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Bifurcation and Stability Analysis of the Dynamics of Gonorrhea Disease in the Population

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ABSTRACT

The model was governed by a system of ordinary differential equations; the population into susceptible individuals (S), Exposed individuals (E), infected individuals (I) and Recovered individuals (Q). Theory of positivity and boundedness was used to investigate the well-posedness of the model. Equilibrium solutions were investigated analytically. The basic reproduction number (R_0) were calculated using the next generation operator method. Bifurcation analysis and global stability of the model were carried out using centre manifold theory and Lyapunov functions respectively. The effects of parameters such as efficacy of Condom (κ), Effective Contact Rate (β), Compliance of Condom (θ), Progression Rate (ρ) and Treatment Rate (α) on R_0 were explored through sensitivity analysis. The possibility of mitigating the spread of gonorrhea in the population has been studied. It can be concluded that parameters representing efficacy of Condom, Effective Contact Rate, Compliance of Condom, Progression Rate and Treatment Rate are significant in reducing the burden of gonorrhea disease in the population.

***Keywords*:** Stability Analysis, Basic Reproduction Number, Lyapunov function, Gonorrhea disease, Sensitivity analysis, Bifurcation analysis

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1. INTRODUCTION

The first gonorrhea model was by Cooke and Yorke [1], this commence the use of differential equation models to study the transmission dynamics and control of sexually transmitted diseases (STDs). The use of differential equations for models of STDs can be traced back to R. Ross [2], which was done in the year 1911; he introduced the first differential equation model for the transmission dynamics of vector transmitted diseases. R. Ross model work was motivated by his attempts to develop management strategies for the control of malaria, a disease that is transmitted as part of the life cycle of the Plasmodium parasite. Ross's contributions to the understanding of the malaria life cycle were rewarded with a Nobel Prize in medicine.

The author of [2] made several of remarks that became major components in the modeling of vector- and sexually transmitted diseases, including the fact that the average total rate of contacts between host and vectors must be conserved. This simple conservation law has become the basis for modeling heterogeneous contact structures [3, 4].

The remarks were good and worth to be credited in this setting, as he categorically recognized that STDs could be used / modeled in the same way as vector-transmitted diseases [2]. In addition, he was aware of the role of frequency-dependent dynamics and, consequently, he did not restrict his work to situations where the interacting subpopulations did not change [2]; also the author of [5] did the same thing. Modeling of Sexually transmitted diseases were limited in component because of the assumption that the sizes of interacting populations were constant and not dynamic variables [6, 7].

Garnett et al [8], studied the sexual behavior of gonorrhea patients and it was used to estimate the parameters of their gonorrhea model disease transmission. Their model was used to assess the potential impacts of treatment intervention. Kretzschmar et al [9], proposed a stochastic model for gonorrhea which analyze the underlying structure of sexual contact pattern. They compared the benefits of condom use in an age-structured population of sexually active core group. Prabhakararao [10], analyzed a mathematical model of Gonorrhea disease. They ascertained that the spread of the gonorrhea disease involves interaction of the susceptible and the infective. Leung and Gopalsamy [11], examined a continuous time SIV model for Gonorrhea transmission among homosexuals. [11] Used a non-standard discretization method to study a discrete time model, and compared their results with that of other models. Yorke [12], modelled the spread of Gonorrhea in a population that was classified into “n” group and used it to further study the asymptotic stability of the model. Kishore and Pattabhiramacharyulu [13], formulated a simple non-linear first order Ordinary differential equation model for gonorrhea that measure the growth rates of promiscuous and infective in a homosexual population. Kishore and Pattabhiramacharyulu further used numerical examples to explain the effect of cure rate and infective rate on the spread and control of the disease.

Besides the mathematical models, an equally outstanding contribution has been achieved by the non-mathematical models. Karnath [14], discusses the symptoms and signs of Neisseria Gonorrhea with regards to the genitourinary and extra-genital, and outlines laboratory diagnosis with recommended treatment measures. Benedek [15], discusses the unsuccessfulness of various experiments in an attempt to infect animals with Gonorrhea infection as well as history of researches on causes and spread of Gonorrhea in humans over the decades. Bala [16], compared and compiled the resistance trends of Neisseria Gonorrhea across various countries of south-East Asia Region by means of surveillance.

