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Article

Modelling the dynamics of syphilis infection with personal protection and treatment as optimal control strategies

Elvis Kobina Donkor[1,*], Bismark Ansu[2], Shaibu Osman[3], Dominic Otoo[1], Winnie Mokeira Onsongo[4], Ernest Yeboah Boateng [3]

[1] Department of Mathematics and Statistics, University of Energy and Natural Resources, Sunyani P. O. Box 214, Ghana
[2] Department of Mathematics and ICT, St. Ambrose College of Education, Dormaa-Akwamu P. O. Box 25, Ghana
[3] Department of Basic Sciences, School of Basic and Biomedical Sciences, University of Health and Allied Sciences, Ho PO Box 31, Ghana
[4] Department of Statistics and Actuarial Science, University of Ghana, Legon P. O. Box LG 25, Ghana
*** Corresponding author:** Elvis Kobina Donkor, elvis.donkor@uenr.edu.gh

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Abstract: Syphilis is a sexually transmitted infection which when left untreated would lead to major health problems. Syphilis can easily be contracted by direct contact with Syphilis sore during vaginal, anal, or oral sex. Syphilis can also be passed on from an infected mother to her unborn child. In this paper, a nonlinear deterministic model of Syphilis disease was constructed to determine the dynamics of Syphilis infections. The study deduced model's equilibria and analyzed the local and global stability of these equilibria. The model was extended to optimal control problem by adding time-dependent controls that helped characterize a range of possible controls that minimized the disease. The control system was solved qualitatively and numerically to evaluate the effectiveness of the considered controls using Pontryagin's Maximum Principle. The analysis indicated that strategies B and C are considered most effective as they substantially minimized the exposed, asymptomatic and symptomatic infectious. We recommend that stakeholders should consider strategy B and C in their effort to mitigate the disease from the population as they all have the same effect of substantially minimizing the exposed, symptomatic and asymptomatic populations.

Keywords: Syphilis infection; non-linear model; equilibria; Pontryagin's Maximum Principle; optimal control

1. Introduction

Syphilis can be contracted by direct contact with Syphilis sore during vaginal, anal, or oral sex. Syphilis can also be passed on from an infected mother to her unborn child. A person with primary Syphilis generally has a sore or sores at the original site of infection. Syphilis is a sexually transmitted infection which when left untreated would lead to major health implications [1].

According to the CDC (CDC, 2018), the rate of new cases of syphilis plummeted in the 1990s. In 2000, it reached an all-time low since reporting began in 1941. But the disease has been on the increase ever since. The rate of syphilis in the United States increased 71 from 2014 to 2018 [2].

If a woman becomes infected while she is pregnant, or becomes pregnant when she already has syphilis, it can be very dangerous for her baby if not treated. Infection in pregnancy can cause miscarriage, stillbirth or a serious infection in the baby (congenital syphilis). Screening for syphilis during pregnancy is offered to all pregnant women so the infection can be detected

and treated before it causes any serious problems [3].

Due to the resurgent of infectious diseases owing to drug resistance and other factors in these days, mathematical models have become imperative as they assist in providing better insight of how the transmission dynamics and identify various intervention strategies that could be adopted to minimize or eradicate infections [4].

Integrating mathematical models in the fight of infectious diseases have yielded a positive impact as several works have been able to estimate the most important threshold parameter: the basic reproduction number from the existing data of the considered disease [5–9].

In addition, most models have been able to predict the final size of the disease and the expected duration of which the disease would be abated. Infectious diseases have been one of man's enemy, as they have contributed to more deaths in the society than civil war. It is the main source of poverty, as it deprives a Nation of its human resource base [10–13].

Lajmanovich and Yorke [14] analyzed the spread of STIs in a population is highly non uniform. The mathematical model discussed takes this into account, splitting the population into n groups and stability properties were studied.

Authors in [15–17] investigated mathematically many models for the spread of infectious diseases in populations and applied to specific disease. Theorems involving the basic reproduction number R_0, the contact number, and the replacement number R are reviewed for the SIR epidemic and endemic models. Same concepts were applied to SEIR model.

Researchers in [18] examined two systematic methods presented to guide the construction of Lyapunov functions for general infectious disease models and was applicable to establishing their global dynamics.

A study conduct by authors in [19] findings reveal that mathematical modeling contributes to public health by planning to allow users to estimate future outcomes of events. Models can be used to mobilize support, strategically plan, and monitor key programmatic elements, but they can also help inform policy environments in which programs are conceptualized and implemented to achieve results.

Authors in [20] provided a mathematical model incorporate a dynamic risk of infection figure prominently in the study of infectious diseases epidemiology as a tool to inform public health policy. A study focused on the applications of transmission dynamics modeling, explains different modelling methodologies and defines commonly encountered terms to provide an introductory and conceptual understanding of the vocabulary and frameworks used in the study.

Researchers in [21] opine that many infectious diseases lead to re-infection. A study examined the relationship between the prevalence of repeat infection and the basic reproductive number R_0. A generic deterministic compartmental model of reinfection is solved to derive an analytic solution.

Authors in [22], a mathematical model that explores various modelling techniques to address debated or unanswered questions about the transmission dynamics of infectious diseases, in particular sexually transmitted ones was developed. A compartmental model that show that infection of uncoupled individuals is usually the predominant route, while transmission within discordant couples is also important, but to a lesser extent.

In a study by authors [23] developed mathematical model to depict the epidemiology of sexually transmitted infections which involved the incremental addition of various forms of biological and behavioral structures to simple mathematical terms. Progress was made by interdisciplinary work between clinician, epidemiologist, and mathematician. Aiming at improving the knowledge of how infection and diseases can best be controlled.

Garnett and Anderson [24] also examined a mathematical model which observes heterogeneity in sexual behavior and determine how individual variation influences epidemiological pattern within a population. In the model, different behaviors were treated separately.

The epidemiological role of migrants in the propagation of syphilis was studied. The syphilis incidence rate per 100,000 of people among legal migrants was 5 to 30 times higher than the population. The authors revealed the risk factors affecting the growth in the syphilis incidence rate among female migrants in labor [25].

2. Model description and formulation

The subsection formulates a compartmental model for Syphilis in a a population that is categorised into Susceptible, S_m, Exposed, E_m, Asymptomatic infectious, A_m, Symptomatic infectious, I_m and Recovered R_m compartments.

The total population N at any tie is denoted by $N = S_m + E_m + A_m + I_m + R_m$ The model assumes that individuals are recruited into the susceptible compartment at rate Λ. Susceptible individuals joins the exposed compartment as a result of their interaction with the asymptomatic and symptomatic infectious at rates $\gamma_\lambda \tau_\lambda$ and α_λ.

The exposed, leaves the compartment and joins the asymptomatic infectious at rate θ, a fraction ψ_m enters the asymptomatic infectious compartment, while the remaining fraction joins the symptomatic compartment. The asymptomatic and symptomatic infectious individuals recover by natural immunity at rates τ_1 and η_1 to the recovery compartment.

