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MATHEMATICAL MODELING OF GONORRHEA TREATMENT DYNAMICS WITH INCORPORATING CONTROL MEASURES

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Abstract

In this Research paper, we proposed mathematical modeling of gonorrhea infection treatment dynamics with incorporating control individual class. We obtained the Disease Free Equilibrium State and Endemic State. The criterion for stability of the Disease Free State was established using the reproduction number R_0. The model equations were solved using Homotopy Perturbation Method. The result of the numerical simulation shows that at high treatment rate of the gonorrhea Disease can be eradicated completely which will also eradicate the transfer of the disease to Human

Keyword: Mathematical Model, Dynamics, Gonorrhea Infectious, Treatment and Incorporating Control Measures

1. Introduction

Gonorrhea is a sexual transmission infectious disease; it is caused by a bacterium known as Neisseria gonorrhea, which invades the genital organs and reproductive track causing inflammation of the tube which carries sperm and infertility (Oworu *et al.*, 2013). New cases of gonorrhea diagnosed each year are estimated to be 78 million; in the united states alone there an estimated 820,000 new gonorrhoea infections each year while in 9963, WHO found Logos (Nigeria) to have the highest gonorrhea in the world (Ogunbanjo BO 1989 and Lorismith 2018). Historically, gonorrhoea was discovered in 1792, in Edinburg where the surgeon Benjamin Bell clearly differentiated it from syphilis infectious disease (Benedek, 200). It does not only invade the reproduce track, but can also attack throat, mucous membranes of the eyes, throat, mouth rectum and anus (Lorismith 2018). Untreated gonorrhea in female may lead to pelvic inflammatory disease (PID) which causes permanent damage to the reproductive organ leading to infertility while in male it develops an epididymitis condition while causes fever, severe pain and swelling (Los olivos women's medical group, 2019). Gonorrhea is transmitted sexually through the penis, vagina, mouth or anus of infected individuals spread of gonorrhoea does not depend on ejaculation; it does not have to occur for gonorrhea to be spread of acquired. Gonorrhea can also be transmitted prenatally from infected mother to baby during child delivery. The treated individuals may be reinfected if sexual contact occurs between them and infected person (CDC, 2016). Untreated gonorrhea infections in human increase a person's risk of acquiring or transmitting HIV, which may lead to AIDs (Flaming *et al.,* 1999). If a child contacts gonorrhea from infected mother during childbirth, it can cause blindness, joint infection in the body or a life threatening blood infection in the child, urgent treatment of gonorrhea infection in pregnant woman as soon as it is discovered reduces the risk of above complications. The major signs and symptoms of gonorrhea includes Vagina discharge, burning during urination, low abdominal pain, pus discharge from the male genital organ or bleeding, unusual sores, inflammation of the genital organ (Oworu *et al.,* 2013). can cause blindness, joint infection in the body or a life threatening blood infection in the child, urgent treatment of gonorrhea infection in pregnant woman as soon as it is discovered reduces the risk of above complications. The major signs and symptoms of gonorrhea includes Vagina discharge, burning during urination, low abdominal pain, pus discharge from the male genital organ or bleeding, unusual sores, inflammation of the genital organ (Oworu *et al.,* 2013).

The surest way to prevent transmission of gonorrhea or other sexually transmitted diseases is to abstain from sexual activities or to be a long term mutually monogamous relationship with a tested partner who is uninfected (CDC, 2016). According to (Semchenko EA, *et al,* 2018) Bersero is a meningococcal B vaccine approved in the united states since 2015, induces antibodies in humans that target Neisseria gonorrheae. Kate L, Seib, PhD, associated professor and research leader in the institute for glycomics at Griffith university in Southport, Australia, and colleagues the ability of Bexsero (Glaxosmith Kline) to illicit and immune response against N. gonorrhea and discovered it very protective. The vaccine was approved in the U.S for people between 10 to 25 years (Semchenko EA, *et al,* 2018). According to (Leung *et al.,* 2012), developed a continuous transmission among homosexuals. They equally applied a non – standard discretization method to formulate a discrete time model and the results of their models were compared.

2. Model Formulation

In this chapter, we developed and analyzed a mathematical model of transmission dynamics of gonorrhea by incorporating with quarantine and control measure. In this model, the mathematical

model for the human transmission dynamics were incorporated to come up with a mathematical model of gonorrhoea infection.

Following Ibrahim *et al.*, (*20*18), we shall study the transmission mechanism of gonorrhea disease using a deterministic compartmental model. In order to formulate the model mathematically, the model in this research work is a model with vital dynamics. The total host population $N(t)$ at time t is divided into six subpopulations, viz; Susceptible individuals $S(t)$, Exposed individuals $E(t)$, Asymptomatic Infected individuals $A(t)$, Infected individuals $I(t)$, Treated individuals $T(t)$ and Recovered individuals $R(t)$. Individuals are recruited into the susceptible population $S(t)$ at a constant birth rate Λ. A susceptible individual in $S(t)$ comes into contact with neisseria gonorrhea infection through an asymptomatic infectious individual in $A(t)$ at a rate β and move to $E(t)$. An individual in $E(t)$ either moves back to the susceptible compartment $S(t)$ at a rate δ_2 due to immunity response or moves to the asymptomatic infected compartment $A(t)$ after the disease incubation period at a rate α and also or moves to the infected compartment $I(t)$ after the disease incubation period at a rate α. An individual in $A(t)$ is either infected and move to infected compartment $I(t)$ at a constant rate ϕ and both of them is either treated and move to the treated compartment $T(t)$ at a constant rate δ_1, δ_3 or die as a result of the neisseria gonorrhea infection at a rate σ_I and also die as a result of the neisseria gonorrhoea asymptomatic infection at a rate σ_A. A treated individual in $T(t)$ recover with temporary immunity conferred by treatment and move to $R(t)$ at a rate γ. All individuals in $R(t)$ moves back to the susceptible compartment $S(t)$ at a rate θ after waning their temporary immunity. All individuals in the six subpopulations suffer natural mortality at a uniform constant rate μ. The cycle continues in this manner. All model parameters in this research work are strictly nonnegative. The schematic flow diagram of the model described in this section is given in figure 3.1.

