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Sensitivity Analysis of the Gonorrhea Disease Transmission and its Equilibria

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Abstract: In this paper we formulate an SEIR (Susceptible – Exposed - Infective - Recovered) model of Gonorrhea disease transmission with constant recruitment. The threshold parameter $R_0 < 0$, known as the Basic Reproduction Number was found. This model has two equilibria, disease-free equilibrium and endemic equilibrium. By constructing suitable Lyapunov function, it was discovered that the disease-free equilibrium is globally asymptotic stable whenever R_0 is less than one and when it is greater than one, the endemic equilibrium is globally asymptotic stable. Increasing the value of any of the parameters, $\pi, \beta or \rho$, increases the basic reproduction number, R_o, and the magnitude of the infectious individual in the community increases accordingly. Conversely, increasing the value of either $\kappa, \theta, \mu, d or \alpha$, decreases the basic reproduction number, R_o, and the magnitude of the infectious individuals in the community decreases accordingly. Therefore, it is pertinent to conclude that efforts at reducing the basic reproduction number of a disease should be encouraged in order to achieve a disease-free population.

Keywords: Stability Analysis, Basic Reproduction Number, Lyapunov function, Sensitivity Analysis

Introduction.

The formulation of the first gonorrhea model by [1] instigated the use of differential equation models to study the transmission dynamics and control of sexually transmitted diseases (STDs). However, the use of differential equations for models for STDs goes back to [2], who in 1911, introduced the first differential equation model for the transmission dynamics of vector transmitted diseases. The modeling work of [2] 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. The life cycle of this parasite requires, at different stages, human and vector hosts for its completion. Humans can only become infected by being bitten by infected vectors (female mosquitoes) and vectors can only become infected by biting infected humans. Ross's contributions to the understanding of the malaria life cycle were rewarded with a Nobel Prize in medicine.

The author of [2] made a series of observations that became important 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 contributions were extensive and deserve to be credited in this setting, as he explicitly recognized that STDs could be modeled in the same way as vector-transmitted diseases [2]. Furthermore, 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]; see also [5]. The assumption that the sizes of interacting populations were constant and not dynamic variables became an important but limiting component in the modeling of sexually transmitted diseases [6, 7].

Garnett et al. [8], examined the sexual behavior of gonorrhea patients in New York, and used it to estimate the parameters of their gonorrhea model. 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 disease involves interaction of the susceptible and the infective. Leung and Gopalsamy [11], formulated a continuous time SIV model for Gonorrhea transmission among homosexuals. They also used a non-standard discretization method to formulate a discrete time model, and they compared the results of their models. Yorke [12], modelled the spread of Gonorrhea in a population that was categorized into n group and used it to further study the asymptotic stability of the model. Kishore and Pattabhiramacharyulu [13], proposed a simple non-linear first order ODE model for Gonorrhea that measure the growth rates of promiscuous and

infective in a homosexual population. They 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.

Model Formation

The model is an heterosexually active population. The disease that guides the modeling is gonorrhea and, consequently, infective recover after treatment. 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 decreases at the rate of contact of infection β and natural death μ. The exposed increases as the infection β invade the susceptible and decrease 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. Where the N(t) means the total population.

Model Equation

We have the following non-linear system of differential equations,

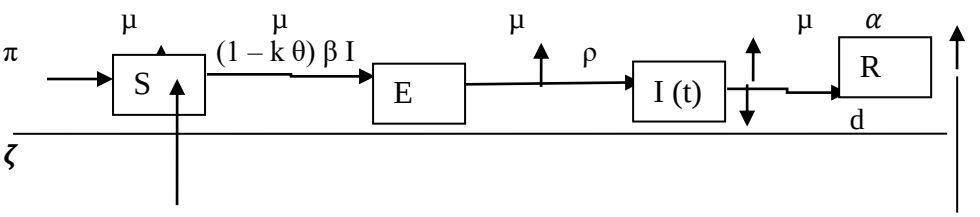
$$\frac{dS}{dt} = \pi - (1-\kappa\theta)\beta S(t) I(t) - \mu S(t) + \zeta R(t)$$