The symptomatic infectious individuals die as a result of the disease at a rate η_2. Due to the possibility of individuals loosing their immunity at the recovered class, the recovered individuals re-enters the susceptible class at rate ω. Natural death μ occurs at all compartments of the model. The schematic describing the model is presented in **Figure 1**. Hence, the nonlinear differential equation system is given by:

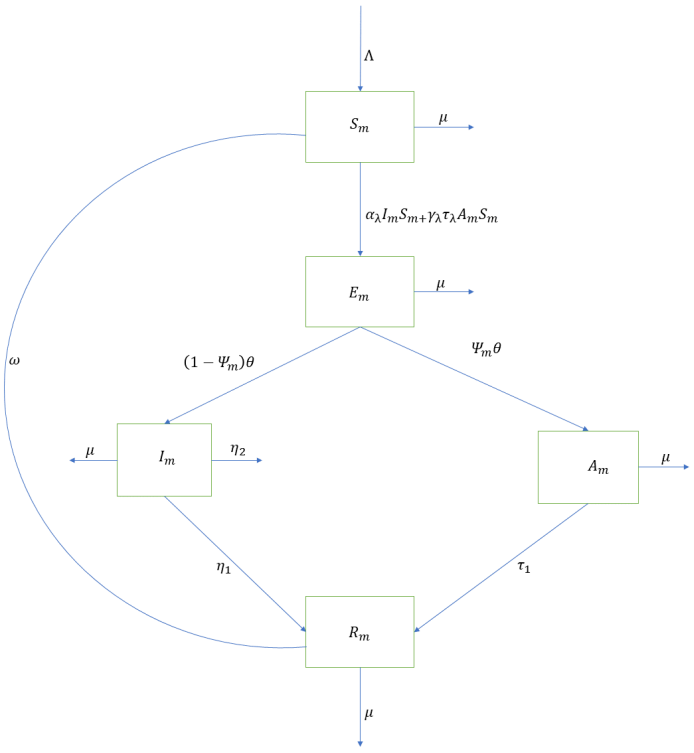


Figure 1. Schematic of the Syphilis model.

$$\begin{cases} \frac{d}{dt}S_m = \pi - \mu S_m + \omega R_m - \alpha_\lambda I_m S_m - \gamma_\lambda \tau_\lambda A_m S_m \\ \frac{d}{dt}E_m = \alpha_\lambda I_m S_m + \gamma_\lambda \tau_\lambda A_m S_m - \big((1-\psi_m)\theta + \psi_m\theta + \mu\big)E_m \\ \frac{d}{dt}A_m = \psi_m \theta E_m - (\tau_1 + \mu)A_m \\ \frac{d}{dt}I_m = (1-\psi_m)\theta E_m - (\eta_1 + \eta_2 + \mu)I_m \\ \frac{d}{dt}R_m = \eta_1 I_m + \tau_1 A_m - (\omega + \mu)R_m \end{cases} \tag{1}$$

with $S_{m_0} \geq 0, E_{m_0} \geq 0, A_{m_0} \geq 0, I_{m_0} \geq 0$ and $R_{m_0} \geq 0$

3. Boundedness of solution

Theorem 1. *The set* $\{S_m(t), E_m(t), A_m(t), I_m(t), R_m(t)\}$ *being the solution of the state system 1 with parameters which are not negatives is positive with the initial condition given by;*

$$\{S_{m_0} \geq 0, E_{m_0} \geq 0, A_{m_0} \geq 0, I_{m_0} \geq 0, R_{m_0} \geq 0\}.$$

Theorem 2. *The deterministic model system 1 has solutions bounded within the invariant region,* $\Theta \in R^5$ *given by;*

$$\Theta = \{(S_m, E_m, A_m, I_m, R_m) \in R_+^5 |\ S_m + E_m + A_m + I_m + R_m \leq \pi - \mu N\}$$

Proof. It can be confirmed that $N(t) = S_m + E_m + A_m + I_m + R_m$. Hence, the nonlinear equation of system 1 is given by:

$$\frac{d}{dt}N(t) = \pi - \eta_2 I_m - \mu N \tag{2}$$

$$\frac{d}{dt}N(t) \leq \pi - \mu N \tag{3}$$

Integrating inequality 3 gives;

$$N(t) = N(0)e^{-\mu t} + \frac{\pi}{\mu}(1 - e^{(-\mu t)}) \tag{4}$$

Therefore, we notice from equation 4 that the possible solution set of the state variables S_m, E_m, A_m, I_m, R_m is bounded and the model equation 1 is positively invariant in R_+^5. □

4. Disease free equilibrium

The disease free equilibrium of the Syphilis model 1 is given by;

$$(S_{m_0}, E_{m_0}, A_{m_0}, I_{m_0}, R_{m_0}) = \left(\frac{\pi}{\mu_\lambda}, 0, 0, 0, 0\right) \tag{5}$$

5. Basic reproduction number

It measures the status of the disease at any time. Thus, the basic reproduction number determines the possibility of disease persistence or die out of the population. It is denoted by R_0. When $R_0 < 1$, it is a clear indication that infection will be terminated. However, when $R_0 > 1$, the infection will remain in the population unless strategic efforts are implemented. In view of calculating the basic reproduction number, the method of [26] would be considered. Thus according to [26,27], R_0 is determined by;

$$R_0 = \rho(FV^{-1}) \tag{6}$$

The ρ is considered as the largest entry in the derivation of the next generation matrix of $R_0 = \rho(FV^{-1})$, where F is the coming infection into compartment i and v. Thus, the transfer of individuals out of compartment i by death. Technically, the R_0 becomes the largest eigenvalue of the matrix resulting from the partial derivative of 6.

What happens next is the infected compartments of model 1 is given by

$$\begin{aligned}
\frac{d}{dt}E_m &= \alpha_\lambda I_m S_m + \gamma_\lambda \tau_\lambda A_m S_m - \big((1-\psi_m)\theta + \psi_m\theta + \mu\big)E_m \\
\frac{d}{dt}A_m &= \psi_m\theta E_m - (\tau_1 + \mu)A_m \\
\frac{d}{dt}I_m &= (1-\psi_m)\theta E_m - (\eta_1 + \eta_2 + \mu)I_m
\end{aligned}$$

From the infective system, F and V are derived as follows;

$$\mathcal{F} = \begin{pmatrix} \alpha_\lambda I_m S_m + \gamma_\lambda \tau_\lambda A_m S_m \\ 0 \\ 0 \end{pmatrix}, \quad \mathcal{V} = \begin{pmatrix} \big((1-\psi_m)\theta + \psi_m\theta + \mu\big)E_m \\ -\psi_m\theta E_m + (\tau_1 + \mu)A_m \\ -(1-\psi_m)\theta E_m + (\eta_1 + \eta_2 + \mu)I_m \end{pmatrix}$$

When $\mathcal{F}$ is evaluated at $(S_{m_0}, E_{m_0}, A_{m_0}, I_{m_0}, R_{m_0}) = (\frac{\pi}{\mu_\lambda}, 0, 0, 0, 0)$, we get;

$$F = \begin{pmatrix} 0 & \frac{\alpha_\lambda}{\mu_\lambda}\pi & \frac{\alpha_\lambda}{\mu_\lambda}\tau_\lambda\pi \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix}$$

In the same way, evaluating $\mathcal{V}$ at $(S_{m_0}, E_{m_0}, A_{m_0}, I_{m_0}, R_{m_0}) = (\frac{\pi}{\mu_\lambda}, 0, 0, 0, 0)$ gives;

$$V = \begin{pmatrix} \big((1-\psi_m)\theta + \psi_m\theta + \mu\big) & 0 & 0 \\ -(1-\psi_m)\theta & (\eta_1 + \eta_2 + \mu) & 0 \\ -\psi_m\theta & 0 & (\tau_1 + \mu) \end{pmatrix}$$

Hence, applying the next generation method of [28], the basic reproduction number of model system 1 is given as;

$$R_0 = \frac{\alpha_\lambda(1-\psi_m)\theta\pi}{\mu_\lambda\big((1-\psi_m)\theta + \psi_m\theta + \mu\big)(\eta_1 + \eta_2 + \mu)} + \frac{\gamma_\lambda\tau_\lambda\pi}{\mu_\lambda\big((1-\psi_m)\theta + \psi_m\theta + \mu\big)(\tau_1 + \mu)}$$

