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Research Article

Mathematical Modeling and Analysis of Dynamics of Neisseria Gonorrhea Disease with Self Protection, Treatment and Natural Immunity

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ABSTRACT

In this article, we present a robust mathematical framework for modeling the infection dynamics of Neisseria Gonorrhoea. Unlike earlier models in the literature, our approach is more comprehensive, integrating a wide array of concepts related to the infection process. We conduct an in-depth analysis of both local and global stability, along with a sensitivity analysis of the model parameters. The results are supported by algorithmic experiments that illustrate the model's behavior under varying conditions. Additionally, we perform a thorough computational analysis, emphasizing how different parameter values influence the model's dynamics. This work not only enhances our understanding of gonorrhea transmission but also identifies potential avenues for further research in this critical area of public health.

1. INTRODUCTION

A type of bacteria known as Neisseria gonorrhoeae is the source of the sexually transmitted infection gonorrhea. It is transferred from one individual to another through oral, anal, and vaginal sex. It is present in the urethra, anus, vagina, and throat. Infections in newborns can result in eye infections. After ten days of exposure to the germ, the symptoms manifest and then go away [1]. Although women frequently show no symptoms at all, men and women who have lost their symptoms are nonetheless infected and contagious. If one has an oral sex infection, they may have symptoms such as sore throat. The risk of gonorrhea spreading to the throat during penis-mouth sex is higher than during mouth-vagina sex, which is rare [2].

Neisseria gonorrhoea, the bacteria that causes gonorrhea, is one of the most prevalent STIs in the world and poses serious health risks to the general public. Despite advances in diagnostic tools, treatment, and prevention strategies, the persistence and resurgence of gonorrhea highlight the need for innovative approaches to control its spread [3]. In males, gonorrhea can result in epididymitis, a painful testicular infection that, if ignored, can occasionally result in infertility. If left untreated, gonorrhea can cause scarring inside the urethra, which can make it difficult to urinate. It can also affect the prostate. An estimated 78 million new cases of gonorrhea are detected annually; in the United States alone, there are an estimated 820,000 new cases each year, and in 9963, the World Health Organization determined that Logos, Nigeria, had the highest

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rate of gonorrhea worldwide. In the past, gonorrhea was identified in Edinburg in 1792 by surgeon Benjamin Bell, who distinguished it from the contagious syphilis sickness. It can affect the throat, the mucous membranes in the eyes, the mouth, the anus, and the throat in addition to the reproductive track [4]. If the treated people had sexual contact with an infected person, they could become reinfected [5]. Human gonorrhea infections that are left untreated raise the risk of contracting or spreading HIV, which can result in AIDS [6]. The risk of blindness, joint infections, and potentially fatal blood infections in children who contract gonorrhea from an infected mother during birthing can be decreased by treating gonorrhea infections in pregnant women as soon as they are identified. The emergence of antibiotic-resistant strains of Neisseria gonorrhoeae further complicates control efforts, necessitating a deeper understanding of transmission dynamics and the effectiveness of various interventions [7]. Mathematical modeling has proven to be a valuable tool in epidemiology, offering insights into the dynamics of infectious diseases and informing public health interventions [8]. Models of gonorrhea transmission can help elucidate the potential impact of different control strategies, including the role of self-protection behaviors, such as condom use, regular STI testing, and partner notification [9]. Self-protection behaviors are critical in reducing the transmission of gonorrhea, yet their effectiveness is influenced by a range of factors, including individual adherence, public health policies, and the characteristics of the sexual network [10]. For instance, condom use has been shown to reduce the risk of transmission significantly, but inconsistent use and behavioral factors can diminish its overall impact [11]. According to [12], the ameningococcal B vaccine Bersero, which has been authorized in the US since 2015, causes humans to develop antibodies that fight Neisseria gonorrhoeae. Frequent STI testing and prompt treatment of infected persons are also essential, but they rely on healthcare availability and the willingness of the individual to seek testing [13]. An investigation conducted by Kermack & McKendrick [14]. examining the effects of self-defense practices in a mathematical model to investigate the dynamics of gonorrhea transmission [14]. By incorporating various self-protection strategies into the model, we aim to quantify their effects on the transmission of gonorrhea and identify the conditions under which these strategies can effectively reduce the prevalence of the disease. This research contributes to ongoing efforts to combat STIs by providing a theoretical framework that can inform public health policies and individual decision-making understanding the interplay between self-protection behaviors and gonorrhea transmission is essential for designing targeted interventions that can adapt to changing epidemiological patterns and societal behaviors. The insights gained from this study have the potential to inform future public health strategies aimed at curbing the spread of gonorrhea.

2. MODEL CONSTRUCTION

Neisseria gonorrhoea can spread between individuals. To account for this, the $SPEITR$ model was developed, dividing the population into six epidemiological compartments. $P(t)$ is the class of people who continue to take care of themselves and protect themselves from the sickness, while $S(t)$ represents those who have not yet contracted the disease but have the potential to do so. $E(t)$ denotes the class of exposed individuals, and $I(t)$ displays the infected class that was ever infected by the Neisseria gonorrhoea bacterium. t, In the model, the class of recovered individuals is represented by $R(t)$, whereas the treatment class is denoted by $T(t)$. The epidemic model is shown in Figure 1, which we suggested.

$$\begin{cases} \frac{d}{dt}S = \Lambda - (\mu + \beta I + \eta)S + \psi_1 R + \psi_2 E + \tau P \\ \frac{d}{dt}P = \eta S - (\mu + \tau)P, \\ \frac{d}{dt}E = \beta I S - (\mu + \rho + \psi_2)E, \\ \frac{d}{dt}I = \rho E - (\mu + \delta + \phi)I, \\ \frac{d}{dt}T = \phi I - (\mu + \nu)T, \\ \frac{d}{dt}R = \nu T - (\mu + \psi_1)R \end{cases} \tag{1}$$

With initial conditions: $S(0) > 0,\ P(0) \geq 0,\ E(0) \geq 0,\ I(0) \geq 0,\ I(0) \geq (0),\ R(0) \geq (0))$. The parameters are described in Table 1.