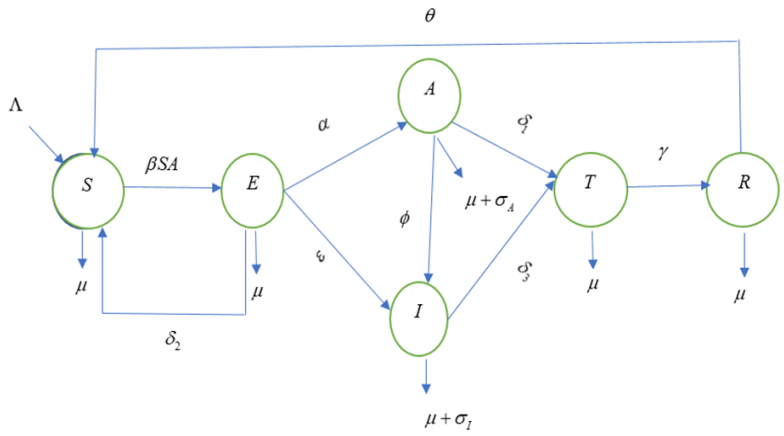

Figure 3.1 a schematic diagram of the model equation

Table 1: State variables and model parameters.

Symbol	Description
$S(t)$	Susceptible human at time t
$E(t)$	Exposed human at time t
$I(t)$	Infected human at time t
$A(t)$	Asymptomatic human at time t
$T(t)$	Treatment human at time t
$R(t)$	Recovered humans at time t
Λ	Recruitment rate into the population
β	Contact rate with infected individuals
α	Rate asymptomatic due to exposure
δ_1	Treatment rate
δ_2	Natural recovery rate due to immunity after asymptomatic
δ_3	Natural recovery rate due to immunity after infected
ε	Rate infection due to exposure
γ	Recovery rate
ϕ	Rate infected due to asymptomatic
μ	Natural death rate
σ_I	Disease-induced death rate to infected

σ_A	Disease-induced death rate to asymptomatic
θ	Proportion of recovered moving back to susceptible

2.1 The Model Equations

From the assumptions and the dynamics between the compartments shown in the model compartments in figure 1, the effect of immunization on the epidemiology of gonorrhoea infection is modeled by the following system of ordinary differential equations;

$$\left.\begin{aligned}
\frac{dS}{dt} &= \Lambda - \beta SA + \delta_2 E + \theta R - \mu S \\
\frac{dE}{dt} &= \beta SA - (\delta_2 + \alpha + \varepsilon + \mu) E \\
\frac{dI}{dt} &= \phi A + \varepsilon E - (\delta_3 + \mu + \sigma_I) I \\
\frac{dA}{dt} &= \varepsilon E - (\delta_1 + \phi + \mu + \sigma_A) A \\
\frac{dT}{dt} &= \delta_1 A + \delta_3 I - (\mu + \gamma) T \\
\frac{dR}{dt} &= \gamma T - (\mu + \theta) R
\end{aligned}\right\} \quad (1)$$

2.2 Model Analysis

We provide comprehensive qualitative analysis of the model equation in this section.

2.2.1 The Positive Invariant Region

$$N = S, E, I, A, T, R \quad (2)$$

$$\frac{dN}{dt} = \frac{dS}{dt} + \frac{dE}{dt} + \frac{dI}{dt} + \frac{dA}{dt} + \frac{dT}{dt} + \frac{dR}{dt} \quad (3)$$

Adding equation (1) we have;

$$\frac{dN}{dt} = \Lambda - \mu N - \sigma_I I(t) - \sigma_A A(t) \quad (4)$$

The positive invariant region can be obtained by using the following theorem as applied by

(Adedayo *et al.*, 2023).

Theorem 1: The solutions of the system of equations (1) are feasible if they are contained in the invariant region Ω. $\forall \ \ t \geq 0$.

Proof:

Let $\eta = (S, E, I, A, T, R,) \in \Re_+^6$ (5)

Be any solution of the system of equations (1) with non-negative initial conditions. Then from equation (4) we have

$$\frac{dN}{dt} \leq \Lambda - \mu N(t) \tag{6}$$

by multiplying through with the integrating factor on equation (5) we obtain;

$$e^{\mu t}(\frac{dN}{dt} + \mu N) \leq \Lambda e^{\mu t} \tag{9}$$

By virtue of product rule in reverse we have;

$$\Rightarrow \frac{d}{dt}(Ne^{\mu t}) \leq \Lambda e^{\mu t} \tag{10}$$

Integrating both sides;

We have;

$$Ne^{\mu t} \leq \frac{\Lambda}{\mu} e^{\mu t} + c \tag{12}$$

Applying the initial conditions $t = 0, N(0) = N_0$

$$N_0 \leq \frac{\Lambda}{\mu} + c \Rightarrow N_0 - \frac{\Lambda}{\mu} \leq c \tag{15}$$

From equation (11)

$$Ne^{\mu t} - \frac{\Lambda}{\mu} e^{\mu t} \leq c \tag{16}$$

Comparing (15) and (16)

$$N(t) \leq \frac{\Lambda}{\mu} + (N_0 - \frac{\Lambda}{\mu})e^{-\mu t} \tag{19}$$

At $t \to \infty$ in equation (20) the human population $N(t)$ approaches $\kappa = \frac{\Lambda}{\mu}$ (that is $N \to k = \frac{\Lambda}{\mu}$)

the parameter $\kappa = \frac{\Lambda}{\mu}$ is called the carrying capacity.