6. Endemic equilibrium

Consider model system 1, there exists a unique endemic equilibrium given by;

$$S_m^* = \frac{\pi - \mu S_m^* + \omega R_m^*}{\alpha_\lambda I_m^* + \gamma_\lambda \tau_\lambda A_m^*}$$

$$E_m^* = \frac{\alpha_\lambda I_m^* S_m^* + \gamma_\lambda \tau_\lambda A_m^* S_m^*}{\big((1-\psi_m)\theta + \psi_m\theta + \mu\big)}$$

$$A_m^* = \frac{\psi_m\theta E_m^*}{(\tau_1 + \mu)}$$

$$I_m^* = \frac{(1-\psi_m)\theta E_m^*}{(\eta_1+\eta_2+\mu)}$$

$$R_m^* = \frac{\eta_1 I_m + \tau_1 A_m}{(\omega+\mu)}$$

7. Disease free equilibrium stability analysis

Using the Hartman-Grobmann theorem as discussed in [29], we linearise the nonlinear system of model system 1 as:

$$J_{df} = \begin{pmatrix} A & 0 & -\alpha_\lambda S_m & -\gamma_\lambda \tau_\lambda S_m & \omega \\ \alpha_\lambda I_m + \gamma_\lambda \tau_\lambda A_m & B & \alpha_\lambda S_m & \gamma_\lambda \tau_\lambda S_m & 0 \\ 0 & (1-\psi_m)\theta & -(\eta_1+\eta_2+\mu) & 0 & 0 \\ 0 & \psi_m\theta & 0 & -(\tau_1+\mu) & 0 \\ 0 & 0 & \eta_1 & \tau_1 & -(\omega+\mu) \end{pmatrix}$$

where $A = -\mu_\lambda - \alpha_\lambda I_m - \gamma_\lambda \tau_\lambda A_m$ and $B = -\Big((1-\psi_m)\theta + \psi_m\theta + \mu\Big)$

When J_{df} is evaluated at $(S_{m0}, E_{m0}, I_{m0}, A_{m0}, R_{m0}) = (\frac{\pi}{\mu_\lambda}, 0, 0, 0, 0)$, we get

$$J_{D_0} = \begin{pmatrix} -\mu_\lambda & 0 & -\alpha_\lambda \frac{\pi}{\mu_\lambda} & -\gamma_\lambda \tau_\lambda \frac{\pi}{\mu_\lambda} & \omega \\ 0 & -\Big((1-\psi_m)\theta + \psi_m\theta + \mu\Big) & \alpha_\lambda \frac{\pi}{\mu_\lambda} & \gamma_\lambda \tau_\lambda \frac{\pi}{\mu_\lambda} & 0 \\ 0 & (1-\psi_m)\theta & -(\eta_1+\eta_2+\mu) & 0 & 0 \\ 0 & \psi_m\theta & 0 & -(\tau_1+\mu) & 0 \\ 0 & 0 & \eta_1 & \tau_1 & -(\omega+\mu) \end{pmatrix}$$

Clearly, $\lambda_1 = -\mu_\lambda$ and $\lambda_2 = -(\omega+\mu)$. It follows that the remaining matrix becomes;

$$\begin{pmatrix} -\Big((1-\psi_m)\theta + \psi_m\theta + \mu\Big) & \alpha_\lambda \frac{\pi}{\mu_\lambda} & \gamma_\lambda \tau_\lambda \frac{\pi}{\mu_\lambda} \\ (1-\psi_m)\theta & -(\eta_1+\eta_2+\mu) & 0 \\ \psi_m\theta & 0 & -(\tau_1+\mu) \end{pmatrix}$$

Let $a = -\Big((1-\psi_m)\theta + \psi_m\theta + \mu\Big)$, $b = \alpha_\lambda \frac{\pi}{\mu_\lambda}$, $c = \gamma_\lambda \tau_\lambda \frac{\pi}{\mu_\lambda}$, $d = (1-psi_m)\theta$

$e = -(\eta_1+\eta_2+\mu)$, $f = \psi_m\theta$, $g = -(\tau_1+\mu)$

The characteristic equation is given by;

$$Y_1^3 + a_0 Y_2^2 + a_1 Y_1 + a_2 \tag{7}$$

where

$$a_0 = (a+e+g)$$

$$a_1 = (ae - bd + ag - cf + eg)$$

$$a_2 = (aeg - bdg - cef)$$

8. Global stability analysis of the disease-free equilibrium

The present subsection gives a discussion of the method of global asymptotic stability analysis of Castillo-Chavez's et al. [30,31] for model 1 as follows;

$$\frac{dh_1}{dt} = A(h_1, h_2),$$

$$\frac{dh_2}{dt} = B(h_1, h_2),$$

where h_1 and h_2 represents respectively the number of uninfected and infected individuals. It follows that $h_1 = \big(S_m, R_m\big) \in R^2$ and $h_2 = \big(E_m, I_m, A_m\big) \in R^3$. Then, the disease-free equilibrium E_0 for the model system 1 is denoted by $E_0 = (h_1^0, 0)$. Hence, the global stability at E_0 would be satisfied on below conditions;

- Given $\dfrac{dh_1}{dt} = A(h_1, 0), h_1^0$ is globally asymptotically stable.
- $B(h_1, h_2) = Dh_2 - \hat{\mathrm{B}}(h_1, h_2)$, where $\hat{\mathrm{B}}(h_1, h_2) \geq 0$ for $(h_1, h_2) \in \tau_1$

where $D = Ph_2B(h_1^0, 0)$ is an M-matrix, with a positive off-diagonal entries and τ_1 is the feasible biological region of model 1. When the above conditions are satisfied by model system 1, then the underlying theorem holds.

Theorem 3. *When $R_0 < 1$ and th two conditions above are satisfied, then, the equilibrium point $E_0 = (h_1^0, 0)$ is globally asymptotically stable.*

Proof. With model 1, we deduces;

$$\frac{dh_1}{dt} = B(h_1, h_2)$$

$$\frac{dh_1}{dt} = \begin{pmatrix} \pi - \mu_\lambda S_m - \alpha_\lambda I_m S_m - \gamma_\lambda \tau_\lambda A_m S_m + \omega R_m \\ \eta_1 I_m + \tau_1 A_m - \mu R_m - \omega R_m \end{pmatrix},$$

Hence $B(h_1, 0)$ becomes, $H(h_1, 0) = \begin{pmatrix} \pi - \mu_\lambda S_{m_0} \\ 0 \end{pmatrix}$, and $B(h_1, h_2) = Dh_2 - \hat{\mathrm{B}}(h_1, h_2)$ is given by $Dh_2 - \hat{\mathrm{B}}(h_1, h_2)$, where $Dh_2 - \hat{\mathrm{B}}(h_1, h_2) =$ is;

$$\begin{pmatrix} -\big((1-\psi_m)\theta + \psi_m\theta + \mu\big) & \alpha_\lambda S_{m_0} & \gamma_\lambda \tau_\lambda S_{m_0} \\ (1-\psi_m)\theta & -(\eta_1 + \eta_2 + \mu) & 0 \\ \psi_m\theta & 0 & -(\tau_1 + \mu) \end{pmatrix} \begin{pmatrix} E_m \\ I_m \\ A_m \end{pmatrix}$$

$$- \begin{pmatrix} \alpha_\lambda I_m(S_{m_0} - S_m) & \gamma_\lambda \tau_\lambda A_m(S_0 - S_m) & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0, \end{pmatrix}$$

with

$$D = \begin{pmatrix} -\big((1-\psi_m)\theta + \psi_m\theta + \mu\big) & \alpha_\lambda S_{m_0} & \gamma_\lambda \tau_\lambda S_{m_0} \\ (1-\psi_m)\theta & -(\eta_1 + \eta_2 + \mu) & 0 \\ \psi_m\theta & 0 & -(\tau_1 + \mu) \end{pmatrix}$$

and

$$B(y_1, y_2) = \begin{pmatrix} \alpha_\lambda I_m(S_{m_0} - S_m) & \gamma_\lambda \tau_\lambda A_m(S_0 - S_m) & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix}$$

As can be seen, the total population of model 1 is bounded by S_{m_0}. Thus $S_m, E_m, I_m, A_m, R_m \leq S_{m_0}$, and $\alpha_\lambda S_m \leq \alpha_\lambda S_{m_0}$, $\gamma_\lambda \tau_\lambda S_m \leq \gamma_\lambda \tau_\lambda S_{m_0}$ which implies $\hat{\mathrm{B}}(h_1, h_2)$ is positive

definite. Additionally, matrix D is an M-matrix, with the off-diagonal entries positive. Hence, the requirement of the two conditions are met, which is a proof of the globally asymptotically stability of E_0. □

9. Stability analysis of endemic equilibrium

This section studies systematically the local and global stability of the Syphilis model 1 at the EDE. Lyapunov stability theorem is therefore applied to study the global stability [32–34].