TABLE I. Description of Parameters and definition of State Variables

Parameters/Variables	Descriptions/Definitions
$S(t)$	At time t, the number of susceptible persons
$P(t)$	Class of individuals who keep self protection at time t
$E(t)$	Total number of people exposed to Neisseria gonorrhoea at time t
$I(t)$	Number of people with Neisseria gonorrhoea infection at time t
$T(t)$	Number of patients receiving treatment at time t
$R(t)$	Number of people that have recovered at that moment t
Λ	Recruitment rate to susceptible population
β	The contact rate of those who are infected
η	The rate of people transfer from S to P
γ	The number of Gonorrhea patients who recovered
ψ_1	Rate of recovered people moving back to susceptible
ρ	Transfer rate of Gonorrhea exposed individuals into infected individuals
ψ_2	Rate of exposed individuals moving back to susceptible
μ	Natural death rate
ρ	Transmission rate from E into I
δ	Disease death rate
ϕ	The transfer rate from the infected class into the treatment class
τ	Individuals' migration rate from susceptible class $S(t)$ to protection class P
ν	Rate of recovered individuals

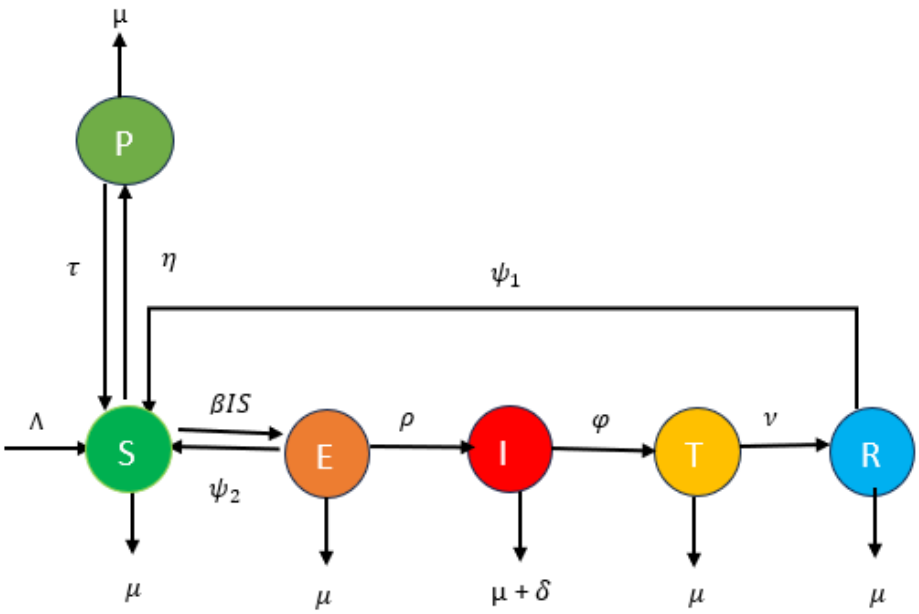

Fig. 1. The Model's flow chart

3. THE POSITIVE INVARIANT REGION

$$N(t) = S(t) + P(t) + E(t) + I(t) + T(t) + R(t)$$

. Differentiate with respect t we have;

$$\frac{dN}{dt} = \frac{dS}{dt} + \frac{dP}{dt} + \frac{dE}{dt} + \frac{dI}{dt} + \frac{dT}{R}.$$

By adding system (1) we get

$$\frac{dN}{dt} = \Lambda - \mu N - \delta I \tag{2}$$

By applying the following theorem, the positive invariant region

Theorem 1. *The system of equations is feasible if, for all* $t \geq (0)$*, the solutions to* (1) *lie inside the invariant region* Ω*.*

Proof. Suppose that $\Omega = (S, P, E, I, T, R) \in \Re_+^6$. With non-negative initial conditions, be any solution to the system of equations (1). Next, we obtain from equation (2).

$$\frac{dN}{dt} \leq \Lambda - \mu N(t)$$

by using the integrating factor to multiply via

$$e^{\mu t}(\frac{dN}{dt} + \mu N) \leq \Lambda e^{\mu t}$$

After applying the product rule in reverse and integrating the two sides, we have;

$$\frac{d}{dt}(Ne^{\mu t}) \leq \Lambda e^{\mu t}$$

$$\frac{d}{dt}(Ne^{\mu t}) \leq \frac{\Lambda}{\mu} e^{\mu t} + C \tag{3}$$

Applying the initial conditions $t = 0, N = N_0$ we obtain;

$$N_0 \leq \frac{\Lambda}{\mu} + C \Rightarrow (N_0 - \frac{\Lambda}{\mu} \leq C \tag{4}$$

equation (3) implies that

$$\frac{dN}{dt}(Ne^{\mu t}) - \frac{\Lambda}{\mu} e^{\mu t} \leq C \tag{5}$$

When we compare (4) with (5), we have;

$$N(t) \leq \frac{\Lambda}{\mu} + (N_0 - \frac{\Lambda}{\mu})e^{-\mu t} \tag{6}$$

The maximum number of people that the human population can support $N(t)$ approaches $\epsilon = \frac{\Lambda}{\mu} (i.e., \epsilon \to \frac{\Lambda}{\mu})$ at $t \to 0$ in equation (6).
Consequently, the region contains every feasible solution for the Model's human population.

$$\Omega = \{(S, P, E, I, T, R) \in \Re_+^6 : S > 0, P > 0, E > 0, I > 0, T > 0, R > 0, N \leq \frac{\Lambda}{\mu}\} \tag{7}$$

Consequently, in the domain Ω, system (1) is both mathematically and epidemiologically significant, and the region Ω is positively-invariant.

4. POSITIVITY OF THE SOLUTIONS

Theorem 2. *Let the initial solutions be* $\{(S(0), P(0), E(0), I(0), T(0), R(0)) \geq 0\} \in \Omega$ *then the solutions* $\{S(t), P(t), E(t), I(t), T(t), R(t)\}$ *of the system* (1) *is positive* $\forall t \geq 0$.

Proof. From the first equation of (1), we have:

$$\frac{d}{dt}S = \Lambda - (\mu + \beta I + \eta)S + \psi_1 R + \psi_2 E + \tau P \geq -(\mu + \eta)S$$

$$\frac{d}{dt}S(t) \geq -(\mu + \eta)S$$

Separate variables and integrate we have:

$$\int \frac{dS}{S} \geq -\int (\mu + \eta)dt$$

By solving both the left and right hand sides we have :

$$\ln S + C_1 \geq -\mu t - \eta t + C_2$$

whereC_1 and C_2 are the constants of integration.
Subtract C_1 from both sides and combining the constants C_1 and C_2 into a single constant C gives i.e ($C_2 - C_1 = C$):

$$\ln S \geq -\mu t - \eta t + C$$

$$\ln S \geq -(\mu + \eta)t + C$$

Take exponential to both sides:

$$e^{\ln S} \geq e^{-(\mu+\eta)t+C}$$

$$S \geq e^{-(\mu+\eta)t}.e^{C}$$

Since e^C is just another constant, we can denote it as B:

$$S(t) \geq Be^{-(\mu+\eta)t}$$

Applying initial conditions $t = 0$ we have

$$S(0) \geq Be^{-(\mu+\eta)(0)}$$

$$S(0) \geq Be^{0}$$

This implies that

$$S(0) \geq B$$

Thus we conclude that

$$S(t) \geq S(0)e^{-(\mu+\eta)t} > 0$$

From the second equation of (1), we have:

$$\frac{d}{dt}P = \eta S - (\mu + \tau)P \geq -(\mu + \tau)P$$

$$\frac{d}{dt}P \geq -(\mu + \tau)P$$

separate variables and integrate

$$\frac{dP}{P} \geq -(\mu + \tau)dt$$

$$\int \frac{dP}{P} \geq -\int (\mu + \tau)dt$$

$$\ln P + C_1 \geq -(\mu + \tau)t + C_2$$

$$\ln P \geq -(\mu + \tau)t + C_2 - C_1$$

where $C_2 - C_1 = C$,

$$\ln P \geq -(\mu + \tau)t + C$$

Exponentiating both sides we have:

$$e^{\ln P} \geq e^{-(\mu+\tau)t+C}$$

$$P \geq e^{-(\mu+\tau)t}.e^{C}$$

e^C is constant we can use$D = e^C$,

$$P \geq De^{-(\mu+\tau)t}$$

Applying initial conditions $t = 0$ we have

$$P(0) \geq De^{0} \Rightarrow p(0) \geq D$$

$$P(t) \geq P(0)e^{-(\mu+\tau)t} \geq 0$$

From the third equation of (1), we have:

$$\frac{d}{dt}E = \beta IS - (\mu + \rho + \psi_2)E \geq -(\mu + \rho + \psi_2)E$$

$$\frac{d}{dt}E \geq -(\mu + \rho + \psi_2)E$$

separate variables and integrate we have:

$$\frac{dE}{E} \geq -(\mu + \rho + \psi_2)dt \Rightarrow \int \frac{dE}{E} \geq -\int (\mu + \rho + \psi_2)dt$$

$$\int \frac{dE}{E} \geq -\int (\mu + \rho + \psi_2)dt \Rightarrow \ln E \geq -(\mu + \rho + \psi_2)t + C$$

$$e^{\ln E} \geq e^{-(\mu+\rho+\psi_2)t+C}$$

$$E \geq e^{-(\mu+\rho+\psi_2)t}.e^{C},$$

$$E \geq e^{-(\mu+\rho+\psi_2)t}.e^{C},$$

By substituting initial conditions $t = 0$ and equate e^C by M we have:

$$E \geq M,$$

Therefore $E(t) \geq P(0)e^{(\mu+\rho+\psi_2)} \geq 0$, By using a similar procedure we can prove the remaining equations of system (1) are also positive,that as: $I(t) \geq (0),\ T(t) \geq (0),\ R(t) \geq (0)$.

5. DISEASE FREE EQUILIBRIUM STATE

To find the model (1)'s disease-free equilibrium, set

$$\frac{dS}{dt} = \frac{dP}{dt} = \frac{dE}{dt} = \frac{dI}{dt} = \frac{dT}{dt} = \frac{dR}{dt} = 0$$

As $E = I = T = 0$, there is no sickness in this instance. Thus, the DFE of the model we have suggested is provided by: Equation first and second implies that;

$$\begin{cases} \Lambda - (\mu + \eta)S + \tau P = 0 \\ \eta S - (\mu + \tau)P = 0 \end{cases} \tag{8}$$

To solve the system of equations by equating terms, multiply the first equation of (8) by$(\mu + \tau)$ and second by τ we get:

$$\Lambda(\mu + \tau) - (\mu + \tau)(\mu + \eta)S + (\mu + \tau)\tau P = 0 \tag{9}$$

$$\tau\eta S - \tau(\mu + \tau)P = 0 \tag{10}$$

Adding equations, (9) and (10) and calculate S^0 we have;

$$S^0 = \Lambda\frac{\mu + \tau}{(\mu + \eta)(\mu + \tau) - \tau\eta} \Rightarrow \frac{\Lambda(\mu + \tau)}{\mu(\mu + \tau + \eta)} \tag{11}$$

Also for P second equation of (8) implies that:

$$\eta S - (\mu + \tau)P = 0 \Rightarrow P^0 = \frac{\eta S^0}{(\mu + \tau)} \tag{12}$$

Putting values of S^0 from equation (11) in equation (12) and simply further for P^0 we have:

$$\Rightarrow P^0 = \frac{\Lambda\eta}{\mu(\mu + \tau + \eta)} \tag{13}$$

Hence the disease free equilibrium E^0 points are:

$$E^0 = \left(\frac{\Lambda(\mu + \tau)}{\mu(\mu + \tau + \eta)}, \frac{\Lambda\eta}{\mu(\mu + \tau + \eta)}, 0, 0, 0, 0\right). \tag{14}$$

6. BASIC REPRODUCTION NUMBER (R_0)

The average number of secondary infections that infected people cause during their infectiousness is called R_0. When $R_0 > 1$, the disease will spread throughout the community, meaning that an infectious person will not perpetuate the disease and will not produce more than one secondary infection. When dealing with a more complex epidemic, the next-generation operator approach [4]
can be used to calculate the R_0. We can compute F and V using the system (1).

$$F = \begin{pmatrix} \beta IS \\ 0 \end{pmatrix}, V = \begin{pmatrix} (\mu + \rho + \psi_2)E \\ -\rho E + (\mu + \delta + \phi)I \end{pmatrix}, \tag{15}$$

taking jacobian of matrices F and V we obtain the followings:

$$F^* = \begin{pmatrix} 0 & \beta S \\ 0 & 0 \end{pmatrix}, V^* = \begin{pmatrix} \mu + \rho + \psi_2 & 0 \\ -\rho & \mu + \delta + \phi \end{pmatrix}, \tag{16}$$

inverse of V^* is calculated as:

$$V^{*-1} = \begin{pmatrix} \frac{1}{\mu+\rho+\psi_2} & 0 \\ \frac{\rho}{(\mu+\rho+\psi_2)(\mu+\delta+\phi} & \frac{1}{\mu+\delta+\phi} \end{pmatrix}, \tag{17}$$

From this point on, we will utilize the Next Generation matrix $G = F^*V^{*-1}$.

$$G = \begin{pmatrix} \frac{\rho\beta S^0}{(\mu+\rho+\psi_2)(\mu+\delta+\phi)} & \frac{\beta S^0}{\mu+\rho+\phi} \\ 0 & 0 \end{pmatrix}, \tag{18}$$