2. MODEL FORMATION

The model is a heterosexually active population. The disease that guides the model is gonorrhea and, consequently, infective recover after treatment was considered. It was assumed that the population is genetically and behaviorally homogeneous except for the gender of individuals in the population. The model used is a Susceptible-Exposed-Infective-Recovered model, that is, a homogeneously mixing SEIR model. The assumption here is that once susceptible class increases constantly by constant recruitment π and individuals treated that are re-infected at a rate ζ, it diminish at the rate of contact of infection β and natural death μ. The exposed grows as the infection β invade the susceptible and reduces as the disease progresses ρ to real infection with full symptom and natural death μ. Infected class increases as they progress ρ from expose and decrease at the rate of treatment α, natural μ and diseases induced death d, while the recovered class increases as the move from infected with the rate of treatment α and decrease as the rate of re-infection ζ and natural death μ.

2. 1. Model Equation

The following non-linear system of differential equations,

$$\left.\begin{aligned}
\frac{dS}{dt} &= \pi - (1-\kappa\theta)\beta S(t) I(t) - \mu S(t) + \zeta R(t) \\
\frac{dE}{dt} &= (1-\kappa\theta)\beta S(t) I(t) - (\rho+\mu) E(t) \\
\frac{dI}{dt} &= \rho E(t) - (\mu + d + \alpha) I(t) \\
\frac{dR}{dt} &= \alpha I(t) - (\mu+\zeta) R(t)
\end{aligned}\right\} \quad (1)$$

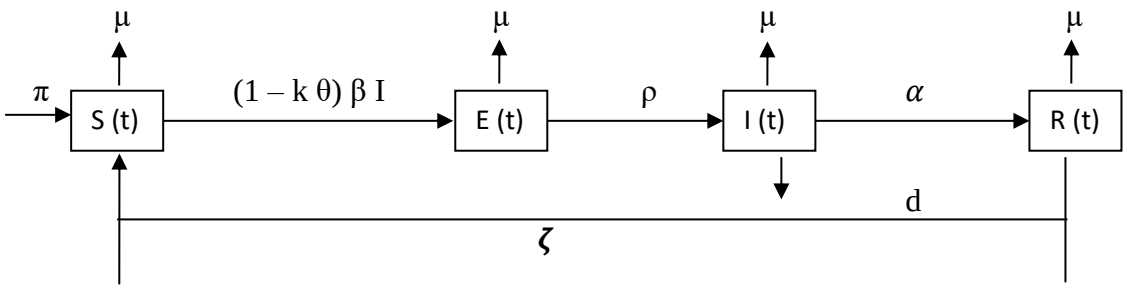
Table 1. Descriptions of Parameters and Values.

Parameters	Definitions	Value
π	Recruitment Rate	20
ƙ	Efficacy of Condom	0.2
θ	Compliance of Condom	0.5
β	Effective Contact Rate	0.6
μ	Natural Death Rate	0.1
ζ	Loss of Immunity	0.03
ρ	Progression Rate	0.6
d	Induced Death Rate	0.2
α	Treatment Rate	0.6

Table 2. Description of Variables.

Variables	Definitions
S	Susceptible Individual
E	Exposed Individual
I	Infected Individual
R	Recovered Individual

2. 2. Flow Chart



3. MODEL ANALYSIS

3. 1. Positively Invariant Region

3. 1. 1. Theorem 1

The closed set $D=\left\{\left(S+E+I+R\right)\in R_{+}^{4}:N\leq\frac{\pi}{\mu}\right\}$ is positively-invariant and attracting with respect to the model in (1)

3. 1. 2. Proof

Consider the biologically-feasible region *D*, defined above. The rate of change of the total population, obtained by adding all equations of the model in (1), is given by

$$\frac{dN}{dt}=\pi-\mu N-\delta \qquad (2)$$

It follows that $\frac{dN}{dt}<0$ whenever $N>\frac{\pi}{\mu}$, furthermore,

Since $\frac{dN}{dt}\leq\pi-\mu N$

it is clear that $\mathrm{N}(t) \leq \frac{\pi}{\mu}$

if $\mathrm{N}(0) \leq \frac{\pi}{\mu}$

Therefore, all solutions of the model with initial conditions in *D* remain in *D* for all $t > 0$ (i.e., the ω-limits sets of the system in (1) are contained in *D*). Thus, *D* is positively-invariant and attracting. In this region, the model can be considered as been epidemiologically and mathematically well posed

3. 2. Existence and Uniqueness of the solution

3. 2. 1. Theorem 2

The closed set

$D = \left\{S + E + I + R : \left|S - S(0)\right| \leq a,\ \left|E - E(0)\right| \leq b, \left|I - I(0)\right| \leq c, \left|R - R(0)\right| \leq d\right\}$ Then model in (1) has a unique solution in D

3. 2. 2. Proof

Consider the biologically-feasible region D, defined above. The model in (1) must be continuous and bounded in D.