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

$$\frac{dI}{dt} = \rho E(t) - (\mu + d + \alpha)I(t)$$

$$\frac{dR}{dt} = \alpha I(t) - (\mu + \zeta)R(t)$$

Flow Chart



2.2 Table 1 Descriptions of Parameters and ValuesTable 2 Description of Variables

Parameters	Definitions	Value [17]
π	Recruitment Rate	2000
ƙ	Efficacy of Condom	0.7
θ	Compliance of Condom	0.5
β	Effective Contact Rate	0.1
μ	Natural Death Rate	0.01
ζ	Loss of Immunity	0.03
ρ	Progression Rate	0.6
d	Induced Death Rate	0.01
α	Treatment Rate	0.6
N	Total Population	Varried

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

Model Analysis

Positively Invariant Region

Theorem 1

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

Let the initial data for the model (1) be S*(0)* >0, E*(0)* >0, I*(0)* >0 and R *(0)* >0 . Then the solutions *(*S(t), E(t), I(t), R(t)*)*

of the model (2.1), with positive initial data, will remain positive for all time t >0.

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

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

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 $N(t) \leq \frac{\pi}{\mu}$

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

Therefore, all solutions of the model with initial conditions in *D*remain in *D*for all $t \geq 0$ (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 beconsidered as been epidemiologically and mathematically well posed

Existence and Uniqueness of the solution

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, Let the initial data for the model (1) be S*(0)* >0, E*(0)* >0, I*(0)* >0 and R(0) > 0. Then the solutions (S(t), E(t), I(t), R(t)) of the model (1), with positive initial data, will positive for time t > 0.

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 (2) 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.

Existence of Disease Free Equilibrium (DFE)

For critical points, we set;

$$\frac{dS}{dt} = \frac{dE}{dt} = \frac{dI}{dt} = \frac{dR}{dt} = 0 \quad (3)$$

At disease free equilibrium, we assumed there is no infection in the population.

Let $\mathcal{E}_0$ denotes the disease free equilibrium. Thus;

The model in (1) has disease free equilibrium given by

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

Existence of Endemic Equilibrium Point (EEP)

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

$$\frac{dS}{dt} \neq \frac{dE}{dt} \neq \frac{dI}{dt} \neq \frac{dR}{dt} \neq 0 \quad (5)$$

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

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

Where

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

$$A = \rho\pi\beta(\kappa\theta+1) - \mu K_1 K_2 \qquad B = (\rho\zeta\alpha - K_1 K_2 K_3)(\kappa\theta - 1)$$

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;

$$F = \begin{pmatrix} (1-\kappa\theta)\beta S I \\ 0 \\ 0 \end{pmatrix}, \quad \text{and} \quad V = \begin{pmatrix} (\mu+\rho)E \\ -\rho E + (\mu + d + \alpha)I \\ -\alpha E + (\mu+\zeta)R \end{pmatrix} \quad (7)$$

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

$$F=\begin{pmatrix} 0 & \frac{(1-\kappa\theta)\beta\pi}{\mu} & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix} \text{ at DFE} \quad (8)$$

$$V=\begin{pmatrix} \mu+\rho & 0 & 0 \\ -\rho & \mu+d+\alpha & 0 \\ -\alpha & 0 & \mu+\zeta \end{pmatrix} \quad (9)$$

Thus;

$$R_0=\frac{(1-\kappa\theta)\beta\pi\rho}{\mu(\mu+\rho)(\mu+d+\alpha)} \quad (10)$$

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].

Local Stability of the DFE

Theorem 3: The disease-freeequilibrium of the model (1) is locally asymptotically stable (LAS) if $R_0 < 1$ and unstable if $R_0 > 1$.

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 $\left(\frac{\pi}{\mu},0,0,0\right)$. We have

$$J_G=\begin{pmatrix} -(\mu+\lambda) & 0 & \frac{-\beta(1-\kappa\theta)\pi}{\mu} & \zeta \\ 0 & -(\rho+\mu)-\lambda & \frac{\beta(1-\quad 53}{\mu} & 0 \\ 0 & \sigma & -(\mu+d+\alpha)-\lambda & 0 \\ 0 & 0 & \alpha & -(\mu+\zeta)-\lambda \end{pmatrix} \quad (11)$$

The characteristics polynomial of the above matrix is given by

$$B_4\lambda^4+B_3\lambda^3+B_2\lambda^2+B_1\lambda+B_0=0 \quad (12)$$

And

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

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

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

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, alleigen-values of the polynomial are negative which shows that the disease free equilibrium is locally asymptotically stable.