Therefore, all feasible solution of the human population of the Model is in the region

$$\Omega = \{(S,V,I,H,P,D) \in \Re^6 : S > 0, V > 0, I > 0, H > 0, P > 0, D > 0, N \leq \frac{\Lambda}{\mu}\} \quad (20)$$

Therefore, the region D is positively-invariant and system (1) is epidemiologically meaningful and mathematically well-posed in the domain D.

2.2.2 Positivity of the Solutions

Theorem 3 Let the initial solutions be $\{(S(0), V(0), I(0), H(0), P(0), D(0) \geq 0\} \in \Omega$ then the solutions $\{S(t), V(t), I(t), H(t), P(t), D(t)\}$ of the system (3.1 to 3.6) is positive $\forall t \geq 0$

Proof

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

$$\frac{dS(t)}{dt} = \Lambda - \beta SA + \delta_2 E + \theta R - \mu S \geq -\mu S \quad (21)$$

$$\frac{dS(t)}{dt} \geq -\mu S \quad (22)$$

By separating the variable and integrating equation we have;

$$\int \frac{dS}{S} \geq -\int \mu dt \quad (23)$$

We have,

$$\ln S \geq -\mu t + c \quad (24)$$

Take the e of both sides

$$e^{\ln S} \geq e^{-\mu t + c} \quad (25)$$

we have

$$S(t) \geq e^{-\mu t + c} \quad (26)$$

$$= S(t) \geq e^{-\mu t}.e^{c} \text{ let } e^{c} = A \quad (27)$$

$$S(t) \geq Ae^{-\mu t} \quad (28)$$

where $K = e^c$ using the initial condition $t = 0 \Rightarrow S(0) \geq K$

therefore,

$$S(t) \geq S(0)e^{-(\mu+\alpha)t} > 0 \tag{29}$$

From equation (2) we have;

$$\frac{dE(t)}{dt} = \beta SA - (\delta_2 + \alpha + \varepsilon + \mu)E \geq -(\delta_2 + \alpha + \varepsilon + \mu)E \tag{30}$$

$$\frac{dE(t)}{dt} \geq -(\delta_2 + \alpha + \varepsilon + \mu)E \tag{31}$$

$$E \geq kE = e^{-(\delta_2+\alpha+\varepsilon+\mu)t} \tag{32}$$

Where $k = E = e^c$

where $K = e^c$ using the initial condition $t = 0 \Rightarrow E(0) \geq K$

therefore,

Therefore $E(t) \geq E(0)E = e^{-(\delta_2+\alpha+\varepsilon+\mu)t} \geq 0$ (33)

Similarly, it can be verified that the rest of the equations are positive for all $t > 0$,

3.3 Disease Free Equilibrium State

The disease-free equilibrium of the model (1) is obtained by setting

$$\frac{dS}{dt} = \frac{dE}{dt} = \frac{dI}{dt} = \frac{dA}{dt} = \frac{dT}{dt} = \frac{dR}{dt} = 0 \tag{34}$$

In this case there is no disease: $E = I = A = T$. Hence, the DFE of our equation is given by:

Therefore, the disease free equilibrium state (D.F.E.S) is given as

$$E^0 = (S^0, E^0, I^0, A^0, T^0, R^0) = \left(\frac{\Lambda}{\mu}, 0, 0, 0, 0, 0\right) \tag{35}$$

3.4 Basic Reproduction Number (R_0)

Basic Reproduction Number (R_0) defined as the average number of secondary infections produced by individuals that are infectious during his or her entire period of infectiousness. R_0 determines

if a disease will persist or will die out in a community. If $R_0 < 1$ it indicates that infectious individual will cause less than one secondary infection and hence the disease will not remain, then when $R_0 > 1$ the disease will take over the population. In a more complicated epidemic, the R_0 can be calculated by using the next generation operator approach by (van den Driessche & Watmough, 2002).

From the system (1) we define f_i and v_i as:

$$f_i = \begin{pmatrix} \beta SA \\ 0 \\ \phi A + \varepsilon E \end{pmatrix} \text{ and } v_i = v^- - v^+ = \begin{pmatrix} (\delta_2 + \alpha + \varepsilon + \mu)E \\ (\delta_1 + \phi + \mu + \sigma_A)A - \alpha E \\ (\delta_3 + \mu + \sigma_I)I \end{pmatrix} \tag{36}$$

$$FV^{-1} = \left(\frac{dF_i}{dx_i}\right)\left(\frac{dV_i}{dx_i}\right)^{-1} \tag{37}$$

$$FV^{-1} = \begin{pmatrix} 0 & 0 & \dfrac{\beta\Lambda}{\mu(\delta_3 + \mu + \sigma_I)} \\ \dfrac{\alpha}{(\delta_2 + \alpha + \varepsilon + \mu)(\delta_3 + \mu + \sigma_I)} & 0 & 0 \\ \dfrac{\varepsilon}{(\delta_2 + \alpha + \varepsilon + \mu)} & 0 & \dfrac{\phi}{(\delta_3 + \mu + \sigma_I)} \end{pmatrix} \tag{38}$$

$$\left|FV^{-1} - I\lambda\right| = 0$$

$$\begin{pmatrix} -\lambda_1 & 0 & \dfrac{\beta\Lambda}{\mu(\delta_3 + \mu + \sigma_I)} \\ \dfrac{\alpha}{(\delta_2 + \alpha + \varepsilon + \mu)(\delta_3 + \mu + \sigma_I)} & -\lambda_2 & 0 \\ \dfrac{\varepsilon}{(\delta_2 + \alpha + \varepsilon + \mu)} & 0 & \dfrac{\phi}{(\delta_3 + \mu + \sigma_I)} - \lambda_3 \end{pmatrix} = 0 \tag{39}$$

$$-\lambda_1\begin{vmatrix}-\lambda_2 & 0\\ 0 & \dfrac{\phi}{\left(\delta_3+\mu+\sigma_I\right)}-\lambda_3\end{vmatrix}+\frac{\beta\Lambda}{\mu\left(\delta_3+\mu+\sigma_I\right)}\begin{vmatrix}\dfrac{\alpha}{\left(\delta_2+\alpha+\varepsilon+\mu\right)\left(\delta_3+\mu+\sigma_I\right)} & -\lambda_2\\ \dfrac{\varepsilon}{\left(\delta_2+\alpha+\varepsilon+\mu\right)} & 0\end{vmatrix}=0 \tag{40}$$