Therefore, $\left|\frac{dx_i}{dx_j}\right|, i, j = 1,2,3,4,5,6$ are continuous and bounded. All solution of the model (1) with initial conditions in D.Hence the model (1) has a unique solution in D, which means that the model (1) is epidemiologically and mathematically well posed.

3. 3. Existence of Disease Free Equilibrium (DFE)

Disease free mean that there is no incidence of disease in the population; I = 0,

$$\frac{dS}{dt} = \frac{dE}{dt} = \frac{dI}{dt} = \frac{dR}{dt} = 0 \tag{3}$$

Then, system (1) is solved simultaneously, and $\mathcal{E}_0$ denotes the disease free equilibrium. Thus; the model in (1) has disease free equilibrium given by

$$\varepsilon_0 = (S,\ E, I, R) = \left(\frac{\pi}{\mu}, 0, 0, 0\right) \tag{4}$$

3. 4. Existence of Endemic Equilibrium Point (EEP)

When there is disease in the population, it is called Endemic state; it implies that

$$S \neq E \neq I \neq R \neq 0 \tag{5}$$

and now solve model (1) simultaneously to get the endemic equilibrium point, it given below;

$$\left.\begin{aligned} S^{**} &= \frac{K_1 K_2}{\beta\,\rho\left(\kappa\,\theta+1\right)} & I^{**} &= \frac{K_3\,A}{\beta\,B} \\ E^{**} &= \frac{K_2\,K_3\,A}{\beta\,\rho\,B} & R^{**} &= \frac{\alpha\,A}{\beta\,B} \end{aligned}\right\} \quad (6)$$

where:

$$K_1 = \mu+\rho \qquad K_2 = \alpha+\mu+d \qquad K_3 = \zeta+\mu$$

$$A = \rho\,\pi\,\beta\left(\kappa\,\theta+1\right) - \mu\,K_1K_2 \qquad B = \left(\rho\,\zeta\,\alpha - K_1K_2K_3\right)\left(\kappa\,\theta-1\right)$$

3. 5. Basic Reproduction Number (R_0)

Using next generation matrix [14], the non-negative matrix F (new infection terms) and non-singular matrix V (other transferring terms) of the model are given respectively by;

$$F = \begin{pmatrix} \left(1-\kappa\,\theta\right)\beta\,S\,I \\ 0 \\ 0 \end{pmatrix}, \qquad \textit{and} \qquad V = \begin{pmatrix} \left(\mu+\rho\right)E \\ -\rho\,E + \left(\mu+d+\alpha\right)I \\ -\alpha\,E + \left(\mu+\zeta\right)R \end{pmatrix}$$

After taking partial derivatives of F and V, we have:

$$\text{F=} \begin{pmatrix} 0 & \dfrac{\left(1-\kappa\,\theta\right)\beta\,\pi}{\mu} & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix} \text{ at DFE} \quad (7)$$

$$\text{V=} \begin{pmatrix} \mu+\rho & 0 & 0 \\ -\rho & \mu+d+\alpha & 0 \\ -\alpha & 0 & \mu+\zeta \end{pmatrix} \quad (8)$$

Thus;

$$R_0 = \frac{\left(1-\kappa\,\theta\right)\beta\,\pi\,\rho}{\mu\left(\mu+\rho\right)\left(\mu+d+\alpha\right)} \quad (9)$$

The threshold quantity R_0 is the basic reproduction number of the system (1) for Gonorrhea infection. It is the average number of new secondary infections generated by a single infected individual in his or her infectious period [10].

3. 6. Local Stability of the DFE

3. 6. 1. Theorem 3

The disease free equilibrium of the model (1) is locally asymptotically stable (LAS) if $R_0 < 1$ and unstable if $R_0 > 1$.