Using $|G - \lambda I| = 0$ as the characteristic equation of G, we can find the eigenvalues:

$$|G - \lambda I| = \begin{pmatrix} \frac{\rho\beta S^0}{(\mu+\rho+\psi_2)(\mu+\delta+\phi)} - \lambda & \frac{\beta S^0}{\mu+\rho+\phi} \\ 0 & 0 - \lambda \end{pmatrix} = 0, \tag{19}$$

it is clear that $\lambda_2 = 0$ and $\lambda_1 = \frac{\rho\beta S^0}{(\mu+\rho+\psi_2)(\mu+\delta+\phi}$ is maximum eigenvalue, by putting the value of S^0 from (11) we set $\lambda_1 = R_0$ is the basic reproduction number as:

$$R_0 = \frac{\Lambda\rho\beta(\mu + \tau)}{\mu(\mu + \rho + \psi_2)(\mu + \delta + \phi)(\mu + \tau + \eta)}. \tag{20}$$

Theorem 3. *The disease-free equilibrium is locally asymptotically stable if $R_0 < 1$ and unstable otherwise that is $R_0 > 1$.*

Proof. To do the local stability analysis of the disease-free equilibrium, we will employ the Jacobean stability technique. The system of equations at disease-free equilibrium has the following Jacobean matrix:

$$J = \begin{pmatrix} -(\mu + \beta I + \eta) & \tau & \psi_2 & -\beta S & 0 & \psi_1 \\ \eta & -(\mu + \tau) & 0 & 0 & 0 & 0 \\ \beta I & 0 & -(\mu + \rho + \psi_2) & \beta S & 0 & 0 \\ 0 & 0 & \rho & -(\mu + \delta + \phi) & 0 & 0 \\ 0 & 0 & 0 & \phi & -(\mu + \nu) & 0 \\ 0 & 0 & 0 & 0 & \nu & -(\mu + \psi_1) \end{pmatrix} \tag{21}$$

At DFE E_0 the above metrics becomes:

$$J = \begin{pmatrix} -(\mu+\eta) & \tau & \psi_2 & -\beta S^0 & 0 & \psi_1 \\ \eta & -(\mu+\tau) & 0 & 0 & 0 & 0 \\ 0 & 0 & -(\mu+\rho+\psi_2) & \beta S^0 & 0 & 0 \\ 0 & 0 & \rho & -(\mu+\delta+\phi) & 0 & 0 \\ 0 & 0 & 0 & \phi & -(\mu+\nu) & 0 \\ 0 & 0 & 0 & 0 & \nu & -(\mu+\psi_1) \end{pmatrix} \tag{22}$$

Utilise row operation $.-(\mu+\eta)R_2 + \eta R_1$ we have

$$J = \begin{pmatrix} -(\mu+\eta) & \tau & \psi_2 & -\beta S^0 & 0 & \psi_1 \\ 0 & \tau\eta - (\mu+\tau)(\mu+\eta & \psi_2\eta & -\beta\eta S^0 & 0 & \psi_1\eta \\ 0 & 0 & -(\mu+\rho+\psi_2) & \beta S^0 & 0 & 0 \\ 0 & 0 & \rho & -(\mu+\delta+\phi) & 0 & 0 \\ 0 & 0 & 0 & \phi & -(\mu+\nu) & 0 \\ 0 & 0 & 0 & 0 & \nu & -(\mu+\psi_1) \end{pmatrix} \tag{23}$$

From first column, we get $\lambda_1 = 0$ the remaining matrix will be:

$$J = \begin{pmatrix} \tau\eta - (\mu+\tau)(\mu+\eta & \psi_2\eta & -\beta\eta S^0 & 0 & \psi_1\eta \\ 0 & -(\mu+\rho+\psi_2) & \beta S^0 & 0 & 0 \\ 0 & \rho & -(\mu+\delta+\phi) & 0 & 0 \\ 0 & 0 & \phi & -(\mu+\nu) & 0 \\ 0 & 0 & 0 & \nu & -(\mu+\psi_1) \end{pmatrix} \tag{24}$$

Expand above by first column to obtain $\lambda_2 = \tau\eta - (\mu+\tau)(\mu+\eta$ then:

$$J = \begin{pmatrix} -(\mu+\rho+\psi_2) & \beta S^0 & 0 & 0 \\ \rho & -(\mu+\delta+\phi) & 0 & 0 \\ 0 & \phi & -(\mu+\nu) & 0 \\ 0 & 0 & \nu & -(\mu+\psi_1) \end{pmatrix} \tag{25}$$

By column fourth we have $\lambda_3 = -(\mu+\psi_1)$:

$$J = \begin{pmatrix} -(\mu+\rho+\psi_2) & \beta S^0 & 0 \\ \rho & -(\mu+\delta+\phi) & 0 \\ 0 & \phi & -(\mu+\nu) \end{pmatrix} \tag{26}$$

again by third column we get $\lambda_4 = -(\mu+\psi_1)$,

$$J_1 = \begin{pmatrix} -(\mu+\rho+\psi_2) & \beta S^0 \\ \rho & -(\mu+\delta+\phi) \end{pmatrix} \tag{27}$$

The determinant must be larger than zero and the trace of matrix J_1 must be smaller than zero in order for the DFE to be regarded locally stable. This is the maximum number of individuals that can exist.

$$TraceJ_1 = -(\mu+\rho+\psi_2) - (\mu+\delta+\phi) < 0$$

$$TraceJ_1 = -((\mu+\rho+\psi_2) + (\mu+\delta+\phi)) < 0$$

And determinant.

$$Det(J_1) = (\mu+\rho+\psi_2)(\mu+\delta+\phi) - \rho\beta S^0 > 0$$

$$(\mu+\rho+\psi_2)(\mu+\delta+\phi) > \rho\beta S^0$$

$$\rho\beta S^0 < (\mu+\rho+\psi_2)(\mu+\delta+\phi)$$

Divide both sides by $(\mu+\rho+\psi_2)(\mu+\delta+\phi)$, and substitute value of S^0 (11) we conclude:

$$Det(J_1) = \frac{\rho\beta\Lambda(\mu+\tau)}{\mu(\mu+\rho+\psi_2)(\mu+\delta+\phi)(\mu+\eta)} = R_0 < 1.$$

Hence by the Routh-Hurwitz criteria of stability the disease-free equilibrium point E_0 is locally asymptotically stable if $R_0 < 1$ otherwise unstable. Thus, DFE is Locally Asymptotically Stable (LAS) if and only if $R_0 < 1$. The theorem's epidemiological implication states that, if the initial size of the sub-populations falls under the DFE's basin of attraction, gonorrhea can be eradicated (control) from the population when $R_0 < 1$.