Therefore, the reproduction number

$$R_0=\frac{\varepsilon\beta\Lambda}{\left(\delta_2+\alpha+\varepsilon+\mu\right)\mu\left(\delta_3+\mu+\sigma_I\right)} \tag{41}$$

3.5 Local Stability Analysis of Disease Free Equilibrium State.

Theorem 3: The disease-free equilibrium, E^* of (41) is locally asymptotically stable (LAS) in D if $R_0<1$

Proof: We shall use Jacobean stability technique to carry out the local stability analysis of the disease disease-free equilibrium.

Jacobean matrix of the system of equations at disease-free equilibrium is:

Where $B_1=\left(\delta_2+\alpha+\varepsilon+\mu\right), B_2=\left(\delta_I+\phi+\mu+\sigma_A\right),\left(\delta_3+\mu+\sigma_I\right), B_4=\left(\mu+\gamma\right)$ and $B_5=\left(\mu+\theta\right)$

$\left(\frac{\Lambda}{\mu},0,0,0,0,0\right)$

$$J\left(E^0\right)=\begin{bmatrix}-\mu & \delta_2 & 0 & -\dfrac{\beta\Lambda}{\mu} & 0 & \theta\\ 0 & -B_1 & 0 & \dfrac{\beta\Lambda}{\mu} & 0 & 0\\ 0 & \alpha & -B_2 & 0 & 0 & 0\\ 0 & \varepsilon & \phi & -B_3 & 0 & 0\\ 0 & 0 & \delta_1 & \delta_3 & -B_4 & 0\\ 0 & 0 & 0 & 0 & \gamma & -B_5\end{bmatrix} \tag{42}$$

Reducing equation (3.208) to upper triangular matrix,

$$\left|J\left(E^0\right)-\lambda I\right|=$$

$$\begin{bmatrix} -\mu-\lambda & \delta_2 & 0 & -\frac{\beta\Lambda}{\mu} & 0 & \theta \\ 0 & -B_1-\lambda & 0 & \frac{\beta\Lambda}{\mu} & 0 & 0 \\ 0 & 0 & -B_2-\lambda & \frac{\alpha\beta\Lambda}{B_1\mu} & 0 & 0 \\ 0 & 0 & 0 & -\frac{\mu B_1B_2B_3-\phi\Lambda\alpha\beta-\Lambda\beta\varepsilon B_2}{B_2B_1\mu}-\lambda & 0 & 0 \\ 0 & 0 & 0 & 0 & -B_4-\lambda & 0 \\ 0 & 0 & 0 & 0 & 0 & -B_5-\lambda \end{bmatrix}=0 \quad (43)$$

The determinates gives;

$$\begin{bmatrix} \left(-\mu-\lambda_1\right)\left(-B_1-\lambda_2\right)\left(-B_2-\lambda_3\right)\left(-\frac{\mu B_1B_2B_3-\phi\Lambda\alpha\beta-\Lambda\beta\varepsilon B_2}{B_2B_1\mu}-\lambda_4\right)\left(-B_4-\lambda_5\right) \\ \left(-B_6-\lambda_6\right)=0 \end{bmatrix} \quad (44)$$

Therefore,

$$\lambda_1=-\mu \text{ or } \lambda_2=-B_1 \text{ or } \lambda_3=-B_2 \text{ or } \lambda_4=-\frac{\mu B_1B_2B_3-\phi\Lambda\alpha\beta-\Lambda\beta\varepsilon B_2}{B_2B_1\mu} \text{ or } \lambda_5=-B_4 \text{ or}$$

$$\lambda_6=-B_5 \quad (45)$$

From equation (45)

$$\lambda_1,\lambda_2,\lambda_3,\lambda_4,\lambda_5,\lambda_6<0 \quad (46)$$

Hence, DFE is Locally Asymptotically Stable (LAS) if $R_0<1$. The epidemiology implication of the theorem is that polio can be eliminated (control) from the population when $R_0<1$, if the initial size of the sub-populations are in the basin of attraction of the DFE.

3.6 Global Stability of Disease Free Equilibrium (E^*)

Theorem 3.9.1 The disease free equilibrium of equations (1) to (6) is globally asymptotically stable provided $R_O<1$ and unstable if $R_O>1$

Proof: Referring to Castillo-Chaves *et al.* (2002), the system of equations (1) to (6) can be written as,

$$\begin{aligned}\frac{dx(t)}{dt} &= F(x,y),\\ \frac{dy(t)}{dt} &= G(x,y)\end{aligned} \tag{47}$$

Where $x=(S,T,R)\in\Re_+^3$ denote the different compartments of uninfected humans, $y=(E,A,I)\in\Re_+^3$ denote the different compartments of infected humans.

