frac{(1 - \alpha)\beta \tau}{\mu + v},
\]
\[
\frac{\partial^2 f_1}{\partial x_1 \partial x_4} = \frac{(1 - \alpha)(1 - \eta)\beta \tau}{\mu + v},
\]
\[
\frac{\partial^2 f_2}{\partial x_1 \partial x_4} = \frac{(1 - \alpha)\beta \tau}{\mu + v},
\]
\[
\frac{\partial^2 f_2}{\partial x_1 \partial x_4} = \frac{(1 - \alpha)(1 - \eta)\beta \tau}{\mu + v},
\]

From (A.7), the author has

\[
a = \frac{2u_1 (1 - \alpha)\beta v_2 (u_3 + (1 - \eta)u_4) \tau}{\mu + v} < 0.
\] (A.8)

The sign of \( b \) is associated with the following nonvanishing partial derivatives of \( F \):

\[
\frac{\partial^2 f_1}{\partial x_1 \partial \beta^*} = \frac{(1 - \eta)(1 - \alpha)\beta \tau}{\mu + v},
\]
\[
\frac{\partial^2 f_1}{\partial x_4 \partial \beta^*} = \frac{(1 - \eta)\beta \tau}{\mu + v},
\]
\[
\frac{\partial^2 f_2}{\partial x_4 \partial \beta^*} = \frac{(1 - \eta)(1 - \alpha)\beta \tau}{\mu + v},
\]
\[
\frac{\partial^2 f_2}{\partial x_4 \partial \beta^*} = \frac{(1 - \eta)\beta \tau}{\mu + v}.
\] (A.9)

From the expressions in (A.9), it follows that

\[
b = \frac{v_2 (1 - \alpha)(u_3 + (1 - \eta)u_4) \tau}{\mu + v} > 0.
\] (A.10)

Thus, \( a < 0 \) and \( b > 0 \), and using Theorem 4.1 item (iv) from [47], the author establishes Theorem 7.
\[B\]

\textbf{Proof of Theorem 8}

\textit{Proof.} Using the geometric approach of Li and Muldowney in [71], we obtain simple sufficient conditions, which show that \( \mathcal{R}^{\ast} \) is globally asymptotically stable. We will not give the outline of the geometric approach since we are only concerned about its application, but for further reference of the method; see [71].

To apply the geometric approach, it is helpful that the model system (2) is reduced. Since the author is making use of proportions for our populations, it is worth noting that \( \mathcal{R} = 1 - S - I - D \), the reduction of the model system (2) into a three-dimensional system can be achieved. The reduced system is given by

\[ S' = \tau - \lambda S - (\mu + v)S, \]
\[ I' = \lambda S - (\mu + v + \theta)I, \]
\[ D' = (1 - \rho)\theta I + k(1 - S + I + D) - (\mu + v)D, \]

and the simplex \( \mathcal{E} \in \mathbb{R}_+^3 \) is transformed to the following convex region in \( \mathbb{R}_+^3 \):

\[ \Omega = \left\{ S(t), I(t), D(t) \in \mathbb{R}_+^3 : 0 \leq S + I + D \leq 1 \right\}. \]

The stability, existence, and persistence of our system hold in this region \( \Omega \).

The author now applies the geometric approach. For the reason that \( \mathcal{R}^{\ast} \) exists for \( \mathcal{R}_0 > 1 \), then the Jacobian matrix resulting from system (B.1) along a solution \((S, I, D)\) is

\[
\begin{pmatrix}
-(1 - \alpha)\beta(S + I(1 - \eta)D) - (\mu + v) & -(1 - \alpha)\beta S & -(1 - \alpha)(1 - \eta)\beta S \\
(1 - \alpha)\beta(S + I(1 - \eta)D) & (1 - \alpha)\beta(S - (\mu + \theta)) & (1 - \alpha)(1 - \eta)\beta S \\
-k & (1 - \rho)\theta - k & -(\mu + \theta + k)
\end{pmatrix}
\]

Then, the second additive compound matrix of system (B.1) is given by

\[
J^{[2]} = \begin{pmatrix}
\Lambda & (1 - \alpha)(1 - \eta)\beta S & (1 - \alpha)(1 - \eta)\beta S \\
(1 - \rho)\theta - k & -(1 - \alpha)\beta(S + I(1 - \eta)D) - 2(\mu + v) & -(1 - \alpha)\beta S \\
k & (1 - \alpha)\beta(S + I(1 - \eta)D) & (1 - \alpha)\beta(S - (\mu + \theta) - k)
\end{pmatrix}
\]

with \( \Lambda = (1 - \alpha)\beta(S - 2(\mu + v) - \theta - (1 - \alpha)\beta(S + I(1 - \eta)D) \).