3. 6. 2. Proof

To determine the local stability of E_0, the following Jacobian matrix is computed corresponding to equilibrium point E_0. Considering the local stability of the disease free equilibrium at $\left(\frac{\pi}{\mu},0,0,0\right)$. Thus,

$$J_G = \begin{pmatrix} -(\mu+\lambda) & 0 & \frac{-\beta(1-\kappa\theta)\pi}{\mu} & \zeta \\ 0 & -(\rho+\mu)-\lambda & \frac{\beta(1-\kappa\theta)\pi}{\mu} & 0 \\ 0 & \sigma & -(\mu+d+\alpha)-\lambda & 0 \\ 0 & 0 & \alpha & -(\mu+\zeta)-\lambda \end{pmatrix} \quad (10)$$

The characteristics polynomial of the above matrix is given by

$$B_4\lambda^4 + B_3\lambda^3 + B_2\lambda^2 + B_1\lambda + B_0 = 0 \quad (11)$$

then;

$$B_0 = \frac{-(1-\kappa\theta)\beta\pi\rho}{\mu} + (\mu+\rho)(\mu+d+\alpha) \quad (12)$$

Thus by Routh – Hurwitz criteria, E_o is locally asymptoticly stable as it can be seen for

$$B_1 > 0, B_2 > 0, B_3 > 0, B_4 > 0, B_1B_3 - B_3 > 0 \text{ and } B_1B_2B_3 - B_3^2 - B_1^2B_4 > 0 \quad (13)$$

Thus, using $B_0 > 0$

$$B_0 = \frac{(1-\kappa\theta)\beta\pi\rho}{\mu(\mu+\rho)(\mu+d+\alpha)} < 1 \quad (14)$$

Hence $R_0 < 1$

The result from Routh Hurwitz criterion shows that, all eigen-values of the polynomial are negative which shows that the disease free equilibrium is locally asymptotically stable.

3. 7. Global Stability of the Disease free equilibrium

3. 7. 1. Theorem 4

The DFE, ε_0, of the model (1), is globally asymptotically stable in D if $R_0 \leq 1$.

3. 7. 2. Proof

Consider the Lyapunov function

$$V = A_1 E + A_2 I \tag{15}$$

where,

$$A_1 = 1 \quad \text{and} \quad A_2 = \frac{(\rho+\mu)}{\rho} \tag{16}$$

The associated Lyapunov derivative is given by (where a dot represents differentiation with respect to time t)

$$\overset{*}{V} = A_1 \overset{*}{E} + A_2 \overset{*}{I} \tag{17}$$

$$= \{(1-\kappa\theta)\beta S(t) I(t) - (\rho+\mu) E(t)\} + \frac{(\rho+\mu)}{\rho}\{\rho E(t) - (\mu+d+\alpha) I(t)\}$$

$$= (1-\kappa\theta)\beta S(t) I(t) - (\rho+\mu) E(t) + (\rho+\mu)\rho E(t) - \frac{(\rho+\mu)}{\rho}(\mu+d+\alpha) I(t)$$

$$\leq \frac{(1-\kappa\theta)\beta\pi}{\mu} I(t) - \frac{(\rho+\mu)(\mu+d+\alpha)}{\rho} I(t)$$

$$= \frac{\rho}{(\rho+\mu)(\mu+d+\alpha)}[R_0 - 1] I(t) \tag{18}$$

Thus, $\overset{*}{V} \leq 0$ if $R_o \leq 1$ with $\overset{*}{V} = 0$ if and only if $E = I = R = 0$. Further, the largest compact invariant set in $\left\{(S, E, I, R) \in D : \overset{*}{V} = 0\right\}$ is the singleton $\{\varepsilon_o\}$. It follow, from the LaSalle's Principle [14], that every solution to the equation in (1) with initial conditions in D converge to ε_o as $t \to \infty$. That is, $(E(t), I(t), R(t)) \to (0, 0, 0)$ as $t \to \infty$.

Substituting $E = I = R = 0$ into the first equation of (1) gives $S(t) \to \frac{\pi}{\mu}$ as $t \to \infty$. Thus, $[S(t), E(t), I(t), R(t)] \to \left(\frac{\pi}{\mu}, 0, 0, 0\right)$ as $t \to \infty$. for $R_0 \leq 1$, so that the DFE,, is globally asymptotically stable in D if $R_0 \leq 1$.

3. 8. Global Stability of the Endemic Equilibrium Point

3. 8. 1. Theorem 5

Consider the model (1) with λ defined by (1). The associated unique endemic equilibrium of the model is globally asymptotically stable in $D^* \backslash D_0$ if $R_0^m > 1$ and $S \leq S^{**}$.