The disease free equilibrium (DFE)$=(x_0,0)$, where $x_0=\left(\frac{\Lambda}{\mu},0,0\right)$

We are required to proof that,

$\frac{dx(t)}{dt}=F(x,0)$, x_0 is globally asymptotic ally stable, and
$G(x,y)=Cy-\hat{G}(x,y)$, $\hat{G}(x,y)\geq 0$ for $(x,y)\in\Omega$

Case 1: consider the uninfected subsystem,

$$\frac{dx(t)}{dt}=F(x,y)=\begin{pmatrix}\Lambda-\beta SA+\delta_2 E+\theta R-\mu S\\ \delta_1 A+\delta_3 I-(\mu+\gamma)T\\ \gamma T-(\theta+\mu)R\end{pmatrix} \tag{48}$$

When $y=0$ that is $E=A=I=0$

Then, equation (48) becomes,

$$F(x,0)=\begin{pmatrix}\Lambda+\theta R-\mu S\\ (\mu+\gamma)T\\ \gamma T-(\theta+\mu)R\end{pmatrix} \tag{49}$$

Solving equation (49), gives

$$\frac{dS(t)}{dt}=\Lambda+\theta R-\mu S \tag{50}$$

$$S(t)=\left(\frac{\Lambda+\theta R}{\mu}\right)-\left(\frac{\Lambda+\theta R}{\mu}\right)e^{-\mu t}+S(0)e^{-\mu t} \tag{51}$$

For T

$$\frac{dT(t)}{dt}=(\mu+\gamma)T \tag{52}$$

$$T(t) = T(0)\, e^{-(\mu+\gamma)t} \tag{53}$$

For $R(t)$

$$\frac{dR(t)}{dt} = \gamma T - (\theta+\mu)R \tag{54}$$

$$R(t) = \left(\frac{\gamma T}{(\theta+\mu)}\right) - \left(\frac{\gamma T}{(\theta+\mu)}\right) e^{-(\theta+\mu)t} + R(0)\, e^{-(\theta+\mu)t} \tag{55}$$

As $t \to \infty$, $S \to \frac{\pi}{\mu}$, $T \to 0$ and $R \to 0$ regardless of the value of $S(0)$, $T(0)$ and $R(0)$

Therefore,

$x_0 = \left(\frac{\Lambda}{\mu}, 0, 0\right)$ is globally asymptotically stable.

Case 2: consider an infected subsystem

$$y' = G(x,y) = \begin{pmatrix} \beta SA - (\delta_2 + \alpha + \alpha + \mu)E \\ \alpha E - (\delta_1 + \phi + \mu + \sigma_A)A \\ \phi A + \varepsilon E - (\delta_3 + \mu + \sigma_I)I \end{pmatrix} \tag{56}$$

Given that,

$$G(x,y) = Cy - \hat{G}(x,y) \tag{57}$$

Then,

$$\hat{G}(x,y) = Cy - G(x,y) \tag{58}$$

$$\hat{G}(x,y) = \begin{pmatrix} \hat{G}_1(x,y) \\ \hat{G}_2(x,y) \\ \hat{G}_3(x,y) \end{pmatrix} = \begin{pmatrix} 0 \\ 0 \\ 0 \end{pmatrix} \tag{59}$$

Then $\hat{G}(x,y) = 0$. (60)

Therefore the disease free equilibrium point is globally asymptotically stable when $R_0 < 1$

3.7 Existence of Endemic Equilibrium Point in Terms of force of Infection

Let $E^1 = (S, E, A, I, T, R) = (S^1, E^1, A^1, I^1, T^1, R^1)$ is the endemic equilibrium point.

Suppose $A \neq 0$

Hence, the endemic equilibrium points of our model equation in terms of forces of infection are given as;

$$\begin{pmatrix} S^* \\ E^* \\ A^* \\ I^* \\ T^* \\ R^* \end{pmatrix} = \begin{pmatrix} \frac{(\delta_2+\alpha+\varepsilon+\mu)(\delta_1+\phi+\mu+\sigma_A)}{\varepsilon\beta} \\ \frac{\varepsilon\beta\Lambda+\varepsilon\beta\theta D_1-\mu(\delta_2+\alpha+\varepsilon+\mu)(\delta_1+\phi+\mu+\sigma_A)}{\varepsilon\beta(\alpha+\varepsilon+\mu)} \\ \frac{(\varepsilon\beta\Lambda+\varepsilon\beta\theta D_1-\mu B_1B_2)}{B_2\beta(\alpha+\varepsilon+\mu)} \\ \frac{\phi(\varepsilon\beta\Lambda+\varepsilon\beta\theta D_1-\mu B_1B_2)+B_2(\varepsilon\beta\Lambda+\varepsilon\beta\theta D_1-\mu B_1B_2)}{B_2B_3\beta(\alpha+\varepsilon+\mu)} \\ \frac{\delta_1B_3(\varepsilon\beta\Lambda+\varepsilon\beta\theta D_1-\mu B_1B_2)+\delta_3(\phi(\varepsilon\beta\Lambda+\varepsilon\beta\theta D_1-\mu B_1B_2)+B_2(\varepsilon\beta\Lambda+\varepsilon\beta\theta D_1-\mu B_1B_2))}{B_2B_3B_4\beta(\alpha+\varepsilon+\mu)} \\ \frac{\gamma\delta_1B_3\varepsilon\beta\Lambda-\gamma\delta_1B_3\mu B_1B_2+\delta_3\phi\varepsilon\beta\Lambda-\delta_3\phi\mu B_1B_2+B_2\varepsilon\beta\Lambda-\mu B_2B_1B_2}{(B_2B_3B_4\beta(\alpha+\varepsilon+\mu)B_5-\gamma\delta_1B_3\theta-\delta_3\phi\varepsilon\beta\theta-B_2\varepsilon\beta\theta)} \end{pmatrix} \quad (61)$$

4. Numerical Simulation

It is difficult to get a reliable data; we estimated the parameter value based on the available data from the Nigeria Centre for Disease Control (NCDC) and reliable literature. The estimates are clearly explained in the following sub-sections as shown in Table 4.1.