Theorem 4. *The Syphilis endemic equilibrium* $EDE = (S_m^*, E_m^*, I_m^*, A_m^*, R_m^*)$ *for the model system (1) is locally asymptomatically stable when* $R_0 > 1$.

Proof. The Jacobian J_{EDE} at the endemic equilibrium $(S_m^*, E_m^*, I_m^*, A_m^*, R_m^*)$ is analyzed as follows:

$$J_{EDE} = \begin{pmatrix} A & 0 & -\alpha_\lambda S_m & -\gamma_\lambda \tau_\lambda S_m & \omega \\ \alpha_\lambda I_m + \gamma_\lambda \tau_\lambda A_m & B & \alpha_\lambda S_m & \gamma_\lambda \tau_\lambda S_m & 0 \\ 0 & (1-\psi_m)\theta & -(\eta_1+\eta_2+\mu) & 0 & 0 \\ 0 & \psi_m \theta & 0 & -(\tau_1+\mu) & 0 \\ 0 & 0 & \eta_1 & \tau_1 & -(\omega+\mu) \end{pmatrix}$$

where $A = -\mu_\lambda - \alpha_\lambda I_m - \gamma_\lambda \tau_\lambda A_m$ and $B = -\Big((1-\psi_m)\theta + \psi_m\theta + \mu\Big)$.

When the Jacobian is evaluated at $(S_m^*, E_m^*, I_m^*, A_m^*, R_m^*)$;

$$J_{EDE} = \begin{pmatrix} A_1 & 0 & -\alpha_\lambda S_m^* & -\gamma_\lambda \tau_\lambda S_m^* & \omega \\ \alpha_\lambda I_m^* + \gamma_\lambda \tau_\lambda A_m^* & B_1 & \alpha_\lambda S_m^* & \gamma_\lambda \tau_\lambda S_m^* & 0 \\ 0 & (1-\psi_m)\theta & -(\eta_1+\eta_2+\mu) & 0 & 0 \\ 0 & \psi_m \theta & 0 & -(\tau_1+\mu) & 0 \\ 0 & 0 & \eta_1 & \tau_1 & -(\omega+\mu) \end{pmatrix}$$

where $A_1 = -\mu_\lambda - \alpha_\lambda I_m^* - \gamma_\lambda \tau_\lambda A_m^*$ and $B_1 = -\Big((1-\psi_m)\theta + \psi_m\theta + \mu\Big)$. □

We choose
$k_1, k_2, k_3, k_4, k_5, k_6, k_7, k_8, k_9, k_{10}, k_{11}, k_{12}, k_{13}, k_{14}, k_{15}$ such that
$k_1 = -\mu_\lambda - \alpha_\lambda I_m^* - \gamma_\lambda \tau_\lambda A_m^*$, $k_2 = -\alpha_\lambda S_m^*$, $k_3 = -\gamma_\lambda \tau_\lambda S_m^*$, $k_4 = \omega$, $k_5 = \alpha_\lambda I_m^* + \gamma_\lambda \tau_\lambda A_m^*$, $k_6 = -\Big((1-\psi_m)\theta + \psi_m\theta + \mu\Big)$, $k_7 = \alpha_\lambda S_m^*$, $k_8 = \gamma_\lambda \tau_\lambda S_m^*$, $k_9 = (1-d)\theta$, $k_{10} = -(\eta_1+\eta_2+\mu)$, $k_{11} = \psi_m\theta$, $k_{12} = -(\tau_1+\mu)$, $k_{13} = \eta_1$, $k_{14} = \tau_1$, $k_{15} = -(\omega+\mu)$.

then, the characteristic equation of model 1 at the endemic equilibrium becomes;

$$T^5 + p_0 T^4 + p_1 T^3 + p_2 T^2 + p_3 T_1 + p_4 = 0 \tag{8}$$

where

$$p_0 = k_1 + k_6 + k_{10} + k_{12} + k_{15}$$

$$P_1 = k_1k_6 + k_1k_{10} + k_1k_{12} + k_6k_{10} - k_7k_9 + k_1k_{15} + k_6k_9 - k_8k_{12} + k_6k_{15} + k_{10}k_{12} + k_{10}k_{15} + k_{12}k_{15}$$

$$p_2 = k_2k_5k_9 + k_1k_6k_{10} - k_1k_7k_9 + k_1k_6k_{12} + k_3k_5k_{11} - k_1k_8k_{11} + k_1k_6k_{15} + k_1k_{10}k_{12} + k_1k_{10}k_{15} + k_6k_{10}k_{12} - k_7k_9k_{12} + k_1k_{12}k_{15} - k_8k_{10}k_{11} + k_6k_{10}k_{15} - k_7k_9k_{15} + k_6k_{12}k_{15} - k_8k_{11}k_{15} + k_{10}k_{12}k_{15}$$

$$p_3 = k_1k_6k_{10}k_{15} - k_1k_7k_9k_{15} - k_4k_5k_{11}k_{14} + k_1k_6k_{12}k_{15} + k_3k_5k_{11}k_{15} - k_1k_8k_{11}k_{15} + k_1k_{10}k_{12}k_{15} + k_6k_{10}k_{12}k_{15} - k_7k_9k_{12}k_{15} - k_8k_{10}k_{11}k_{15} + k_2k_5k_9k_{12} + k_1k_6k_{10}k_{12} - k_1k_7k_9k_{12} + k_3k_5k_{10}k_{11} - k_1k_8k_{10}k_{11} - k_4k_5k_9k_{13} + k_2k_5k_9k_{15}$$

$$p_4 = -k_4k_5k_9k_{12}k_{13} + k_2k_5k_9k_{12}k_{15} - k_4k_5k_{10}k_{11}k_{14} + k_1k_6k_{10}k_{12}k_{15} - k_1k_7k_{12}k_9k_{15} + k_3k_5k_{10}k_{11}k_{15} - k_1k_8k_{10}k_{11}k_{15}$$

Based on Routh-Hurwitz stability criterion [35], for the characteristics equation 8, is given by;

$$\mathcal{T} = \begin{bmatrix} T_1 & T_3 & T_5 \\ T_0 & T_2 & T_4 \\ 0 & T_1 & T_3 \\ 0 & T_0 & T_2 \\ 0 & 0 & T_1 \\ 0 & 0 & T_0 \\ 0 & 0 & 0 \end{bmatrix} > 0$$

The condition requires that the characteristics equation 8 have all positive coefficients, indicating that the eigenvalues are negatives. The fulfilment of this condition implies that the EDE is stable. Otherwise, it is unstable.