3. 8. 2. Proof

Consider the model (1) with λ and $R_0^m > 1$, so that the associated unique endemic equilibrium of the model exists. Further, consider the following non-linear Lyapunov function (of Goh-Volterra type):

$$F = S - S^{**} - S^{**} \ln\left(\frac{S}{S^{**}}\right) + E - E^{**} - E^{**} \ln\left(\frac{E}{E^{**}}\right) + \left(\frac{\beta S^{**}}{\mu + d + \alpha}\right)\left(I - I^{**} - I^{**} \ln\left(\frac{I}{I^{**}}\right)\right) \quad (19)$$

with Lyapunov derivative,

$$\dot{F} = \dot{S} - \frac{S^{**}}{S}\dot{S} + \dot{E} - \frac{E^{**}}{E}\dot{E} + \left(\frac{\beta S^{**}}{\mu + d + \alpha}\right)\left(\dot{I} - \frac{I^{**}}{I}\dot{I}\right) \quad (20)$$

So, that,

$$\dot{F} = \pi - (1 - \kappa\theta)\beta S(t) I(t) - \mu S(t) + \zeta R(t) - \frac{S^{**}}{S}(\pi - (1 - \kappa\theta)\beta S(t) I(t) - \mu S(t) + \zeta R(t)) + (1 - \kappa\theta)\beta S(t) I(t) - (\rho + \mu) E(t) - \frac{E^{**}}{E}((1 - \kappa\theta)\beta S(t) I(t) - (\rho + \mu) E(t)) + \left(\frac{\beta S^{**}}{\mu + d + \alpha}\right)\left(\rho E(t) - (\mu + d + \alpha) I(t) - \frac{I^{**}}{I}(\rho E(t) - (\mu + d + \alpha) I(t))\right) \quad (21)$$

At steady state

$$\pi = \beta S^{**} I^{**} + \mu S^{**} - \zeta R^{**}$$
$$\rho + \mu = \frac{\beta S^{**} I^{**}}{E^{**}} \tag{22}$$
$$\rho = \frac{(\mu + d + \alpha) I^{**}}{E^{**}}$$

Simplifying the above equation, it result into

$$\dot{F} = \beta S^{**} I^{**} + \mu S^{**} - \zeta R^{**} - \mu S + \zeta R - \beta I^{**} \frac{S^{**2}}{S} - \mu \frac{S^{**2}}{S} + \zeta R^{**} \frac{S^{**}}{S} + \beta I S^{**}$$
$$+ \mu S^{**} - \zeta R \frac{S^{**}}{S} - (\rho + \mu) E - \beta I S \frac{E^{**}}{E} - (\rho + \mu) E^{**}$$
$$+ \left(\frac{\beta S^{**}}{\mu + d + \alpha} \right) \left(\rho E(t) - (\mu + d + \alpha) I(t) - \frac{I^{**}}{I} (\rho E(t) - (\mu + d + \alpha) I(t)) \right) \tag{23}$$

$$\dot{F} = \beta S^{**} I^{**} + \mu S^{**} - \zeta R^{**} - \mu S + \zeta R - \beta I^{**} \frac{S^{**2}}{S} - \mu \frac{S^{**2}}{S} + \zeta R^{**} \frac{S^{**}}{S} + \beta I S^{**}$$
$$+ \mu S^{**} - \zeta R \frac{S^{**}}{S} - (\rho + \mu) E - \beta I S \frac{E^{**}}{E} - (\rho + \mu) E^{**} \tag{24}$$

$$\dot{F} = \beta S^{**} I^{**} \left(3 - \frac{S^{**}}{S} - \frac{E}{E^{**} I} - \frac{I S E^{**}}{I^{**} S^{**} E} \right) + \mu S^{**} \left(2 - \frac{S^{**}}{S} - \frac{S}{S^{**}} \right)$$
$$+ \zeta R^{**} \left(1 + \frac{R}{R^{**}} \right) \tag{25}$$

Finally, since the arithmetic means exceeds the geometric mean, the following inequalities hold:

$$3 - \frac{S^{**}}{S} - \frac{E}{E^{**} I} - \frac{I S E^{**}}{I^{**} S^{**} E} \leq 0$$
$$2 - \frac{S^{**}}{S} - \frac{S}{S^{**}} \leq 0 \tag{26}$$
$$1 + \frac{R}{R^{**}} \leq 0$$

Thus, $\dot{F} \leq 0$ for $R_0^m > 1$. Hence, F is a Lyapunov function on D^*. It follows, by LaSelle's Invariance Principle [14], that every solution to the equations of the model (2) with the force of infection and the initial condition in $D^* \backslash D_0$, approaches the associated unique endemic equilibrium of the model as $t \to \infty$ for $R_0^m > 1$.

