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Perturbation and bifurcation analysis of a gonorrhoea dynamics model with control

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ABSTRACT

A model for the transmission dynamics of Gonorrhoea with control incorporating passive immunity is formulated. We show that the introduction of treatment or control parameters leads to transcritical bifurcation. The backward bifurcation coefficients were calculated and their numerical perturbation results in different forms of equilibria. The calculated effective reproduction number of the model with control is sufficiently small. This implies asymptotically stability of the solution, thus, the disease can be controlled in a limited time.

1. Introduction

Due to the increasing rate of infertility among the teaming population as a result of sexually transmitted infections, it becomes necessary to undertake prompt prevention and control activities to tackle the ugly incidence of sexually transmitted diseases. Gonorrhea is one of such sexually transmitted infectious diseases caused by a bacterium called Neisseria gonorrhea (Unemo, 2015). Neisseria gonorrhoea is characterized by a very short period of latency, namely, $2-10$ days (Mushayabasa and Bhunu, 2011). It is commonly found in the glummer epithelium such as the urethra and endo-cervix epithelia of the reproductive track (Garba et al., 2013). Gonorrhoea is transmitted to a newborn infant from the infected mother through the birth canal thereby causing inflammations and eye infections such as conjunctivitis. It is also spread through unprotected sexual intercourse (Ugwu, 2015).

Studies by Usman and Adamu (2017) and the Center for Disease Control Report show that male patients with gonorrhoea have pains in the testicles (known as epididymitis), and painful urination due to scaring inside the urethra while in female patients, the disease may ascend the genital tract and block the fallopian tube leading to pelvic inflammatory disease (PID) and infertility, see also (Rama and Pattabhiramacharyulu, 2011). Other complications associated with this epidemic include arthritis, endocarditis, chronic pelvic pain, meningitis, and ectopic pregnancy; (Riley et al., 2003; Workowski and Bolan, 2015).

Gonorrhoea confers temporal immunity on some individuals in the susceptible class while some others are not immuned (Ugwu, 2015). This immunity through the immune system plays an important role in protecting the body against infection and other foreign substances (CDC, 2013). That is why an immuno-compromised patient has a reduced ability to fight infectious diseases such as gonorrhoea due to certain diseases and a genetic disorder. Such patients may be particularly vulnerable to opportunistic infections such as gonorrhoea. Hence, the immune reaction can be stimulated by a drug-induced immune system such as Thrombocytopenia. This helps to reduce the waning rate of passive immunity in the immune class (And and Henry, 2021). However, if the activity of the immune system is excessive or over-reactive due to a lack of cell-mediated immunity, a hypersensitive reaction develops such as autoimmunity and allergy which may be injurious to the body or may even cause death (WHO, 2006).

Statistically, gonorrhoea infection has spread worldwide with more than 360 million new cases witnessed globally in adults aged $15-49$ years

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(CDC, 2013). In 1999, over 120 million people in African countries were reported to have contracted the disease. While over 82 million people were reported in Nigeria (CDC, 2013). Researches abound on the modeling and control of this epidemic with various approaches and controls (CDC, 2013; Fu et al., 2015; Mushayabasa et al., 2011; Nana-Kyere et al., 2016; Ugwu, 2015; Unemo, 2015; Adamu and Usman, 2018; Whittles et al., 2020; Osnes et al., 2020; Didelot et al., 2021). This present study continues the discussion by incorporating passive immunity in the model and introducing control measures capable of eliminating the disease in Nigeria. To validate the claim, we employ perturbation and bifurcation of the model variables and parameters and mathematically analyze the stability of the system. This underscores the role of mathematical analysis of models to elicit desired results (Omenyi and Uchenna, 2019; Omenyi et al., 2021). Education and enlightenment, use of condoms, and treatment of patients with ampicillin and azithromycin are the control measures adopted to eradicate the disease.

2. Materials and methods

To formulate the model, in time t, we let $Q(t)$ be the passive immune class, $S(t)$ the susceptible compartment, $L(t)$ the latent class $I(t)$, the infectious class, $T(t)$, the treated class and $R(t)$ be the recovered compartment. Let the parameters of the model σ as the level of recruitment, υ as the waning rate of immunity, μ as the rate of natural mortality, λ as the contact rate between the susceptible and the latent classes, η as the treatment rate of latent class, γ as induced death rate due to the infection, α as treatment rate of the infected compartment, β as the infectious rate of latent class, ω as the recovery rate of treated class, δ as the rate at which recovered class become susceptible again, θ as the infectious rate from the susceptible class direct to the infectious class, k_1 as control measure given to the latent class as k_2 as a control measure given to the infected class.