7. GLOBAL STABILITY OF DISEASE FREE EQUILIBRIUM POINTS

The method of Castillo-Chavez et al. [15] is used to examine the global stability of DFE. Afterward, the model system (1) can be expressed as follows:

$$\begin{cases} \frac{dB}{dt} = F(B, Q) \\ \frac{dQ}{dt} = G(B, 0), G(B, 0) = 0 \end{cases} \tag{28}$$

The number of uninfected compartments is denoted by $B \in R^m$, the number of infected compartments is denoted by $Q \in R^n$, and the disease-free equilibrium point is represented by $E^0 = (B^0, 0)$. The following conditions (H_1) and (H_2) must be met in order to ensure the global asymptotic stability of DFE.
(H_1) For $\frac{dB}{dt} = F(B, 0)$, B^0 is globally asymptotically stable (GAS),
(H_2) $G(B, Q) = XQ - \hat{G}(B, Q)$, $\hat{G}(B, Q) \geq 0$ For $(B, Q) \in \Omega$.
Since X's off-diagonal elements are non-negative, $X = A_1(B, 0)$ is the Metzler Matrix, and Ω denotes the region where the Gonorrhea model system (1) provides epidemiological significant information. Accordingly, the following theorem holds if and only if the system satisfies H_1 and (H_2).

Theorem 4. *For a system of equations with $R_0 < 1$, the disease-free equilibrium is globally asymptotically stable; for a system with $R_0 > 1$, it is unstable.*

Proof. One way to express the Gonorrhea model system (1) is as follows: $B = (S, P, T, R) \in R^4$ and $E^0 = (\frac{\Lambda(\mu+\tau)}{\mu(\mu+\tau+\eta)}, \frac{\Lambda\eta}{\mu(\mu+\tau+\eta)}, 0, 0, 0, 0)$ Now, we have:

$$\frac{dB}{dt} = \begin{pmatrix} \Lambda - (\mu + \beta I + \eta)S + \psi_1 R + \psi_2 E + \tau P \\ \eta S - (\mu + \tau)P \\ \phi I - (\mu + \nu)T \\ \nu T - (\mu + \psi_1)R \end{pmatrix}, \tag{29}$$

We arrive at the disease-free equilibrium point at;

$$\frac{dB}{dt} = F(B^0, 0) = \begin{pmatrix} \Lambda - (\mu + \eta)S^0 + \tau P^0 \\ \eta S^0 - (\mu + \tau)P^0 \\ 0 \\ 0 \end{pmatrix}, \tag{30}$$

implies that:

$$\frac{dB}{dt} = F(B^0, 0) = \begin{pmatrix} \Lambda - (\mu + \eta)\frac{\Lambda(\mu+\eta)}{\mu(\mu+\eta+\tau)} + \tau\frac{\Lambda\eta}{\mu(\mu+\eta+\tau)} \\ \eta\frac{\Lambda(\mu+\eta)}{\mu(\mu+\eta+\tau)} - (\mu + \tau)\frac{\Lambda\eta}{\mu(\mu+\tau+\eta)} \\ 0 \\ 0 \end{pmatrix}, \tag{31}$$

$F(B^0, 0)$ has a distinct point of equilibrium. $B^0 = \left(\frac{\Lambda(\mu+\tau)}{\mu(\mu+\tau+\eta)}, \frac{\Lambda\eta}{\mu(\mu+\tau+\eta)}, \right)$., which has asymptotically global stability. Thus, H_1 holds.
For the second condition, (H_2).

$$\hat{G}(B, Q) = \begin{pmatrix} -E(\mu + \rho + \psi_2) + BS^0 I \\ E\delta - I(\mu + \delta + \phi) \end{pmatrix} \tag{32}$$

Then we get,

$$X = A_1(B^0, 0) = \begin{pmatrix} -(\mu + \rho + \psi_2 & BS^0 \\ \rho & -(\mu + \delta + \phi) \end{pmatrix} \tag{33}$$

The matrix X is clearly an M-marix now that its off diagonal components are non-negative.Thus, $\hat{G}(B,Q) = XA - G(B,Q)$ implies

$$\hat{G}(B,Q) = \begin{pmatrix} -(\mu+\rho+\psi_2) & BS^0 \\ \rho & -(\mu+\delta+\phi) \end{pmatrix} \begin{pmatrix} E \\ I \end{pmatrix} - \begin{pmatrix} \beta IS - (\mu+\rho+\psi_2)E \\ \rho E - (\mu+\delta+\phi)I \end{pmatrix} \tag{34}$$

$$\hat{G}(B,Q) = \begin{pmatrix} -(\mu+\rho+\psi_2)E + \beta S^0 I \\ \delta E - (\mu+\delta+\phi)I \end{pmatrix} + \begin{pmatrix} -\beta IS + (\mu+\rho+\psi_2)E \\ -\rho E + (\mu+\delta+\phi)I \end{pmatrix} \tag{35}$$

$$= \begin{pmatrix} \beta S^0 - \beta S \\ 0 \end{pmatrix} = \begin{pmatrix} \beta(S^0 - S) \\ 0 \end{pmatrix} \tag{36}$$

Since $S^0 > S$, $B^0 = \left(\frac{\Lambda(\mu+\tau)}{\mu(\mu+\tau+\eta)}, \frac{\Lambda\eta}{\mu(\mu+\tau+\eta)}\right) \geq 1$, and $\hat{G}(B,Q) \geq 0$ are globally asymptotically stable.

8. ENDEMIC EQUILIBRIUM POINTS

The endemic equilibrium points of our proposed model can be derive by putting all equations of system (1) equal to zero, we have

$$\begin{cases} S^* = \frac{\Lambda+\psi_1 R^*+\psi_2 E^*+\tau P^*}{\mu+\beta I^*+\eta} \\ P^* = \frac{\eta S^*}{\mu+\tau} \\ E^* = \frac{\beta I^* S^*}{\mu+\rho+\psi_2} \\ I^* = \frac{PE^*}{\mu+\delta+\phi} \\ T^* = \frac{\phi I^*}{\mu+\nu} \\ R^* = \frac{\nu T^*}{\mu+\psi_1} \end{cases} \tag{37}$$

9. GLOBAL STABILITY OF ENDEMIC EQUILIBRIUM POINTS

The investigation of the endemic equilibrium point E^* was made possible by computing its global stability using the Lyapunov function that Vargas-De-León [16] devised. If $\frac{dV}{dt} < 0$, At the given point, the Lyapunov function $V(x)$ is considered to be globally asymptotically stable .