Table 4.1 Shows Initial Conditions for Each Plot and Parameters Value

Parameters and State Variables	Value	Source
$S(t)$	2300	Assumed
$E(t)$	1340	Semchenko *et al.*, (2018)
$I(t)$	1200	Assumed
$A(t)$	850	Assumed

$T(t)$	780	Assumed
$R(t)$	680	Semchenko et al., 2018
β	0.00020	Semchenko *et al.,(2018)*
μ	0.0012	Assumed
δ_1	0.02	Semchenko *et al.,(2018)*
δ_2	0.02	Assumed
δ_3	0.02	Assumed
ε	0.002	Assumed
γ	0.3	Semchenko *et al.,* (2018)
Λ	0.320	Semchenko *et al.,(2018)*
ϕ	0.010	Assumed
α	0.02	Assumed
τ_I	0.05	Assumed
τ_A	0.04	Assumed
θ	0.05	Assumed

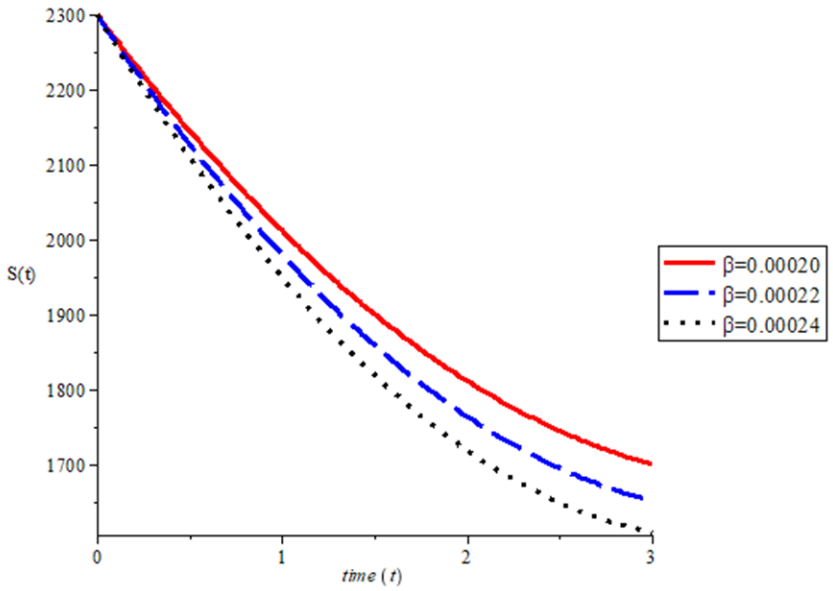

Figure 4.1: Graph of Susceptible Individuals Against Time for Different Contact rate with infected individuals.

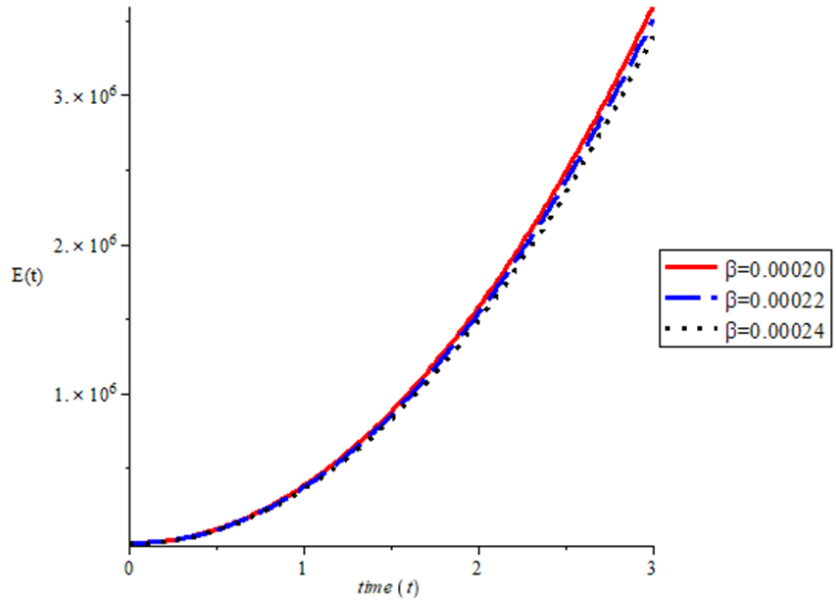

Figure 4.2: Graph of Exposed Individuals Against Time for Different Contact rate with infected individuals.

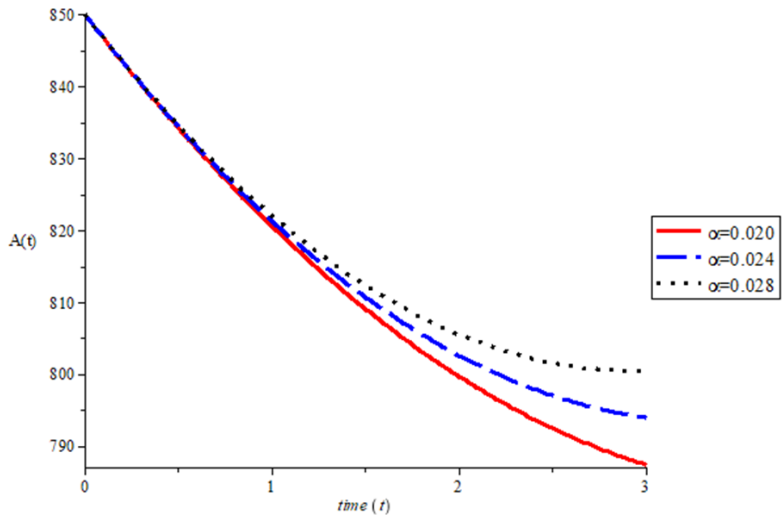

Figure 4.3: Graph of Asymptomatic Individuals Against Time for Different Rate asymptomatic due to exposure.