Global Stability of the Disease free equilibrium

Theorem 4

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

Proof

Consider the Lyapunov function

$$V = A_1 E + A_2 I \quad (15)$$

Where,

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

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

$$\overset{*}{V} = A_1 \overset{*}{E} + A_2 \overset{*}{I} \tag{16}$$

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

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

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

$$= \frac{\rho}{(\rho+\mu)(\mu+d+\alpha)}[R_0 - 1]I(t) \tag{17}$$

Thus, $\overset{*}{V} \leq 0$ if $R_0 \leq 1$ 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 $\{\varepsilon_0\}$. 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 $R_0 \leq 1$.

Global Stability of the Endemic Equilibrium Point

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 $S \leq S^{**}$.

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).

$$F = S - S^{**} - S^{**}\ln\left(\frac{S}{S^{**}}\right) + E - E^{**} - E^{**}\ln\left(\frac{E}{E^{**}}\right) + \left(\frac{\beta S^{**}}{\mu+d+\alpha}\right)\left(I - I^{**} - I^{**}\ln\left(\frac{I}{I^{**}}\right)\right) \tag{18}$$

with Lyapunov derivative,

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

So that,

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

$$+ (1-\kappa\theta)\beta S(t)I(t) - (\rho+\mu)E(t) - \frac{E^{**}}{E}\left((1-\kappa\theta)\beta S(t)I(t) - (\rho+\mu)E(t)\right)$$

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

At steady state

$$\pi = \beta S^{**}I^{**} + \mu S^{**} - \zeta R^{**}$$

$$\rho + \mu = \frac{\beta S^{**}I^{**}}{E^{**}} \quad (21)$$

$$\rho = \frac{(\mu+d+\alpha)I^{**}}{E^{**}}$$

Simplifying the above equation, it result into

$$\dot{F} = \beta S^{**}I^{**} + \mu S^{**} - \zeta R^{**} - \mu S + \zeta R - \beta I^{**}\frac{S^{**2}}{S} - \mu\frac{S^{**2}}{S} + \zeta R^{**}\frac{S^{**}}{S} + \beta I S^{**}$$

$$+ \mu S^{**} - \zeta R\frac{S^{**}}{S} - (\rho+\mu)E - \beta I S\frac{E^{**}}{E} - (\rho+\mu)E^{**}$$

$$+ \left(\frac{\beta S^{**}}{\mu+d+\alpha}\right)\left(\rho E(t) - (\mu+d+\alpha)I(t) - \frac{I^{**}}{I}\left(\rho E(t) - (\mu+d+\alpha)I(t)\right)\right) \quad (22)$$

$$\dot{F} = \beta S^{**}I^{**} + \mu S^{**} - \zeta R^{**} - \mu S + \zeta R - \beta I^{**}\frac{S^{**2}}{S} - \mu\frac{S^{**2}}{S} + \zeta R^{**}\frac{S^{**}}{S}$$

$$+ \mu S^{**} - \zeta R\frac{S^{**}}{S} - \beta I S\frac{E^{**}}{E} + \beta S^{**}I^{**} - \beta S^{**}\frac{E I^{**2}}{E^{**}I} + \beta S^{**}I^{**}$$

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

$$+ \zeta R^{**}\left(\frac{S^{**}}{S} - \frac{R S^{**}}{R^{**} S} - 1 + \frac{R}{R^{**}}\right)$$

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

$$3 - \frac{S^{**}}{S} - \frac{E}{E^{**} I} - \frac{I S E^{**}}{I^{**} S^{**} E} \leq 0$$

$$2 - \frac{S^{**}}{S} - \frac{S}{S^{**}} \leq 0 \qquad (23)$$

$$\frac{S^{**}}{S} - \frac{R S^{**}}{R^{**} S} - 1 + \frac{R}{R^{**}} \leq 0$$

Thus, $\dot{F} \leq 0$ for $R_o^m > 1$. 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 (1) 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 $R_o^m > 1$.

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],[17].