Theorem 5. *For Model System* (1)*, there exists a unique endemic equilibrium point E^* for the gonorrhea disease, which, in the case where $R_0 > 1$, is globally asymptotically stable; otherwise, it is unstable.*

Proof. Consider the Lyapunov function that is quadratic.

$$V(y_1, ..., y_n) = \sum_{i=1}^{n} \frac{1}{2}[y_i - y_i^*]^2$$

For the model system (1), a positive definite function looks like this: in this example, the population of the *i*th compartment is denoted by y_i, and the endemic equilibrium point is y_i.

$$F(S,P,E,I,R) = \sum_{i=1}^{6} \frac{1}{2}[y_i - y_i^*]^2$$

The gonorrhea model system's Lyapunov function is thus expressed as follows:

$$V(x) = \frac{1}{2}[(S - S^*)(P - P^*)(E - E^*)(I - I^*)(T - T^*)(R - R^*)]^2 \tag{38}$$

It is evident that $V : \mathbb{R}_+^6 \to \mathbb{R}$ is a differentiable and continuous function. Next, the function $V(x)$ can be differentiated with respect to time to obtain:

$$\frac{V(x)}{dt} = [(S - S^*)(P - P^*)(E - E^*)(I - I^*)(T - T^*)(R - R^*)]\frac{d}{dt}[S + P + E + I + T + R]$$

$$\frac{V(x)}{dt} = [S + P + E + I + T + R] - (S^* + P^* + E^* + I^* + T^* + R^*)\frac{d}{dt}[S + P + E + I + T + R]. \tag{39}$$

But,

$$(S + P + E + I + T + R) = \Lambda - \mu N - \delta I. \tag{40}$$

And

$$\Lambda - \mu N - \delta I^* = 0.$$

$$\Lambda - \mu(S^* + P^* + E^* + I^* + T^* + R^*) - \delta I^* = 0.$$

$$(S^* + P^* + E^* + I^* + T^* + R^*) = \frac{\Lambda - \delta I^*}{\mu} \tag{41}$$

Substitute (39) and (40) in (38) we have:

$$\frac{dV}{dt} = [(S + P + E + I + T + R) - \frac{(\Lambda - \delta I^*)}{\mu}][\Lambda - \mu N - \delta I]$$

$$\frac{dV}{dt} = [N - \frac{(\Lambda - \delta I^*)}{\mu}][\Lambda - \mu N - \delta I]$$

$$\frac{dV}{dt} = \left[N - \frac{(\Lambda - \delta I^*)}{\mu}\right]\left[-\mu(N + \frac{\Lambda}{\mu} - \frac{\delta I}{\mu})\right]$$

$$\frac{dV}{dt} = \left[N(t) - \frac{\Lambda}{\mu} + \frac{\delta I^*}{\mu}\right]\left[-\mu(N(t) - \frac{\Lambda}{\mu} + \frac{\delta I}{\mu})\right]$$

$$\frac{dV}{dt} = -\mu\left[N(t) - \frac{\Lambda}{\mu} + \frac{\delta I^*}{\mu}\right]\left[(N(t) - \frac{\Lambda}{\mu} + \frac{\delta I}{\mu})\right]$$

$$\frac{dV}{dt} \leq -\mu\left[N(t) - \frac{\Lambda}{\mu}\right]\left[(N(t) - \frac{\Lambda}{\mu})\right]$$

That is

$$\frac{dV}{dt} \leq -\mu\left[N(t) - \frac{\Lambda}{\mu}\right]^2 < 0$$

As a result, it is evident that $\frac{dV}{dt} < 0$, indicating the asymptotic global stability of the Endemic Equilibrium Point E^*.

10. SENSITIVITY ANALYSIS

The basic reproductive number R_0 sensitivity analysis to the model parameters is very important for the current study. It enables us to determine the most crucial factors that affect the spread and management of disease. This section examines the sensitivity of many important parameters of our suggested Gonnerrhea model (1).
This method's goal is to investigate the model contained parameters' significance in relation to R_0. Here, we examine each parameter's sensitivity for the system (1). To do this, we must assess
$\chi_\zeta^{R_0} = \frac{\partial R_0}{\partial \zeta} \times \frac{\zeta}{R_0}$, where, $\zeta \in (\beta, \Lambda, \mu, \delta, \psi_2, \rho, \tau, \phi, \eta)$

1. For β, we have: $\chi_\beta^{R_0} = \frac{\partial R_0}{\partial \beta} \times \frac{\beta}{R_0} \Rightarrow \chi_\beta^{R_0} = 1$,
2. For Λ, we have: $\chi_\Lambda^{R_0} = \frac{\partial R_0}{\partial \Lambda} \times \frac{\Lambda}{R_0} \Rightarrow \chi_\Lambda^{R_0} = 1$,
3. For ρ, we have: $\chi_\rho^{R_0} = \frac{\partial R_0}{\partial \rho} \times \frac{\rho}{R_0} \Rightarrow \chi_\rho^{R_0} = \frac{(\mu+\delta+\phi)(\mu+\tau+\eta)(\eta^2+\mu\psi_2-\rho\mu)}{\mu(\mu+\rho+\psi_2)}$,
4. For δ, we have: $\chi_\delta^{R_0} = \frac{\partial R_0}{\partial \delta} \times \frac{\delta}{R_0} \Rightarrow \chi_\delta^{R_0} = -\frac{\delta(\mu+\tau+\eta)}{\mu(\mu+\delta+\phi)}$,
5. for Ψ_2, we have: $\chi_{\Psi_2}^{R_0} = \frac{\partial R_0}{\partial \Psi_2} \times \frac{\Psi_2}{R_0} \Rightarrow \chi_{\Psi_2}^{R_0} = -\frac{\psi_2(\mu+\tau+\eta)}{\mu(\mu+\delta+\phi)(\mu+\rho+\Psi_2)}$,
6. For τ, we have: $\chi_\tau^{R_0} = \frac{\partial R_0}{\partial \tau} \times \frac{\tau}{R_0} \Rightarrow \chi_\tau^{R_0} = \frac{(\Lambda\rho\beta\eta)}{[\mu(\mu+\rho+\psi_2)(\mu+\delta+\phi)(\mu+\tau+\eta)]^2}$,
7. For η, we have: $\chi_\eta^{R_0} = \frac{\partial R_0}{\partial \eta} \times \frac{\eta}{R_0} \Rightarrow \chi_\eta^{R_0} = -\frac{\eta(\mu+\delta+\phi)}{\mu(\mu+\tau+\eta)}$,
8. For ϕ, we have: $\chi_\phi^{R_0} = \frac{\partial R_0}{\partial \phi} \times \frac{\phi}{R_0} \Rightarrow \chi_\phi^{R_0} = -\frac{\phi(\mu+\tau+\eta)}{\mu(\mu+\delta+\phi)}$.
9. For μ, we have: $\chi_\mu^{R_0} = -\frac{\mu\left((\delta+\mu+\phi)(\eta+\mu+\tau)+(\eta+\mu+\tau)(\mu+\rho+\psi_2)+(\delta+\mu+\phi)(\mu+\rho+\psi_2)-\frac{(\eta+\mu+\tau)(\delta+\mu+\phi)(\mu+\rho+\psi_2)}{\mu+\tau}\right)+(\eta+\mu+\tau)(\delta+\mu+\phi)(\mu+\rho+\psi_2)}{(\eta+\mu+\tau)(\delta+\mu+\phi)(\mu+\rho+\psi_2)}$.