We assume that recruitment into the population is by birth or immigration; all the parameters of the model are positive, some proportions of the new birth are immunized against the infection; the immunity conferred on the new birth wanes after some time, and the rate of contact of the disease due to interaction λ rate is due to the movement of the infected population. Consequently, the total population at time t is:

$$N(t) = Q(t) + S(t) + L(t) + I(t) + T(t) + R(t).$$

So, the flow diagram of the model is shown in Fig. 1.

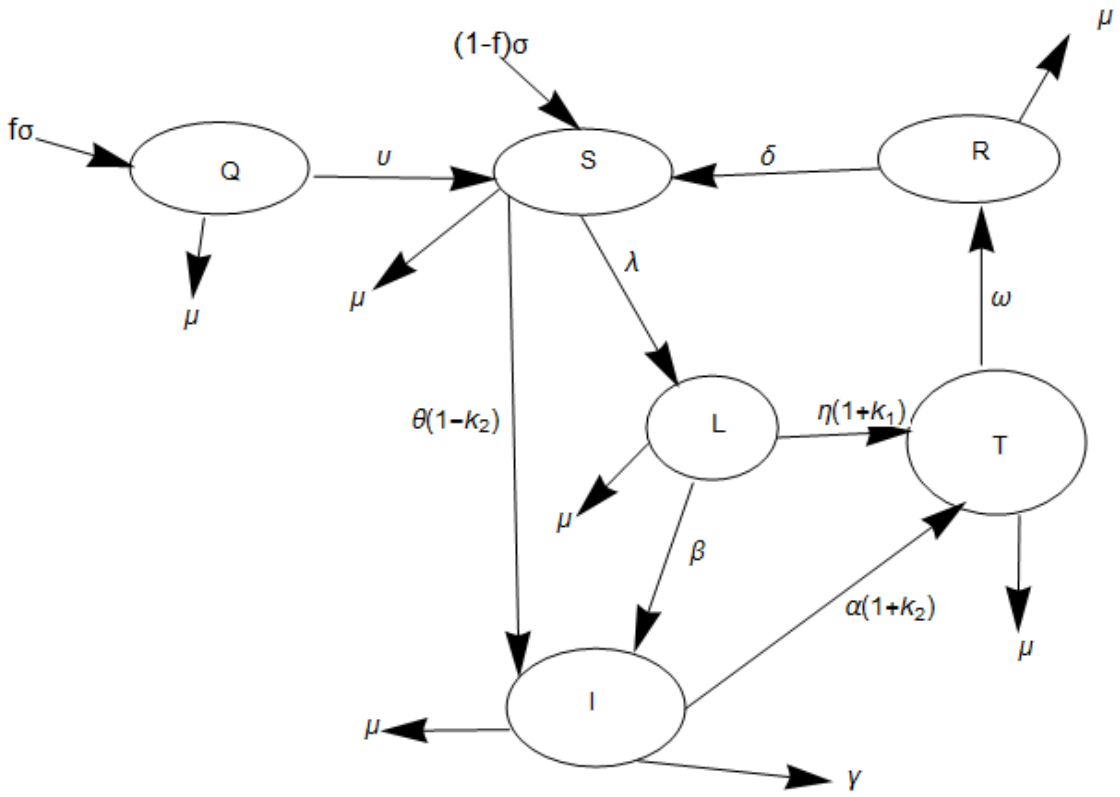

Fig. 1: The extended model with control

So, the model for the gonorrhea transmission dynamics is given by the following deterministic systems of non-linear differential Eq. 1:

$$\left.\begin{aligned}
\frac{dQ}{dt} &= f\sigma - \upsilon Q - \mu Q \\
\frac{dS}{dt} &= \upsilon Q + (1-f)\sigma - \theta S(1-k_2) + \delta R - \mu S - \theta SI \\
\frac{dL}{dt} &= \theta SI - \beta L - \mu L - \eta(1+k_1)L \\
\frac{dI}{dt} &= \beta L + \theta S(1-k_2) - ((\mu+\gamma) + \alpha(1+k_2))I \\
\frac{dR}{dt} &= \omega T - \mu R - \delta R \\
\frac{dT}{dt} &= \eta(1+k_1)L + \alpha(1+k_2)I - \mu T - \omega T.
\end{aligned}\right\} \quad (1)$$

We will use the bifurcation theory states that perturbation in the parameter of a model leads to a change in the behavior of the equilibrium solution (Garba et al., 2013). In the model, we use the center manifold method to assess the direction of bifurcation (i.e., either forward or backward). The method reduces the system to a smaller system that has the same qualitative properties and can be studied in a relatively easier way. This leads to a result of endemic equilibrium and backward bifurcation for our model.