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

$$\gamma_p^v = \frac{\partial v}{\partial p} \times \frac{p}{v} \qquad (24)$$

In particular, sensitivity indices of the basic reproduction number, R_o, with respect to the model parameter. For example, using the above equation, we obtain:

$$\gamma_{\kappa}^{R_0}=\frac{\partial R_0}{\partial \kappa}\times\frac{\kappa}{R_0}=\frac{-\kappa\theta}{1-\kappa\theta},\qquad \gamma_{\rho}^{R_0}=\frac{\partial R_0}{\partial \rho}\times\frac{\rho}{R_0}=\frac{\mu}{\mu+\rho},$$

$$\gamma_{d}^{R_0}=\frac{\partial R_0}{\partial d}\times\frac{d}{R_0}=\frac{-d}{d+\alpha+\mu},\qquad \gamma_{\theta}^{R_0}=\frac{\partial R_0}{\partial \theta}\times\frac{\theta}{R_0}=\frac{-\kappa\theta}{1-\kappa\theta},$$

$$\gamma_{\alpha}^{R_0}=\frac{\partial R_0}{\partial \alpha}\times\frac{\alpha}{R_0}=\frac{-\alpha}{d+\alpha+\mu},\qquad \gamma_{\pi}^{R_0}=\gamma_{\beta}^{R_0}=\frac{\partial R_0}{\partial \pi}\times\frac{\pi}{R_0}=\frac{\partial R_0}{\partial \pi}\times\frac{\pi}{R_0}=1$$

$$\gamma_{\mu}^{R_0}=\frac{\partial R_0}{\partial \mu}\times\frac{\mu}{R_0}=\frac{-\left[\rho(\alpha+2\mu+d)+\mu(2\alpha+3\mu+2d)\right]}{(\mu+\rho)(d+\alpha+\mu)} \qquad (25)$$

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_o of the model (1) and asymptotically results into persistence (or elimination) of the disease in the community . For instance $\gamma_{\beta}^{R_O}=1$ means that increasing (or decreasing) by β 10% increases (or decreases) $R_o$by 10%. 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).

Conclusion

This work presents both theoretical and quantitative analyses of a deterministic epidemiological model of a Gonorrhea disease infection. The results obtained are highlighted as follows:

The model is epidemiologically well posed. The solution exists and is unique.

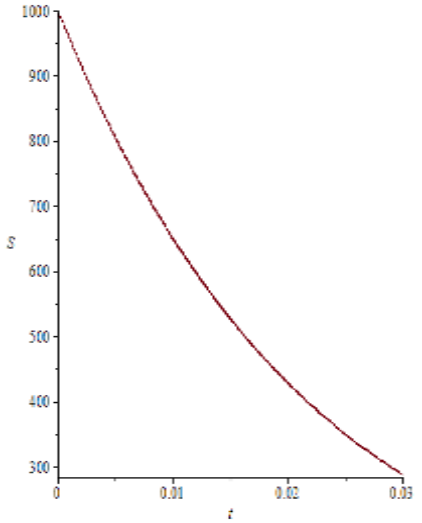
The disease-free equilibrium is locally asymptotically stable when the threshold quantity, R_o, is less than one.

The model has a globally asymptotically stable disease – free equilibrium when the threshold parameter $R_o< 1$ was done using Lyapounov function different from the method used by [17].

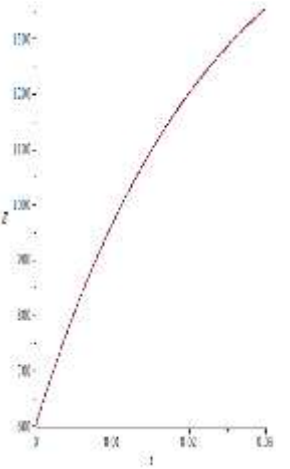
The endemic equilibrium of the formulated model is globally asymptotically stable whenever the threshold quantity, R_o, is greater than one, it was carried out using Lyapounov function different from the method used by [17].

Increasing the value of any of the parameters, $\pi, \beta \text{ or } \rho$, increases the basic reproduction number, R_o, and the magnitude of the infectious individual in the community increases accordingly. Conversely, increasing the value of either $\kappa, \theta, \mu, d \text{ or } \alpha$, decreases the basic reproduction number, R_o, and the magnitude of the infectious individuals in the community decreases accordingly.

Therefore, it is pertinent to conclude that efforts at reducing the basic reproduction number of adisease should be encouraged in order to achieve a disease-free population.