Theorem 5. *When $R_0 \geq 1$, the endemic equilibrium $(S_m^*, E_m^*, I_m^*, A_m^*, R_m^*)$ of model 1 is globally stable when $S_m = S_m^*$, $E_m = E_m^*$, $I_m = I_m^*$, $A_m = A_m^*$, and $R_m = R_m^*$, otherwise unstable.*

Proof. We consider a Lyapunov function of the form

$$L_{ya} = \left(S_m - S_m^* - S_m^* ln\left(\frac{S_m}{S_m^*}\right)\right) + \left(E_m - E_m^* - E_m^* ln\left(\frac{E_m}{E_m^*}\right)\right) + \left(I_m - I_m^* - I_m^* ln\left(\frac{I_m}{I_m^*}\right)\right) + \left(A_m - A_m^* - A_m^* ln\left(\frac{A_m}{A_m^*}\right)\right) + \left(R_m - R_m^* - R_m^* ln\left(\frac{R_m}{R_m^*}\right)\right).$$

When L_{ya} is differentiated with respect to time;

$$\frac{dL_{ya}}{dt} = \left(\frac{S_m - S_m^*}{S_m}\right)\frac{dS_m}{dt} + \left(\frac{E_m - E_{Hh}^*}{E_m}\right)\frac{dE_m}{dt} + \left(\frac{I_m - I_m^*}{I_m}\right)\frac{dI_m}{dt} + \left(\frac{A_m - A_m^*}{A_m}\right)\frac{dA_m}{dt} + \left(\frac{R_m - R_m^*}{R_m}\right)\frac{dR_m}{dt} \quad (9)$$

$$\frac{dL_{ya}}{dt} = \left(\frac{S_m - S_m^*}{S_m}\right)\left(\pi - \mu(S_m - S_m^*) + \omega(R_m - R_m^*) - \alpha_\lambda(I_m - I_m^*)(S_m - S_m^*) - \gamma_\lambda\tau_\lambda(A_m - A_m^*)(S_m - S_m^*)\right)$$
$$+ \left(\frac{E_m - E_{Hh}^*}{E_m}\right)\left(\alpha_\lambda(I_m - I_m^*)(S_m - S_m^*) + \gamma_\lambda\tau_\lambda(A_m - A_m^*)(S_m - S_m^*) - \big((1-\psi_m)\theta + \psi_m\theta + \mu\big)(E_m - E_m^*)\right)$$
$$+ \left(\frac{I_m - I_m^*}{I_m}\right)\left((1-\psi_m)\theta(E_m - E_m^*) - (\eta_1 + \eta_2 + \mu)(I_m - I_m*)\right)$$
$$+ \left(\frac{A_m - A_m^*}{A_m}\right)\left(\psi_m\theta(E_m - E_m^*) - (\tau_1 + \mu)(A_m - A_m^*)\right)$$
$$+ \left(\frac{R_m - R_m^*}{R_m}\right)\left(\eta_1(I_m - I_m^*) + \tau_1(A_m - A_m^*) - (\omega + \mu)(R_m - R_m^*)\right) \tag{10}$$

$$\frac{dL_{ya}}{dt} = \left(\pi\left(\frac{S_m - S_m^*}{S_m}\right) - \mu\left(\frac{(S_m - S_m^*)^2}{S_m}\right) + \omega(R_m - R_m^*)\left(\frac{S_m - S_m^*}{S_m}\right) - \alpha_\lambda(I_m - I_m^*)\left(\frac{(S_m - S_m^*)^2}{S_m}\right)\right.$$
$$\left. - \gamma_\lambda\tau_\lambda(A_m - A_m^*)\left(\frac{(S_m - S_m^*)^2}{S_m}\right)\right)$$
$$+ \left(\alpha_\lambda(I_m - I_m^*)(S_m - S_m^*)\left(\frac{E_m - E_{Hh}^*}{E_m}\right)\right.$$
$$+ \gamma_\lambda\tau_\lambda(A_m - A_m^*)(S_m - S_m^*)\left(\frac{E_m - E_{Hh}^*}{E_m}\right)$$
$$\left. - \big((1-\psi_m)\theta + \psi_m\theta + \mu\big)\left(\frac{(E_m - E_{Hh}^*)^2}{E_m}\right)\right)$$
$$+ \left((1-\psi_m)\theta(E_m - E_m^*)\left(\frac{I_m - I_m^*}{I_m}\right)\right. \tag{11}$$
$$\left. - (\eta_1 + \eta_2 + \mu)\left(\frac{(I_m - I_m^*)^2}{I_m}\right)\right)$$
$$+ \left(\psi_m\theta(E_m - E_m^*)\left(\frac{A_m - A_m^*}{A_m}\right)\right.$$
$$\left. - (\tau_1 + \mu)\left(\frac{(A_m - A_m^*)^2}{A_m}\right)\right)$$
$$+ \left(\eta_1(I_m - I_m^*)\left(\frac{R_m - R_m^*}{R_m}\right)\right.$$
$$+ \tau_1(A_m - A_m^*)\left(\frac{R_m - R_m^*}{R_m}\right)$$
$$\left. - (\omega + \mu)\left(\frac{(R_m - R_m^*)^2}{R_m}\right)\right)$$

$$\frac{dL_{ya}}{dt} = \left(\pi - \pi\left(\frac{S_m^*}{S_m}\right) - \mu\left(\frac{(S_m - S_m^*)^2}{S_m}\right) + \omega(R_m - R_m^*)\left(\frac{S_m - S_m^*}{S_m}\right) - \alpha_\lambda(I_m - I_m^*)\left(\frac{(S_m - S_m^*)^2}{S_m}\right)\right.$$
$$\left. - \gamma_\lambda\tau_\lambda(A_m - A_m^*)\left(\frac{(S_m - S_m^*)^2}{S_m}\right)\right) + \left(\alpha_\lambda(I_m - I_m^*)(S_m - S_m^*)\left(\frac{E_m - E_{Hh}^*}{E_m}\right)\right.$$
$$\left. + \gamma_\lambda\tau_\lambda(A_m - A_m^*)(S_m - S_m^*)\left(\frac{E_m - E_{Hh}^*}{E_m}\right) - \left((1-\psi_m)\theta + \psi_m\theta + \mu\right)\left(\frac{(E_m - E_{Hh}^*)^2}{E_m}\right)\right)$$
$$+ \left((1-\psi_m)\theta(E_m - E_m^*)\left(\frac{I_m - I_m^*}{I_m}\right) - (\eta_1 + \eta_2 + \mu)\left(\frac{(I_m - I_m^*)^2}{I_m}\right)\right)$$
$$+ \left(\psi_m\theta(E_m - E_m^*)\left(\frac{A_m - A_m^*}{A_m}\right) - (\tau_1 + \mu)\left(\frac{(A_m - A_m^*)^2}{A_m}\right)\right)$$
$$+ \left(\eta_1(I_m - I_m^*)\left(\frac{R_m - R_m^*}{R_m}\right) + \tau_1(A_m - A_m^*)\left(\frac{R_m - R_m^*}{R_m}\right) - (\omega + \mu)\left(\frac{(R_m - R_m^*)^2}{R_m}\right)\right)$$

With careful manipulation of the above algebraic expression;

$$\frac{dL_{ya}}{dt} = P_1 - P_2$$

where

$$P_1 = \pi + \omega(R_m - R_m^*)\left(\frac{S_m - S_m^*}{S_m}\right) + \alpha_\lambda(I_m - I_m^*)(S_m - S_m^*)\left(\frac{E_m - E_{Hh}^*}{E_m}\right)$$
$$+ \gamma_\lambda\tau_\lambda(A_m - A_m^*)(S_m - S_m^*)\left(\frac{E_m - E_{Hh}^*}{E_m}\right) + (1-\psi_m)\theta(E_m - E_m^*)\left(\frac{I_m - I_m^*}{I_m}\right)$$
$$+ \psi_m\theta(E_m - E_m^*)\left(\frac{A_m - A_m^*}{A_m}\right) + \eta_1(I_m - I_m^*)\left(\frac{R_m - R_m^*}{R_m}\right) + \tau_1(A_m - A_m^*)\left(\frac{R_m - R_m^*}{R_m}\right)$$

and

$$P_2 = \pi\left(\frac{S_m^*}{S_m}\right) + \mu\left(\frac{(S_m - S_m^*)^2}{S_m}\right) + \alpha_\lambda(I_m - I_m^*)\left(\frac{(S_m - S_m^*)^2}{S_m}\right)$$
$$+ \gamma_\lambda\tau_\lambda(A_m - A_m^*)\left(\frac{(S_m - S_m^*)^2}{S_m}\right) + \left((1-\psi_m)\theta + \psi_m\theta + \mu\right)\left(\frac{(E_m - E_{Hh}^*)^2}{E_m}\right)$$
$$+ (\eta_1 + \eta_2 + \mu)\left(\frac{(I_m - I_m^*)^2}{I_m}\right) + (\tau_1 + \mu)\left(\frac{(A_m - A_m^*)^2}{A_m}\right) + (\omega + \mu)\left(\frac{(R_m - R_m^*)^2}{R_m}\right)$$