4. SENSITIVITY ANALYSIS

This section examines changing effects of the model parameters with respect to basic reproduction number, R_o, of the model (1). To determine how changes in parameters affect the transmission and spread of the disease with recovered, a sensitivity analysis of model (1) is carried out in the sense of [10].

Definition 1. The normalized forward-sensitivity index of a variable, v, depends differentiable on a parameter, p, is defined as:

$$\gamma_p^v = \frac{\partial v}{\partial p} \times \frac{p}{v} \tag{27}$$

In particular, sensitivity indices of the basic reproduction number, R_o, with respect to the model parameter. The following results were obtained:

Table 3. Sensitivity indices with the Parameters

Parameter	Sign
β	Positive
κ	Negative
θ	Negative
α	Negative
π	Positive
μ	Negative
ρ	Positive
d	Negative

The positive sign of S.I of R_o to the model parameters shows that an increase (or decrease) in the value of each of the parameter in this case will lead to an increases (or decrease) in R_0 of the model (1) and asymptotically results into persistence (or elimination) of the disease in the

community . On the contrary, the negative sign of R_o to the model parameters indicates that an increase (or decrease) in the value of each of the parameter in this case leads to a corresponding decrease (or increases) on R_o of the model (1). Hence, with sensitivity analysis, one can get insight on the appropriate intervention strategies to prevent and control the spread of the disease described by model (1).

5. BIFURCATION ANALYSIS

It is observed from the previous result that whenever $R_0 > 1$, the asymptotic local stability of the disease-free equilibrium is lost. Here, bifurcation analysis is used to explore how the asymptotic stability of disease-free equilibrium is exchanged for asymptotic stability of endemic equilibrium of model (1) as the threshold quantity, R_0, cross the unity. In other words, investigate the bifurcation at $R_o = 1$, using a center manifold theory of bifurcation analysis described by Castillo-Chavez and Song 2004, used in some disease models (Buonomo and Vergas De-Leon (2013), Arino et. Al (2003), Olaniyi and Obabiyi (2016) and Gumel et.al (2008)). For convenience, the theorem in Castillo-Chavez and Song 2004 is reproduced here as follows:

Choosing β as the bifurcation parameter, then at $R_o = 1$

$$R_0 = \frac{(1-k\theta)\beta^* \pi \rho}{\mu(\mu+\rho)(\mu+d+\alpha)} = 1$$

Then,

$$\beta = \beta^* = \frac{\mu(\mu+\rho)(\mu+d+\alpha)}{(1-k\theta)\beta^* \pi \rho} \tag{28}$$

So that the disease-free equilibrium, D_o, is locally stable when $\beta < \beta^*$, and is unstable when $\beta > \beta^*$, this, β^*, is bifurcation value.

The linearized matrix of the system (1) around the disease-free equilibrium E_o and evaluated at β^* is given by;

Then,

$$J(E_o, \beta^*) = \begin{pmatrix} -\mu & 0 & \frac{-\beta(1-k\theta)\pi}{\mu} & \zeta \\ 0 & -(\rho+\mu) & \frac{\beta(1-k\theta)\pi}{\mu} & 0 \\ 0 & \rho & -(\mu+d+\alpha) & 0 \\ 0 & 0 & \alpha & (-\mu+\zeta) \end{pmatrix} \tag{29}$$

The eigenvalues (λ), of $J(E_o, \beta^*)$ given by (29) are the roots of the characteristic equation of the form:

$$(\lambda + \mu)(\lambda + \mu - \zeta)P(\lambda) = 0 \qquad (30)$$

where $P(\lambda)$ is a polynomial of degree four whose roots are real and negative except one zero eigenvalue.

5. 1. Determination of right eigen-vector and left eigen-vector

The right eigenvector, $w = (w_1, w_2, w_3, w_4)^T$, associated with this simple zero eigenvalue can be obtained from $J(D_o, \beta^*)w = 0$. Furthermore, the left eigenvector, $v = (v_1, v_2, v_3, v_4)$, corresponding to the simple zero eigenvalue of (29) is obtained from $v\, J(D_o, \beta^*) = 0$

5. 2. Computation of "a" and "b"

The direction of the bifurcation at $R_o = 1$ is determined by the signs of bifurcation coefficient "a" and "b", obtained from the above partial derivatives, given, respecting, by

5. 2. 1. Computation of "a" and "b"

$$a = \frac{(\zeta\alpha\rho\mu - \mu(\mu+\rho)(\mu+d+\alpha)(\mu-\zeta))}{\mu\pi(\mu-\zeta)} v_3 w_2^2 \qquad (31)$$