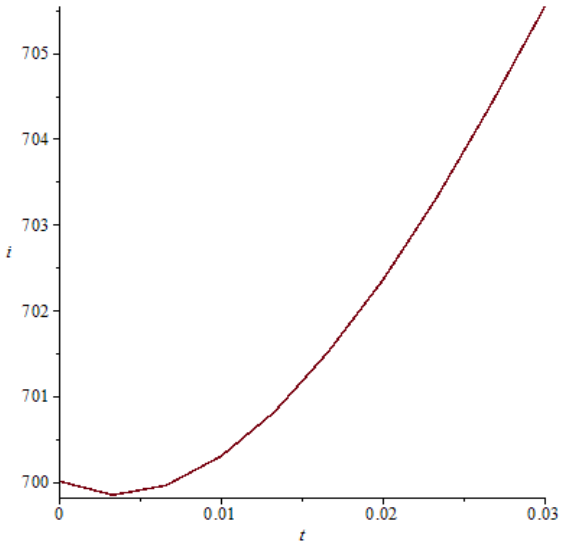


Graph of Susceptible individual

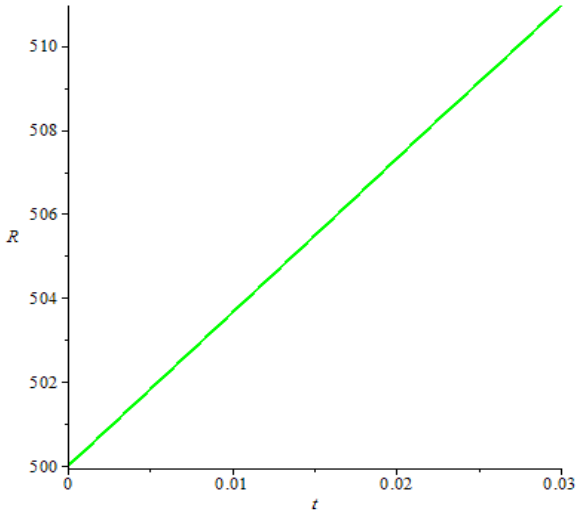


Graph of Exposed individual

Graph of Infected individual



Graph of Infected individual



Graph of Recovered individual

References

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

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

[3] Busenberg, S. and Castillo-chavez, C. (1989) Interaction, pair formation, and force of infection terms in sexually transmitted diseases, Lecture Notes in Biomath. 19, Springer- Verlag, New York, pp: 63-77.

[4] Castillo-Chavez, C., Martelli, M. and Buisenberg, S. (1990) On the solution of the two-sex i mixing problem, in Proceedings of the International Conference on Differential Equations and Applications to Biology and Population Dynamics, Lecture Notes in Biomathematics 92: 80-98.

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

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

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

[8] Garnett, G.P, Mertz, K.J, Finelli, L (1999) The transmission dynamics of Gonorrhea: modeling the reported behavior of infected patients from Newark, New Jersey Philos. Trans. Royal Soc., B; 354: 787-797.

[9] Krestzschmar, M, Yvonne, T. H, Van-Duynhoven, P and Severijnen, A. J (1996) Modeling prevention strategies for Gonorrhea and Chlamydia using stochastic network stimulation, Am. J. Epidermiol., 144, 54: 77-97.

[10] Prabhakararao, G. (2013) Mathematical modeling of gonorrhea disease a case study with reference to Anantapur district-Andhrapradesh-India, Global journals Inc., mathematics and Decision Sciences. 13, 975-996.

[11] Leung, I. K. C and Gopalsamy, K, (2012) Dynamics of continuous and discrete time SIV models of Gonorrhea transmission, Dyn. of Cont. Dis. and Imp. Sys., ser. B, 19, 3, 351-375.

[12] Lajmanvovich, A and Yorke, J.A, (1976) A deterministic model for gonorrhea in a non-homogeneous population. Mathematical bioscience 28, 221-236. American Elsevier publishing company, Inc.

[13] Ramakishore, R and Pattabhiramacharyulu, N. C. H, (2011) A numerical approach for the spread of gonorrhea in homosexuals. ARPN Journal of Engineering and Applied Sci., 6, 6, ISSN 1819 6608.

[14] Karnett, B .M. (2009) Manifestation of gonorrhea and Chlamydia infection, Review of Clinical signs. White. Pp: 44 - 48.

[15] Benedek, T .G. (2005) Gonorrhea and the beginning of clinical research ethics, Johns Hopkins University Press, V. 48, pp: 54 -73.

[16] Bala, M. (2011) Antimicrobial resistance in Neissseria gonorrhea south-east Asia, Regional health forum - vol. 15, number 1.

[17] Adesanya, A. O., Olopade, I. A., Akanni, J. O., Oladapo, A. O. and Omoloye, M. A. (2016) Mathematical and Sensitivity Analysis of efficacy of condom on the dynamical transmission of Gonorrhea disease. Imp. J. Interdiscip. Res. Finlogy Publication (IJIR). 2(11), 368-375.