Besides, the theory of epidemiology signifies the phenomenon of backward bifurcation, which is the classical requirement for the model's effective reproduction number $R_e < 1$. Although this is necessary, it is no longer sufficient to conclude the effective control or elimination of gonorrhoea in a population. Therefore, in this model, we consider the nature of the equilibrium solution near the bifurcation point $R_e = 1$ in the neighborhood of the disease-free equilibrium (E_0). The disease-free equilibrium is locally asymptotically stable if $R_e < 1$ and unstable if $R_e > 1$. But when $R_e = 1$, another equilibrium point bifurcates from the disease-free equilibrium. In this case, the disease would invade the population in the case of backward bifurcation.

3. Results and discussion

We first observe that setting the right-hand side of the system (1) to zero gives the disease-Free Equilibrium (DFE) of the model as the equilibria. Suppose, however that

$$L \neq 0, I \neq 0, R \neq 0 \text{ and } T \neq 0$$

then the model attains endemic equilibrium and solving the endemic equilibria system of the model gives the endemic state to be:

$$\begin{aligned}
Q^* &= \frac{f\sigma}{\mu+\upsilon}; \\
S^* &= \frac{(\mu+\delta)(\mu+\omega)f\sigma + (\mu+\upsilon)(\mu+\delta)(\mu+\omega)\sigma(1-f) + (\mu+\upsilon)\delta\omega(\alpha+\eta)}{(\mu+\upsilon)(\mu+\delta)(\mu+\omega)}; \\
L^* &= \frac{(\lambda)(\mu+\delta)(\mu+\omega)f\sigma + (\mu+\upsilon)(\mu+\delta)(\mu+\omega)\sigma(1-f) + (\mu+\upsilon)\delta\omega(\alpha+\eta)}{(\mu+\beta+\eta)(\mu+\upsilon)(\mu+\delta)(\mu+\omega)}; \\
I^* &= \frac{(\mu+\delta)(\mu+\omega)f\sigma + (\mu+\upsilon)(\mu+\delta)(\mu+\omega)\sigma(1-f) + (\mu+\upsilon)\delta\omega(\alpha+\eta)(\beta\lambda + (\mu+\beta+\eta)\theta)}{(\mu+\alpha+\gamma)(\mu+\beta+\eta)(\mu+\upsilon)(\mu+\delta)(\mu+\omega)}; \\
R^* &= \frac{\omega(\alpha+\eta)}{(\mu+\delta)(\mu+\omega)}; \\
T^* &= \frac{\alpha+\eta}{\mu+\omega}.
\end{aligned}$$

Lemma 3.1: A qualitative change in the behavior of the equilibria due to perturbation results in bifurcation.

Proof: For $\mu_0, \mu_1 > 0$, it follows that the model is stable and that at a steady state,

$$\frac{dQ}{dt} = 0,\ \frac{dS}{dt} = 0,\ \frac{dL}{dt} = 0,\ \frac{dI}{dt} = 0,\ \frac{dR}{dt} = 0$$

and,

$$\frac{dT}{dt} = 0.$$

Thus,

$$\begin{aligned}
\frac{dQ}{dt} &= f\sigma - \mu_2 Q \\
\frac{dS}{dt} &= (1-f)\sigma + \upsilon Q + \delta R - \theta S(1-k_2) - \mu S - \theta IS \\
\frac{dL}{dt} &= \theta IS - \mu_1 L \quad (2) \\
\frac{dI}{dt} &= \beta L + \theta S(1-k_2) - \mu_0 \quad (3) \\
\frac{dR}{dt} &= \omega T - \mu_2 R \\
\frac{dT}{dt} &= \eta(1+k_1)L + \alpha(1+k_2)I - \mu_3 T.
\end{aligned}$$