Similarly, from (3.149)

$$b = \frac{\rho(1-\kappa\theta)\pi}{(\mu+d+\alpha)\mu} v_2 w_2 \qquad (32)$$

Note that $a < 0$ if

$\zeta\alpha\rho\mu < \mu(\mu+\rho)(\mu+d+\alpha)(\mu-\zeta)$ and $a > 0$ if $\zeta\alpha\rho\mu > \mu(\mu+\rho)(\mu+d+\alpha)(\mu-\zeta)$

and $b > 0$, it follows from the theorem above (Castillo-Chaez and Song 2004) that the model (1) exhibits a supercritical (forward) bifurcation when $a < 0$ and the endemic equilibrium E* is locally asymptotically stable but the model (1) exhibits a transcritical (backward) bifurcation when $a > 0$ and the endemic equilibrium E* is locally asymptotically unstable.

The following parameters $\zeta, \alpha.$ and ρ plays an important role in the dynamic of gonorrhea disease

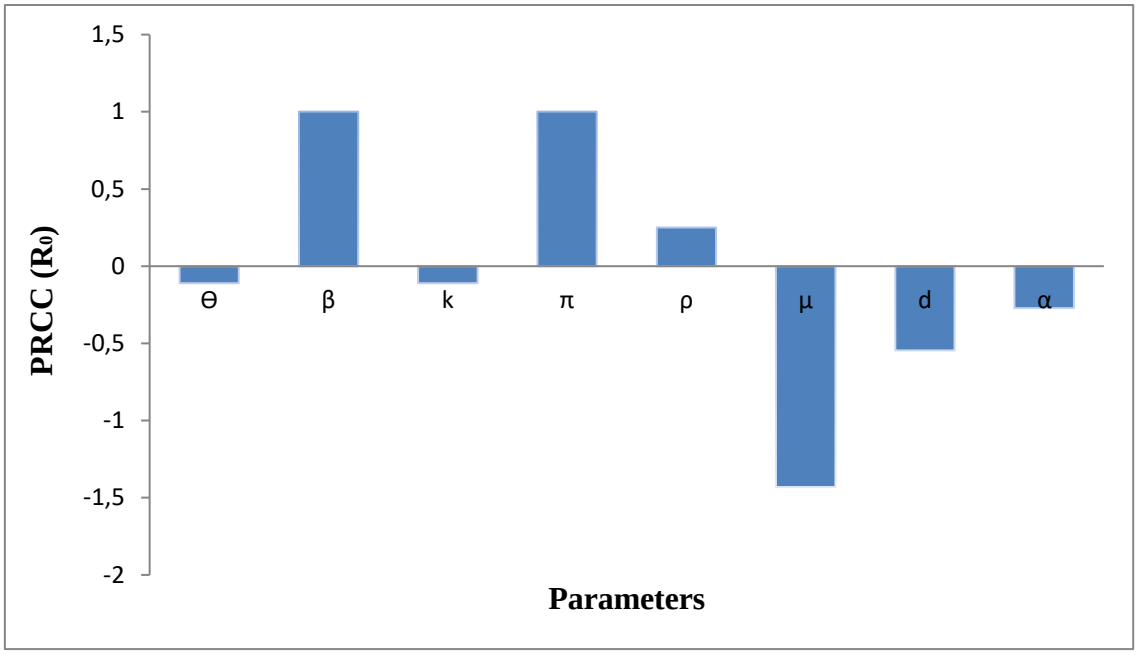


Figure 1. Distribution of PRCC values of the parameters of the model (1) wth R0 as the response function. Parameter values and ranges used are as given in Table 4.

Table 4. PRCC values of the parameters of the model (1) with R_0 as the response function.

Parameters	PRCC (R_0)
Θ	0.11111111
β	**1.00000000**
K	0.11111111
Π	**1.00000000**
ρ	0.25000000
μ	**-1.4318200**
d	**-0.5454545**
α	-0.2727273

A global sensitivity analysis, using partial rank correlation coefficients (PRCC), is carried out to determine the parameters that have the most influence on the model's dynamics (with respect to the reproduction number (R_0).

6. CONCLUSIONS

In this paper, the global stability of the endemic equilibrium point of the SEIR model for gonorrhea disease transmission proposed was examined, in the case, the following parameter;

efficacy of condom, compliance of condom, effective contact rate, natural death rate, progression rate, treatment rate increases to the burden of gonorrhea disease in the population. The existence and uniqueness of the solution and positively invariant region was carried out, we proved the existence and uniqueness of disease – free and endemic equilibrium points, depending on the value of the basic reproduction number R_o. Through Lyapunov functions and LaSalle's Invariance Principle, we proved the global stability of the disease – free equilibrium for $R_o < 1$ and the global stability of the endemic equilibrium point when $R_o > 1$ for negligible efficacy of condom use.