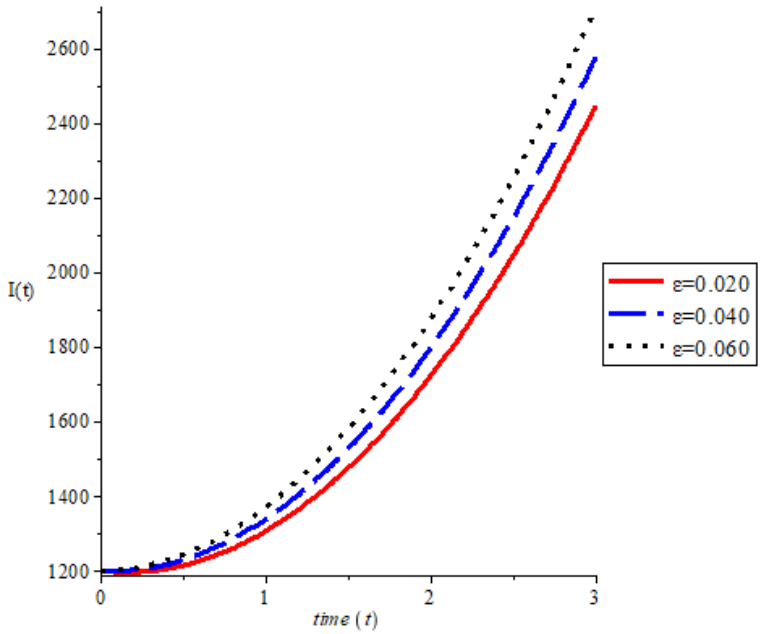

Figure 4.4: Graph of Infected Individuals Against Time at Different Rate infection due to exposure

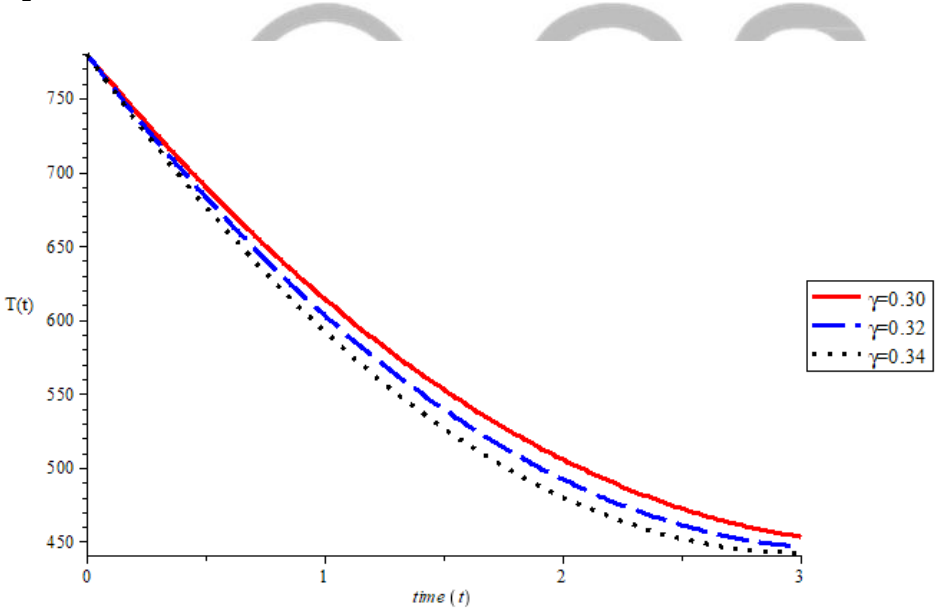

Figure 4.5: Graph of Treatment Individuals Against Time at Different Recovery rate.

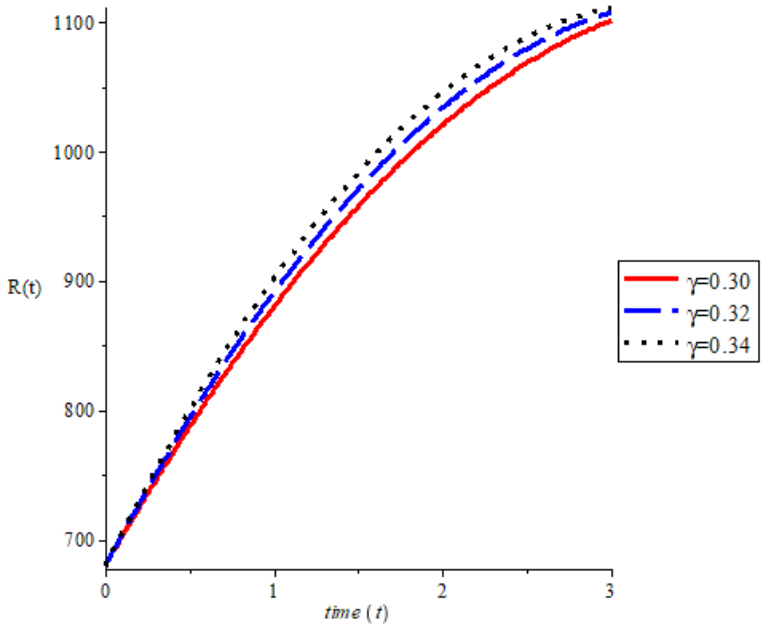

Figure 4.6: Graph of Recovered Individuals Against Time at Different Recovery rate