Setting

\[ P(S, I, D) = \text{diag}\left\{1, \frac{I}{D}, \frac{I}{D}\right\}, \]

then we have

\[ P_f P^{-1} = \text{diag}\left\{0, \frac{I'}{I}, \frac{I'}{D}, \frac{D'}{I}, \frac{D'}{D}\right\}, \]

and the matrix \( Q = P_f P^{-1} + P J^{[2]} P^{-1} \) can be presented in block form as follows:

\[ Q = \begin{pmatrix}
Q_{11} & Q_{12} \\
Q_{21} & Q_{22}
\end{pmatrix}, \]

where

\[ Q_{11} = (1 - \alpha)\beta S - (1 - \alpha)\beta(S + I(1 - \eta)D) - 2(\mu + v) - \theta, \]
\[ Q_{12} = \left(1 - \alpha)(1 - \eta)\beta S \frac{I}{D} \right), \]
\[ Q_{21} = \left(1 - \alpha)(1 - \eta)\beta S \frac{I}{D} \right), \]
\[ Q_{22} = (1 - \alpha)(1 - \eta)\beta S \frac{I}{D}. \]
Let \((x, y, z)\) be a vector in \(\mathbb{R}^3\). The author picks out a vector norm in \(\mathbb{R}^3\) as
\[
| (x, y, z) | = \max \{|x|, |y| + |z| \},
\]
for any vector \((x, y, z) \in \mathbb{R}^3\). Let \(m\) indicate the Lozinskii measure concerning this norm. We can then get
\[
m(Q) \leq \sup \{g_1, g_2\},
\]
with
\[
g_1 = m_1(Q_{11}) + |Q_{12}|, \\
g_2 = |Q_{21}| + m_1(Q_{22}).
\]
Here, \(|Q_{12}|\) and \(|Q_{21}|\) are matrix norms with respect to the \(L_1\) vector norm, and \(m_1\) denotes the Lozinskii measure with respect to the \(L_1\) norm. More specifically,
\[
m_1(Q_{11}) = (1 - \alpha) \beta S - (1 - \alpha) \beta (I + (1 - \eta)D) - 2(\mu + v) - \theta,
\]
\[
m_1(Q_{22}) = \frac{I'}{I} - \frac{D'}{D} - 2(\mu + v) - k + \sup[-\theta, 0],
\]
consequently
\[
g_1 = (1 - \alpha) \beta S - 2(\mu + v) - \theta - (1 - \alpha) \beta (I + (1 - \eta)D)
\]
\[
+ (1 - \alpha)(1 - \eta) \beta S \frac{D}{I}.
\]

Recalling that
\[
\frac{I'}{I} - (1 - \alpha) \beta S + (\mu + v + \theta) = (1 - \alpha)(1 - \eta) \beta S \frac{D}{I},
\]
thus \(g_1\) becomes
\[
g_1 = (1 - \alpha) \beta S - 2(\mu + v) - \theta - (1 - \alpha) \beta (I + (1 - \eta)D)
\]
\[
+ \frac{I'}{I} - (1 - \alpha) \beta S + (\mu + v + \theta)
\]
\[
= \frac{I'}{I} - (\mu + v) - (1 - \alpha) \beta (I + (1 - \eta)D)
\]
\[
\leq \frac{I'}{I} - (\mu + v).
\]
In addition,
\[
g_2 = \frac{I'}{I} - \frac{D'}{D} - 2(\mu + v) - k + (1 - \rho) \theta \frac{I}{D}
\]
\[
= \frac{I'}{I} - 2(\mu + v) - k + (1 - \rho) \theta \frac{D}{D} - \left[(1 - \rho) \theta \frac{I}{D} + (1 - S - I - D) \frac{k}{D} - (\mu + v) \right]
\]
\[
= \frac{I'}{I} - 2(\mu + v) - k + (\mu + v) - (1 - S - I - D) \frac{k}{D}
\]
\[
\leq \frac{I'}{I} - (\mu + v).
Therefore, 
\[
m(Q) \leq \frac{I'}{I} - (\mu + \nu).
\]
(B.17)

Thus, there exists \( T > 0 \) such that when \( t > T \),
\[
\frac{\ln I(t) - \ln I(0)}{t} < -\frac{\mu + \nu}{2}
\]
As a result,
\[
\frac{1}{T} \int_0^T m(Q) \, dt \leq \frac{1}{T} \int_0^T \left( \frac{I'}{T} - (\mu + \nu) \right) \, dt
\]
\[
= \frac{\ln I(t) - \ln I(0)}{t} - (\mu + \nu) < -\frac{\mu + \nu}{2},
\]
which implies that \( \bar{q}_2 < 0 \), thus completing the proof. □

Data Availability

The data used to support the findings of this study are included within the article.

Conflicts of Interest

The author declares that there are no conflicts of interest.

Authors’ Contributions

All the work was done by the author.

Acknowledgments

The author would like to acknowledge the Department of Mathematics and Computational Sciences at the University of Zimbabwe, particularly Professor Steady Mushayabasa.

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