Sensitivity analysis shows that increasing the values of the parameters with positive index will in turn increases the burden of gonorrhea disease in the population and the vice visa. In the contrary, increasing the values of the parameters with negative index will in turn reduce the burden of gonorrhea disease in the population and decreasing the values of the parameters with negative index will in turn increases the burden of gonorrhea disease in the population.

Bifurcation analysis reveals that the model will exhibits a forward bifurcation when $a < 0$ and the endemic equilibrium E* is locally asymptotically stable and the same model will exhibits a backward bifurcation when $a > 0$ and the endemic equilibrium E* is locally asymptotically unstable. The following parameters ζ, α. and ρ are the determining factor of the nature of the bifurcation and the local stable of the endemic.

References

[1] K. L. Cooke and J. A. Yorke (1973). Some equations modelling growth processes and gonorrhea epidemics. *Math. Biosci.* 58, pp. 93-109.

[2] R. Ross (1911). The Prevention of Malaria, 2nd ed., with Addendum, John Murray, London

[3] Carlos Castillo-Chavez, Wenzhang Huang and Jia Li. Competitive Exclusion in Gonorrhea Models and Other Sexually Transmitted Diseases. *SIAM Journal on Applied Mathematics* Vol. 56, No. 2 (Apr., 1996), pp. 494-508

[4] S. Busenberg and C. Castillo-Chavez (1990), On the solution of the two-sex mixing problem, in Proceedings of the International Conference on Differential Equations and Applications to Biology and Population Dynamics, Lecture Notes in Biomathematics 92, S. Busenberg and M. Martelli, eds., Springer-Verlag, Berlin, Heidelberg, New York, London, Paris, Tokyo, Hong Kong, Barcelona, Budapest, pp. 80-98.

[5] A. J. Lotka (1923). Contributions to the analysis of malaria epidemiology. *Amer. J. Hygiene,* 3, Jan. Supplement.

[6] A. Lajmanovich and J. A. Yorke (1976). A deterministic model for gonorrhea in a nonhomoge- neous population. *Math. Biosci.* 28, pp. 221-236.

[7] H. W. Hethcote and J. A. Yorke (1984). Gonorrhea Transmission Dynamics and Control, Lecture Notes in Biomath. 56, Springer-Verlag, New York.

[8] Garnett G.P, Mertz K.J, Finelli L. The transmission dynamics of Gonorrhea: modeling the reported behavior of infected patients from Newark, New Jersey. *Philosophical Transactions of the Royal Society London*, B 1999; 354:787-97.

[9] Kretzschmar M, van Duynhoven YT, Severijnen AJ. Modeling prevention strategies for gonorrhea and Chlamydia using stochastic network simulations. *Am J Epidemiol.* 1996 Aug 1; 144(3): 306-17.

[10] Prabhakararao. G., Mathematical modeling of gonorrhea disease a case study with reference to Anantapur district-Andhrapradesh-India, *Global Journals Inc. Mathematics and Decision Sciences,* Vol 13, 2013.

[11] Leung, I. K. C., & Gopalsamy, K. (2012). Dynamics of continuous and discrete time siv models of Gonorrhea transmission. *Dynamics of Continuous, Discrete and Impulsive Systems Series B: Applications and Algorithms*, 19(3), 351-375.

[12] Lajmanvovich A and Yorke J.A. A deterministic model for gonorrhea in a Nonhomogeneous population. *Mathematical Bioscience* 28, 221-236 (1976).

[13] Ramakishore R and Pattabhiramacharyulu N. CH. A numerical approach for the spread of gonorrhea in homosexuals. *ARPN Journal of Engineering and Applied Sciences* vol. 6, no. 6, June 2011.

[14] Karnett B. M. Manifestation of gonorrhea and Chlamydia infection. *Review of Clinical Signs*, May / June 2009, pp 44-48.

[15] Benedek T. G. Gonorrhea and the beginning of clinical research ethics. *Perspectives in Biology and Medicine*, volume 48, number 1 (winter 2005): 54–73

[16] Bala M. Antimicrobial resistance in Neissseria gonorrhea south-east Asia. *Regional Health Forum* vol. 15, number 1, 2011