Evidently, the inequality $P_1 \leq P_2$ can be affirmed. It can therefore be verified that $\frac{dL_{ya}}{dt} \leq 0$ when $P_1 \leq P_2$. Hence $\frac{dL_{ya}}{dt} = 0$, when $S_m = S_m^*$, $E_m = E_m^*$, $A_m = A_m^*$,$I_m = I_m^*$ and $R_m = R_m^*$. Hence the largest compact invariant set $\{(S_m, E_m, A_m, I_m, R_m) \in \Upsilon : dL_{ya}dt\} = 0$ is the singleton endemic equilibrium. Hence, from [36–38], EE is globally asymptotically stable. □

10. Optimal control model formulation

Identifying strategies that will help to mitigate the Syphilis infection, we introduced two controls that are time dependent by modifying model system 1. These are personal protection, u_1 and treatment, u_2 controls to examine their impact on the disease. Hence, the control system

becomes:

$$\begin{cases} \frac{d}{dt}S_m = \pi - \mu S_m + \omega R_m - (1-u_1)\alpha_\lambda I_m S_m - (1-u_1)\gamma_\lambda \tau_\lambda A_m S_m \\ \frac{d}{dt}E_m = (1-u_1)\alpha_\lambda I_m S_m + (1-u_1)\gamma_\lambda \tau_\lambda A_m S_m + \big((1-\psi_m)\theta + \psi_m\theta + \mu\big)E_m \\ \frac{d}{dt}A_m = \psi_m \theta E_m - (\tau_1 + \mu)A_m \\ \frac{d}{dt}I_m = (1-\psi_m)\theta E_m - (\eta_1 + \eta_2 + u_2 + \mu)I_m \\ \frac{d}{dt}R = \eta_1 I_m + \tau_1 A_m + u_2 I_m - (\omega + \mu)R_m \end{cases} \tag{12}$$

The objective functional $\mathcal{J}$ that minimizes the exposed and infectious individuals and maximizes the recovered through treatment control of u_2 is denoted by $\mathcal{J}$ is given by:

$$J(u_1, u_2) = \int_0^{t_f} \left[B_1 E_m + B_2 I_m + B_3 A_m + \frac{1}{2}(b_1 u_1^2 + b_2 u_2^2) \right].dt \tag{13}$$

The quantities B_1, B_2 and B_3 are the coefficients of the exposed, symptomatic and asymptomatic individuals. The terms $\frac{b_1 u_1^2}{2}$ and $\frac{b_2 u_2^2}{2}$ are the cost related to minimizing the exposed, symptomatic and asymptomatic individuals. It follows that, we seek an optimal u_1^*, u_2^* such that:

$$\mathcal{J}(u_1^*, u_2^*) = min\{J(u_1, u_2) : (u_1, u_2) \in U\} \tag{14}$$

with

$$U = \{(u_1, u_2) | \text{ where } 0 \leq u_1, u_2 \leq 1, \text{is Lebesgue measurable}\} \tag{15}$$

The Pontryagin's maximum principle [39] converts 21 and 13 into a minimization of the Hamiltonian (H), where

$$\begin{aligned} H = &\left[B_1 E_m + B_2 I_m + B_3 A_m + \frac{1}{2}(b_1 u_1^2 + b_2 u_2^2) \right] + \lambda_1\{\pi - \mu S_m + \omega R_m - (1-u_1)\alpha_\lambda I_m S_m \\ &- (1-u_1)\gamma_\lambda \tau_\lambda A_m S_m\} + \lambda_2\{(1-u_1)\alpha_\lambda I_m S_m + (1-u_1)\gamma_\lambda \tau_\lambda A_m S_m + \big((1-\psi_m)\theta + \\ &\psi_m\theta + \mu\big)E_m\} + \lambda_3\{\psi_m \theta E_m - (\tau_1 + \mu)A_m\} + \lambda_4\{(1-\psi_m)\theta E_m - (\eta_1 + \eta_2 + u_2 + \mu)I_m\} \\ &+ \lambda_5\{\eta_1 I_m + \tau_1 A_m + u_2 I_m - (\omega + \mu)R_m\} \end{aligned} \tag{16}$$

Theorem 6. *There exists an optimal control $U^* = (u_1^*, u_2^*) \in U$ such that*

$$\mathcal{J}(u_1^*, u_2^*) = \min_U \mathcal{J}(u_1, u_2), \tag{17}$$

subject to the control system 21 with the initial conditions.

Proof. As evidence in [40], the existence of the optimal control can be be proved. The state and control variables are positive values. It follows that in minimizing the control problem, the necessary and convexity of the objective functional in u_1 and u_2 are satisfied. The control space $U = \{u | u_1, u_2$ are measurable, $0 \leq u_1, u_2 \leq u_m ax \ < \infty, \ t \in [0, t_f]\}$ is also convex and closed by definition. The optimal system is bounded which confirms the compactness needed for the existence of the optimal control. Further, the integrand in the functional 13, $\left[B_1 E_m + B_2 I_m + B_3 A_m + \frac{1}{2}(b_1 u_1^2 + b_2 u_2^2) \right]$ is convex on the control u. Also, we see that there exist a

constant $p > 1$, positive numbers u_1 and u_2 such that, $J(u_1, u_2) \geq u_1 \left(|u_1|^2 + |u_2|^2\right)^{\frac{p}{2}} - u^2$. □

Therefore we conclude that there exist an optimal control.

Hence, the derivation of optimal solution is done by applying the Pontyagins's maximum principle [39] to the Hamiltonian 16 such that given (x, u) is an optimal solution of the optimal control problem, then there exist a non-trivial vector function $\lambda = (\lambda_1 \ \ldots \ \lambda_5)$ satisfying the below equation;

$$\begin{aligned} \frac{dx}{dt} &= \frac{\partial H(t, x, u, \lambda)}{\partial \lambda} \\ 0 &= \frac{\partial H(t, x, u, \lambda)}{\partial u} \\ \frac{d\lambda}{dt} &= \frac{\partial H(t, x, u, \lambda)}{\partial x} \end{aligned} \tag{18}$$

Hence, the necessary condition is applied to the Hamiltonian 16.

Theorem 7. *Given that S_m, E_m, A_m, I_m and R_m are optimal state solutions with associated control variables (u_1^*, u_2^*) for the optimal control problem 21 and 13, then there exist adjoint variables λ_i for $i = 1, \ldots, 5$, satisfying;*

$$\begin{aligned} \frac{d}{dt}\lambda_1 &= \mu_\lambda \lambda_1 + (\lambda_1 - \lambda_2)(1 - u_1)\alpha_\lambda I_m + (\lambda_1 - \lambda_2)(1 - u_1)\gamma_\lambda \tau_\lambda Am \\ \frac{d}{dt}\lambda_2 &= -B_1 + (1 - \psi_m)\theta(\lambda_2 - \lambda_3) + (\lambda_2 - \lambda_4)\psi_m \theta + \mu_\lambda \\ \frac{d}{dt}\lambda_3 &= -B_2 + (\lambda_1 - \lambda_2)(1 - u_1)\alpha_\lambda S_m + (\lambda_3 - \lambda_5)n_1 + (\lambda_3 - \lambda_5)u_2 + (n_2 + \mu_\lambda)\lambda_3 \\ \frac{d}{dt}\lambda_4 &= -B_3 + (\lambda_1 - \lambda_2)(1 - u_2)\gamma_\lambda \tau_\lambda S_m + (\lambda_4 - \lambda_5)\tau_1 + \mu_\lambda \lambda_4 \\ \frac{d}{dt}\lambda_5 &= \mu_\lambda \lambda_5 + (\lambda_5 - \lambda_4)\omega \end{aligned}$$

with boundary condition;

$$\lambda_i(t_f) = 0, \ \mathrm{i} = 1, 2, \ldots, 5 \tag{19}$$

In addition, the optimal control u_1^* and u_2^* are given by

$$\begin{cases} u_1' &= min\left\{1, max\left\{0, \left(\lambda_2 - \lambda_1\right)\dfrac{\alpha_\lambda I_m S_m}{d_1} + (\lambda_2 - \lambda_1)\dfrac{\gamma_\lambda \tau_\lambda A_m S_m}{d_1}\right)\right\}\right\} \\ u_2' &= min\left\{1, max\left\{0, (\lambda_3 - \lambda_5)\dfrac{\alpha_\lambda I_m}{d_2}\right\}\right\} \end{cases} \tag{20}$$