This analysis is summarized in the following Table 2.

TABLE II. Parametric sensitivity analysis of the model (2)

Parameter	Description	Sensitivity Indices
Λ	Recruitment rate	+1
β	Transmission rate to Gonorrhea	+1
ρ	Transfer rate of Gonorrhea exposed individuals into infected individuals	0.6
μ	Natural death rate	-0.3
τ	People's moving rate from the protection class P to susceptible class $S(t)$	-0.046
ψ_2	Rate of exposed individuals moving back to susceptible	-0.03
δ	Disease death rate	-0.5
ϕ	The transfer rate from infected class into treatment class	-0.4
η	The rate of people transfer from S to P	-0.78

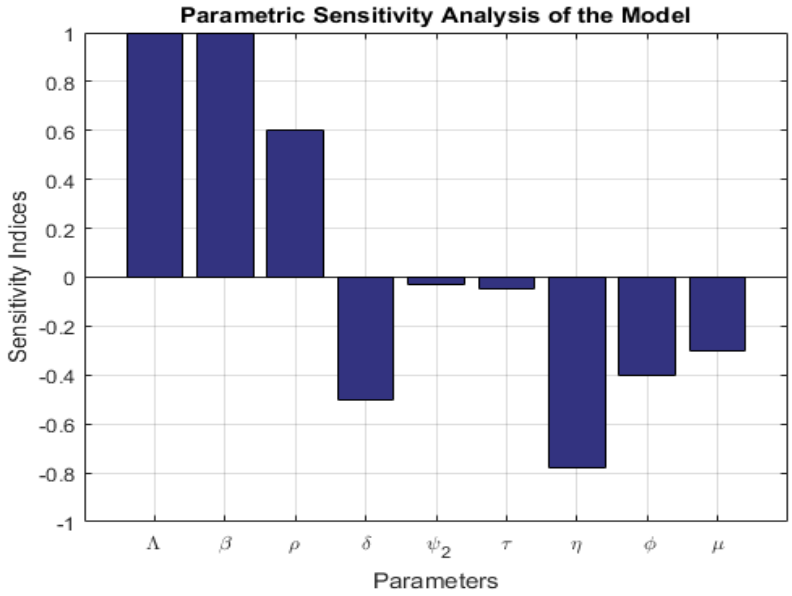

Fig. 2. A graph is provided regarding the parameters and the sensitivity indices of the Gonorrhea model Basic Reproduction number (R_0).

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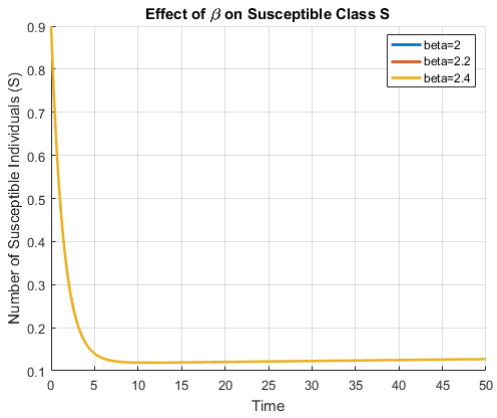
The numerical values of the sensitivity indices for the model are listed in Table 2. The gonorrhea transmission model's sensitivity analysis reveals that the most important parameters are the rate of new infections Λ and the transmission rate per contact β. This suggests that focusing interventions on these areas will have a major effect on disease control. On the other hand, a high negative relationship is seen for parameters like η, which may be associated with partner change rates or antibiotic efficaciousness. This suggests that lowering these rates can significantly reduce transmission. By comparison, ψ_2 and τ have negligible effects, suggesting that the most important parameters should get the majority of resources in order to effectively restrict the spread of gonorrhea. The sensitivity analysis of the model was displayed in Figure (2).

11. SIMULATIONS

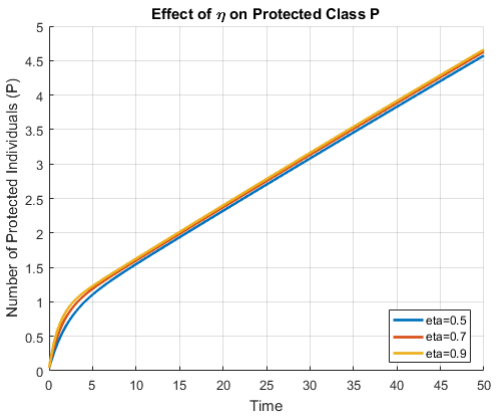
Numerical simulations were performed in this section with the settings listed in Table 3. Importantly, the parameter values displayed in Table 2 were derived from previously published works. On the other hand, the remaining figures were calculated using information from the World Health Organization and other scholars. Utilized in numerical simulations, the predictor-corrector technique is MATLAB-based. There are four differential equation order values considered in this study.

TABLE III. Parameters Values and References

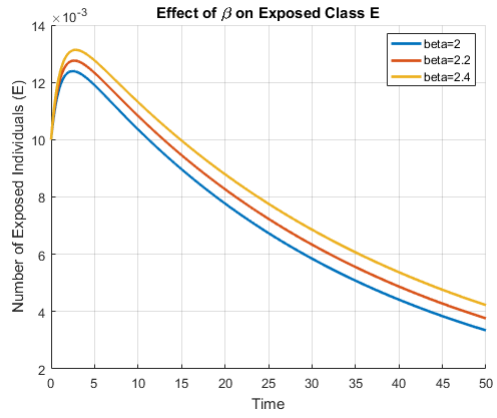
Parameter	Value	Reference
Λ	0.008	[17]
β	2.0, 2.2, 2.4	Assumed
μ	0.000125	[18]
ν	0.5, 0.7, 0.9	[19]
τ	0.02	Assumed
ψ_1	0.005	[20]
ψ_2	0.006	[21]
ρ	0.004	Assumed
ϕ	0.4, 0.5, 0.7	Assumed
η	0.5, 0.7, 0.9	[22]
δ	0.044	[23]