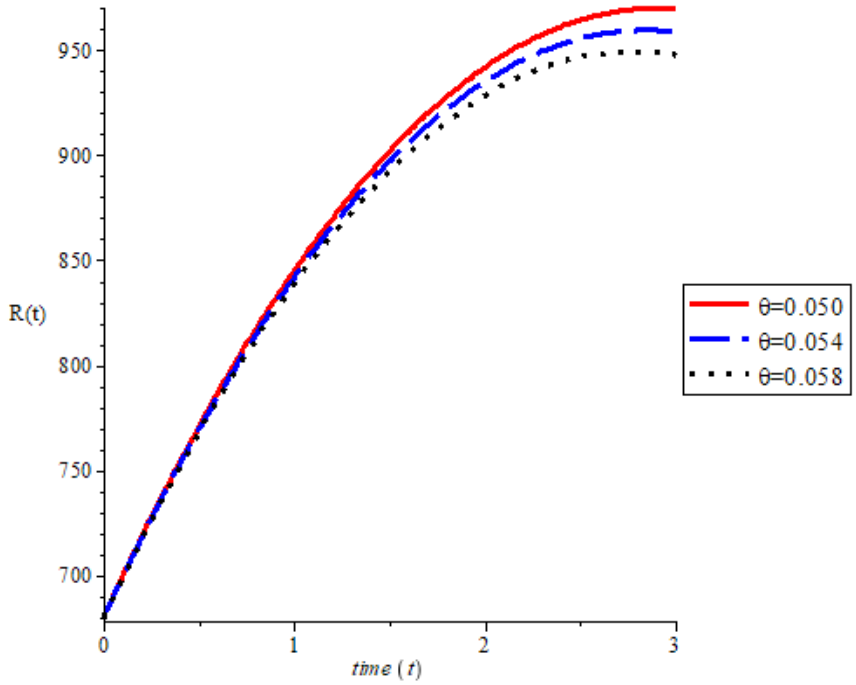

Figure 4.7: Graph of Recovered Individuals Against Time at Different Proportion of recovered moving back to susceptible.

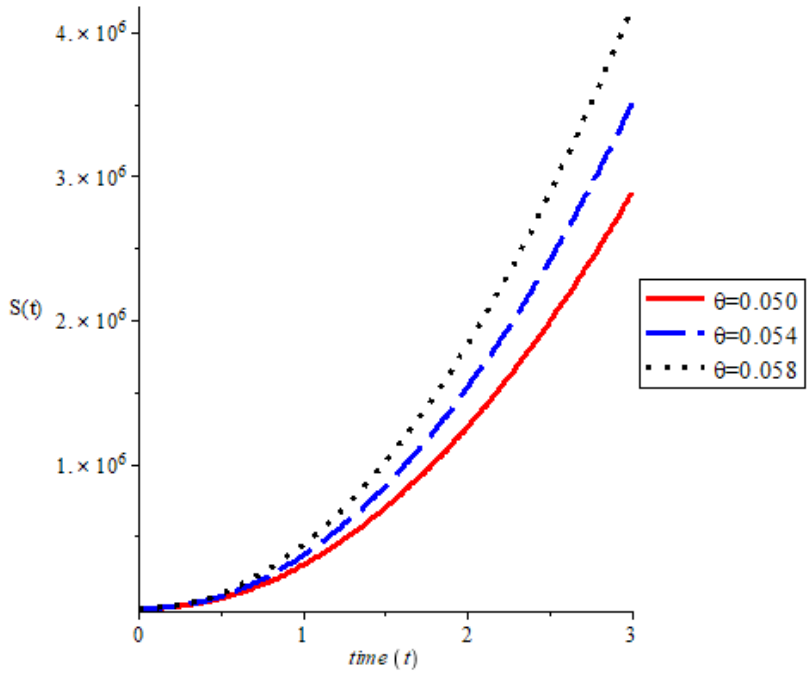

Figure 4.8: Graph of Susceptible Individuals Against Time at Different Proportion of recovered moving back to susceptible.

4.1 Discussion

Figure 4.1: is the graph of Susceptible Individuals Against Time for Different Contact rate with infected individuals. It is observed that the population susceptible individual decreases as the rate of the Contact rate with infected individual's decreases. Figure 4.2: is the graph of Exposed Individuals Against Time for Different Contact rate with infected individuals. It is observed that the population of Exposed individuals increases as the Contact rate with infected individual's decreases. Figure 4.3: is the graph Asymptomatic Individuals Against Time for Different Rate asymptomatic due to exposure. It is observed that the population Asymptomatic Individuals decrease as the asymptomatic due to exposure increase. Figure 4.4: is the graph of Infected Individuals Against Time at Different Rate infection due to exposure. It is observed that the population of the infected individuals increases as the rate infection due to exposure increases. Figure 4.5: is the graph of Treatment Individuals Against Time at Different Recovery rate. It is observed that the population of the treatment individuals decreases as the rate become recovery decreases. Figure 4.6: is the graph of Recovered Individuals Against Time at Different Recovery rate. It is observed that the population of the recovered individuals increases as the rate become recovery increases. Figure 4.7: is the graph of Recovered Individuals Against Time at Different

Proportion of recovered moving back to susceptible. It is observed that the population of the recovered individuals increases as the Proportion of recovered moving back to susceptible rates decreases. Figure 4.8: is the graph of Susceptible Individuals Against Time at Different Proportion of recovered moving back to susceptible. It is observed that the population of susceptible individuals increases as Proportion of recovered moving back to susceptible rate increases.

5. Concluding Remarks

In this work, mathematical model of gonorrhoea treatment dynamics with incorporating control measures was developed and analyzed in this study. The model is a first order Ordinary Differential Equations, in which the human population is divided into six compartments namely; Susceptible individual (S), Exposed individual (E), Asymptomatic infected individual(A), Infected individual(I), Treatment individual (T) and Recovered individual (R). he models solution was found to be positive (establishing the fact that human population cannot be negative). The equilibrium states were obtained and their stabilities were analyzed. The result shows that the disease free equilibrium state is stable and the criteria for stability of the endemic equilibrium state are established. This suggests that the treatment can used to handle the human in the population.

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