Proof. The adjoint and transversality conditions are derived by utilizing the Hamiltonian 16. Thus we equate $S_m = S_m^*, E_m = E_m^*, A_m = A_m^*, I_m = I_m^*$ and $R_m = R_m^*$ and differentiating the Hamiltonian with respect to S_m, E_m, A_m, I_m and R_m to obtain **??**. Further, the equations $\dfrac{\partial H}{\partial u_1} = 0$ and $\dfrac{\partial H}{\partial u_2} = 0$ are determined on the interior of control set and using the optimality conditions and the property of the control space u_1 and u_2, we can determine. From 20, we can characterize the control which is found by solving the optimality system. In solving the optimality system, the transversality and the characterization of the optimal control (u_1, u_2) are use. □

When the control u_1^* and u_2^* are substituted into the controls system 21, it gives;

$$
\begin{cases}
\frac{d}{dt}S_m &= \pi + \omega R_m - \left(1 - min\left\{1, max\left\{0, \left(\lambda_2 - \lambda_1)\frac{\alpha_\lambda I_m S_m}{d_1} + (\lambda_2 - \lambda_1)\frac{\gamma_\lambda \tau_\lambda A_m S_m}{d_1}\right)\right\}\right\}\right)\alpha_\lambda I_m S_m \\
&\quad -(1-u_1)\gamma_\lambda\tau_\lambda A_m S_m - \mu S_m \\
\frac{d}{dt}E_m &= \left(1 - min\left\{1, max\left\{0, \left(\lambda_2 - \lambda_1)\frac{\alpha_\lambda I_m S_m}{d_1} + (\lambda_2 - \lambda_1)\frac{\gamma_\lambda \tau_\lambda A_m S_m}{d_1}\right)\right\}\right\}\right)\alpha_\lambda I_m S_m \\
&\quad +(1-u_1)\gamma_\lambda\tau_\lambda A_m S_m + \big((1-\psi_m)\theta + \psi_m\theta + \mu\big)E_m \\
\frac{d}{dt}A_m &= \psi_m\theta E_m - (\tau_1 + \mu)A_m \\
\frac{d}{dt}I_m &= (1-\psi_m)\theta E_m - (\eta_1 + \eta_2 + min\left\{1, max\left\{0, (\lambda_3 - \lambda_5)\frac{\alpha_\lambda I_m}{d_2}\right\}\right\} + \mu)I_m \\
\frac{d}{dt}R &= \eta_1 I_m + \tau_1 A_m + min\left\{1, max\left\{0, (\lambda_3 - \lambda_5)\frac{\alpha_\lambda I_m}{d_2}\right\}\right\}I_m - (\omega + \mu)R_m
\end{cases}
\tag{21}
$$

11. Numerical simulations

We examined the retrospective impact of the underlying control strategies on the model by considering the controls such as personal protection, u_1 and treatment, u_2 to assessed for their effectiveness. **Table 1** shows the parameter values used in the numerical simulations.

Table 1. Typhoid fever model Parameters.

Parameter	Description	Value	Reference
π	Recruitment rate	100	Assumed
α_λ	Transmission rate	0.8	Assumed
γ_λ	Modifying parameter accounting for infectiousness of Syphilis infected individuals in the exposed class	0.02	Assumed
τ_λ	Transmission rate	0.5	[41]
$\psi_m\theta$	Proportion of individuals who leave the exposed	0.006	Assumed
τ_1	rate at which individuals leave the Asymptomatic to the recovered class	0.06	Assumed
μ	Rate at which individuals naturally leaves the compartment	0.03	[42]
η_1	Rate at which individual leave the infected to recovered class	0.3	Assumed
η_2	Disease induced death	0.068	[43]
ω	Re-infection rate	0.6	[43]

11.1. Strategy A

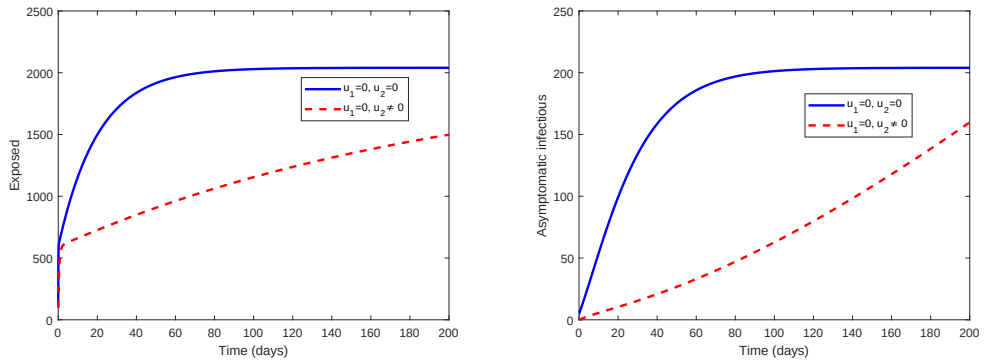
Strategy A sets $u_1 = 0$ and uses $u_2 \neq$ for the simulation. **Figures 2** of 2(a) and 2(b) are the simulated graphs of the exposed and asymptomatic infectious populations.

The control has a positive effects on the expose population and that of the asymptomatic populations as shown in the diagram.

Figures 3 of 3(a) and 3(b) are the simulated graphs of the symptomatic infectious populations and the control plot. The control has a positive effects on the symptomatic population as

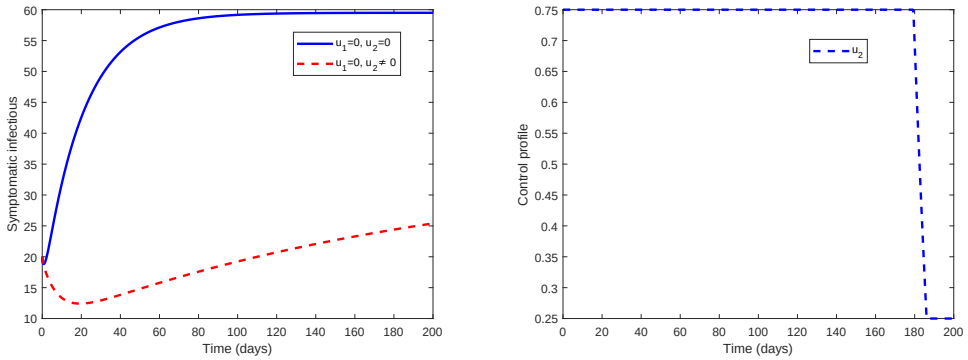
shown in the diagram.

Figure 3 of b(b) is the control profile plot of strategy A. We noticed that the treatment control remained at the upper bound till about 190 days when it dropped to the lower bound and stayed there for the remaining time. The control profile graph means that treatment of individuals could be relaxed after 190 days.



(**a**) (**b**)

Figure 2. (**a**) Expose population with $u_1 \neq 0$,$u_2 \neq 0$, $u_3 \neq 0$ and (**b**) Asymptotic population with $u_1 \neq 0$,$u_2 \neq 0$, $u_3 \neq 0$.



(**a**) (**b**)

Figure 3. (**a**) Symptomatic population with $u_1 \neq 0$,$u_2 \neq 0$, $u_3 \neq 0$ and (**b**) Control plots with $u_1 \neq 0$,$u_2 \neq 0$ and $u_3 \neq 0$.