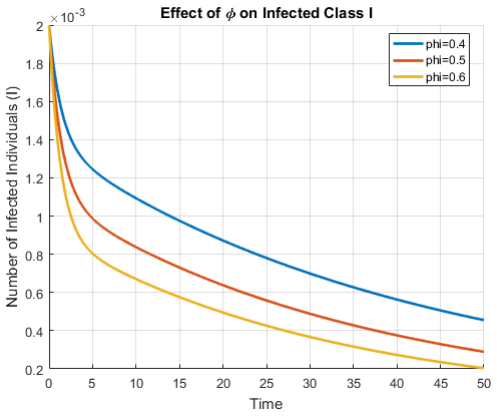
(a) Dynamics of Gonorrhea Susceptible individuals



(b) Dynamics of Gonorrhea Protected individuals

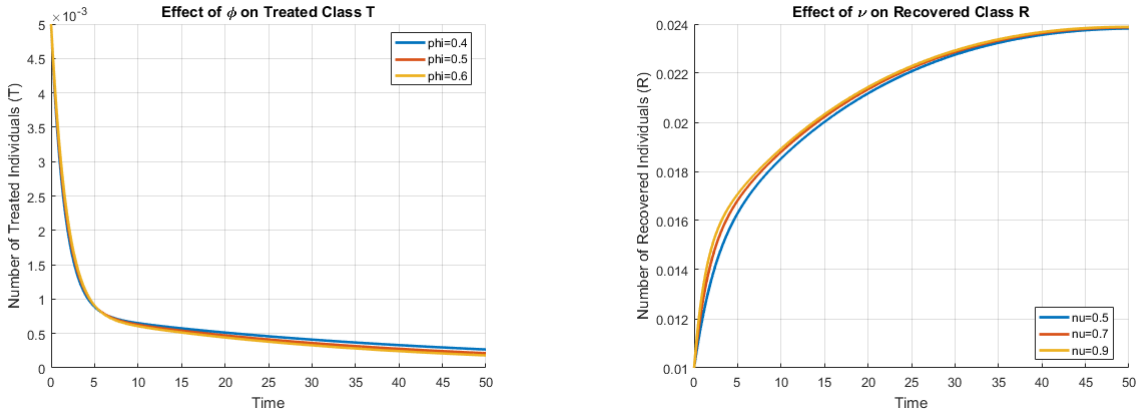


(c) Dynamics of Gonorrhea Exposed individuals



(d) Dynamics of Gonorrhea Infected individuals

Fig. 3. Population dynamics for different groups: (a) Susceptible, (b) Protected, (c) Exposed, (d) Infected individuals.



(a) Dynamics of Treated individuals

(b) Dynamics of Recovered individuals

Fig. 4. Continuation of population dynamics: (e) Treated individuals, (f) Recovered individuals.

In Fig.3 and Fig.4, we can observe that the susceptible S is greatly impacted by increasing the transmission rate β. The rate at which susceptible people are exposed to the infection increases as β rises, which causes S to drop more quickly. This highlights the critical role of controlling transmission rates to prevent rapid depletion of the susceptible pool, which could otherwise accelerate the spread of the infection and overwhelm the healthcare system. Adjusting the protection rate η shows that increasing η enhances the number of protected individuals P, as a higher η reduces the rate at which susceptible individuals are lost to other states and increases the conversion rate from susceptible to protected. Effective protection strategies are therefore crucial in reducing the overall vulnerability of the population and mitigating the impact of an outbreak. The number of exposed individuals E rises with higher β values, as more susceptible individuals are exposed to the infection due to increased transmission. This increase in E eventually leads to a higher number of infected cases if exposed individuals transition to the infected state at a similar rate. This emphasizes the importance of reducing transmission rates to control the initial exposure rate and subsequent infection spread. Higher values of ϕ lead to an increase in the number of infected individuals I by accelerating the progression from exposed to infected states. This accelerated transition can lead to a surge in infected cases, highlighting the need for timely interventions to prevent a rapid increase in the disease burden. Effective management of this transition is essential to avoid overwhelming healthcare systems and reducing the overall impact of the disease. As ϕ increases, the number of treated individuals T also rises because a higher ϕ speeds up the transition from infected to treated states. This highlights the significance of effective treatment approaches in the management and control of the illness. Treating sick people as soon as possible is essential to limiting the illness's contagious period and containing its spread. A greater ν decreases the amount of time spent in the infected and treated states just before recovery, which has a significant effect on the number of recovered persons R. This leads to a quicker reduction in the number of infectious cases and contributes to a higher recovery rate. Effective treatment and recovery strategies are thus vital in minimizing the duration and impact of the disease, promoting overall population health and resilience.

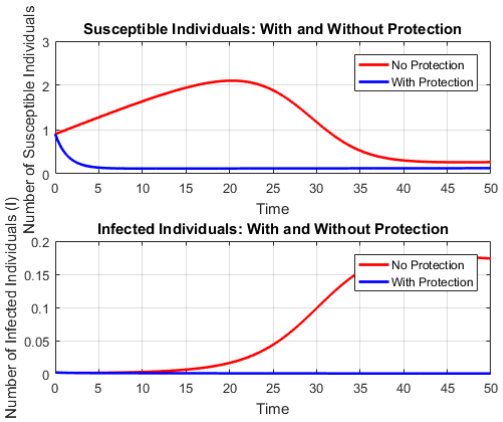


Fig. 5. Dynamics of Gonorrhea transmission with and without Protection

In Fig. 5, we describe comparison between with and without protection reveals distinct differences in the dynamics of the infection. Without protection, the number of susceptible individuals (S) decreases more rapidly, while the number of infected individuals (I) rises quickly due to higher transmission rates. In contrast, with protection, the decline in susceptible individuals is slower, and the infection spreads more gradually, resulting in a lower peak and overall number of infected individuals. This demonstrates that protection significantly mitigates the spread of the disease, reducing both the speed and extent of infection compared to the scenario without protection.

12. CONCLUSION

Examining the dynamics of gonorrhea infection is the aim of this effort. We carry out independent analyses of the Gonorrhea reproduction. We discover that the model's disease-free equilibrium point is locally asymptotically stable for values of $R_0 < 1$. But when the system shows signs of external reinfection, the number of reproductions R_0 is less than unity, which is insufficient to eradicate the disease. We suggest that gonorrhea may be eradicated if R_0 is smaller, based on our findings. Furthermore, we show mathematically that Gonorrhea dynamics has an equilibrium point when R_0 is larger than unity. Sensitivity analyses are examined after the model's stability study. Numerical simulations are performed in the end.

Conflicts of Interest

The authors declare no conflicts of interest.

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