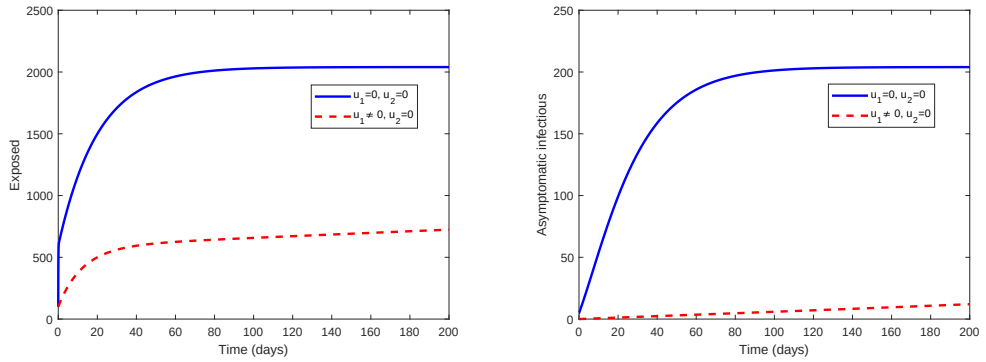
11.2. Strategy B

Strategy B sets $u_1 \neq 0$ and uses $u_2 = 0$ for the simulation. **Figures 4** of $4(a)$ and $4(b)$ are the simulated graphs of the exposed and asymptomatic infectious populations.

The control has a huge impact on the expose population and that of the asymptomatic populations as shown in the diagram. Strategy B is seen to be a better option as compared to strategy A but takes longer days to be implemented as well.

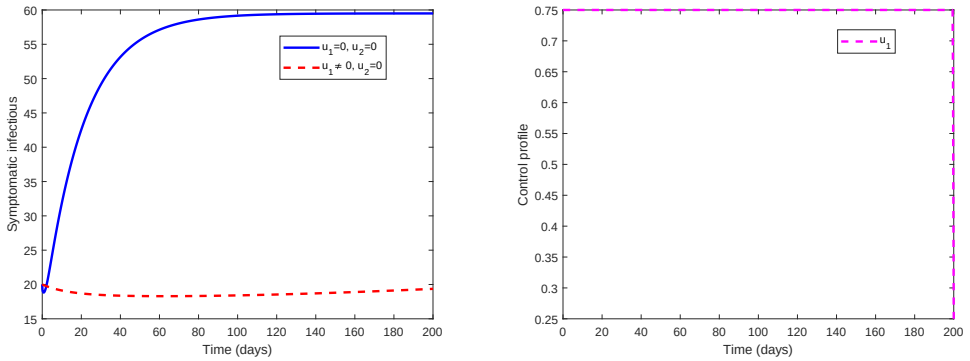
Figures 5 of $5(a)$ and $5(b)$ are the simulated graphs of the symptomatic infectious populations and the control plot. Control has a positive effects on the symptomatic population as shown in the diagram. **Figure 5** of $5(b)$ is the control profile plot of strategy A.

We noticed that the treatment control remained at the upper bound till about 200 days when it dropped to the lower bound and stayed there for the remaining time. The control profile graph means that treatment of individuals could be relaxed after 200 days.



(**a**) (**b**)

Figure 4. (**a**) Expose population with $u_1 \neq 0$,$u_2 \neq 0$, $u_3 \neq 0$ and (**b**) Asymptomatic population with $u_1 \neq 0$,$u_2 \neq 0$, $u_3 \neq 0$.



(**a**) (**b**)

Figure 5. (**a**) Symptomatic population with $u_1 \neq 0$,$u_2 \neq 0$, $u_3 \neq 0$ and (**b**) Control plots with $u_1 \neq 0$,$u_2 \neq 0$ and $u_3 \neq 0$.

11.3. Strategy C

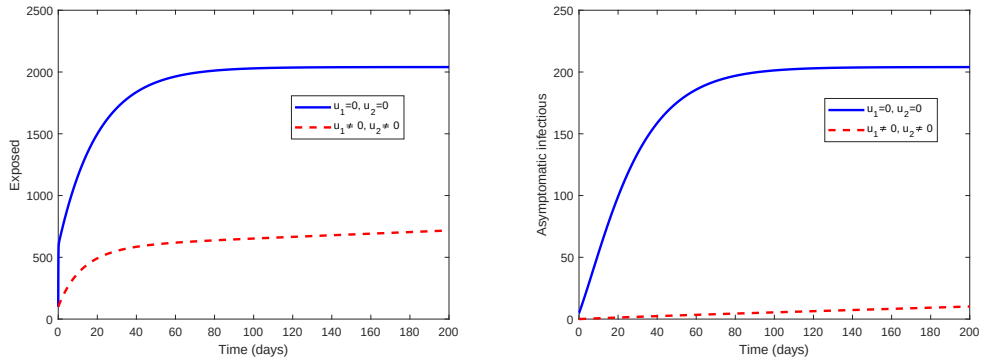
In strategy C, we set $u_1 \neq 0$ and uses $u_2 \neq 0$ for the simulation. **Figures 6** of $6(a)$ and $6(b)$ are the simulated graphs of the exposed and asymptomatic infectious populations.

The control has a huge impact on the expose population and that of the asymptomatic populations as shown in the diagram. Strategy C seem to have a better impact as compared to strategy A but takes longer days to be implemented as well.

Figures 7 of $7(a)$ and $7(b)$ are the simulated graphs of the symptomatic infectious populations and the control plot.

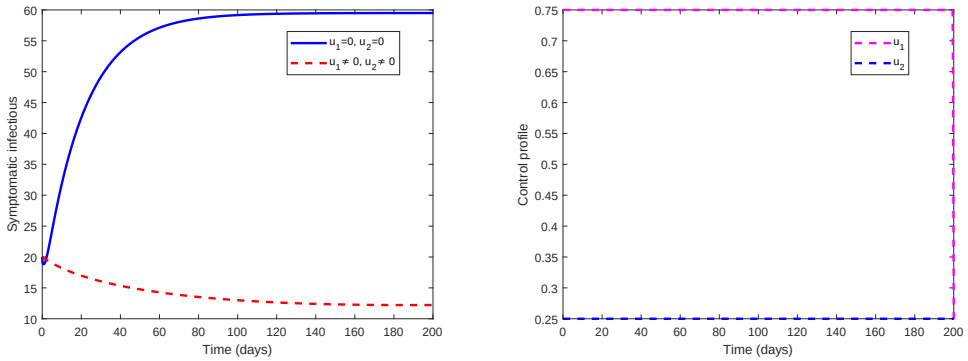
The control has a positive effects on the symptomatic population as shown in the diagram.

Figure 7 of $7(b)$ is the control profile plot of strategy A. The treatment control remained at the upper bound till about 200 days when it dropped to the lower bound and stayed there for the remaining time. This means that treatment of individuals could be relaxed after 200 days.



(**a**) (**b**)

Figure 6. (**a**) Expose population with $u_1 \neq 0$,$u_2 \neq 0$, $u_3 \neq 0$ and (**b**) Asymptomatic population with $u_1 \neq 0$,$u_2 \neq 0$ and $u_3 \neq 0$.



(**a**) (**b**)

Figure 7. (**a**) Symptomatic population with $u_1 \neq 0$,$u_2 \neq 0$, $u_3 \neq 0$ and (**b**) Control plots with $u_1 \neq 0$,$u_2 \neq 0$ and $u_3 \neq 0$.

11.4. Conclusions

In this paper, a nonlinear deterministic model of Syphilis disease was constructed to determine the dynamics of Syphilis infections. The study deduced the model's equilibria and analyzed the local and global stability of these equilibria.

The model was extended to optimal control problem by adding time-dependent controls that helped characterize a range of possible controls that minimized the disease.

The control system was solved qualitatively and numerically to evaluate the effectiveness of the considered controls using Pontryagin's Maximum Principle. The analysis indicated that strategies B and C are considered most effective as they substantially minimized the exposed, asymptomatic and symptomatic infectious.

We recommend that stakeholders should consider strategy B and C in their effort to mitigate the disease from the population as they all have the same effect of substantially minimizing the exposed, symptomatic and asymptomatic populations.

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