So letting,

$$\begin{aligned}
\mu_0 &= \mu + \alpha + \gamma \\
\mu_1 &= \mu + \beta + \eta \\
\mu_2 &= \mu + \upsilon \\
\mu_3 &= \mu + \delta.
\end{aligned}$$

At a steady state, the equilibrium points of Eq. 2 become:

$$0 = \theta IS - \mu_1 L \Rightarrow L = \frac{\theta IS}{\mu_1} \Rightarrow L = (0, \frac{\theta IS}{\mu_1}).$$

While the equilibrium points of Eq. 3 become:

$$0 = \beta L + \theta S(1-k_2) - \mu_0 I \Rightarrow I = \frac{\beta L + \theta S(1-k_2)}{\mu_0} \Rightarrow I = (0, \frac{\beta L + \theta S(1-k_2)}{\mu_0}).$$

This result is consistent with those of perturbed systems in Fu et al (2015). We have the next result.

Proposition 3.2: The disease dynamics are controllable in the population with a sufficient perturbation for a sufficiently long time.

Proof: As shown above, the introduction of the treatment (or control) parameter changes the initial stage of the infection, hence, transcritical bifurcation. Now adding small perturbations to the equilibrium points of the model subject to changes in control or bifurcation parameter, we have:

$$L = 0 + \varepsilon L = \frac{\theta IS}{\mu_1} + \varepsilon L,$$

and,

$$I = 0 + \varepsilon I = \frac{\beta L + \theta S(1-k_2)}{\mu_0} + \varepsilon I.$$

Similarly:

$$\frac{dL}{dt} = \theta IS - \mu_1 L = \theta IS - \mu_1\left(\frac{\theta IS}{\mu_1} + \varepsilon L\right) = -\mu_1 \varepsilon L.$$

Solving this gives,

$$L(t) = Be^{-\mu_1 \varepsilon t} \tag{4}$$

where B is an arbitrary constant. Clearly,$|L| \to 0$ as $|t| \to \infty$.

Observe that Eq. 4 indicates that there is stability for all $\mu_1 > 0$. This means that the infection can be controlled in the population. Moreover:

$$\frac{dI}{dt} = \beta L + \theta S(1-k_2) - \mu_0 I = \beta L + \theta S(1-k_2) - \mu_0 \varepsilon I.$$

Solving this gives

$$I(t) = Ae^{-\mu_0 \varepsilon t} \tag{5}$$

for an arbitrary constant A. So, there is linear stability for all $\mu_0 > 0$. Moreover, $|I| \to 0$ as $|t| \to \infty$. In the addition of treatment or control parameters, we have the bifurcation shown graphically in Fig. 2.

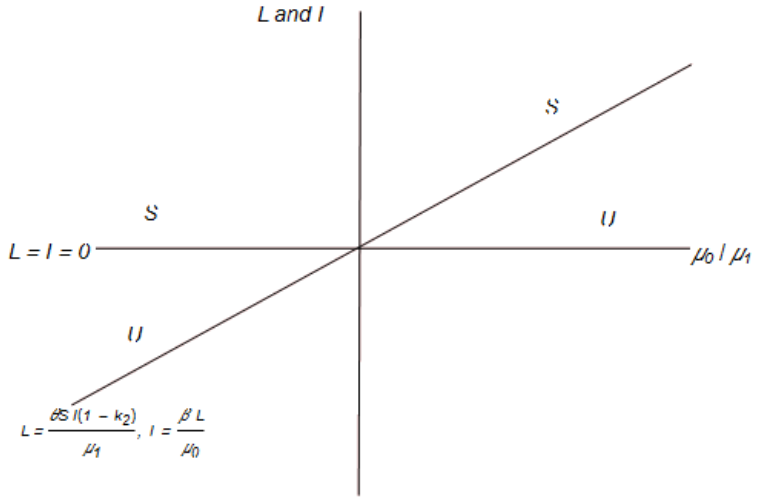

Fig. 2: The transcritical bifurcation of the gonorrhoea model with passive immunity

When one considers the basic reproduction number R_0, which is the expected number of secondary infections produced in a completely susceptible population by a typical or one infected individual (Van den Driessche and Watmough, 2002), other results of this analysis follow. The basic reproduction number is an important parameter used to determine how long an infectious disease can last or prevail in a given population. When $R_0 < 1$, it means that with time the disease will die out of the population thereby giving it a clean health bill (Garba et al., 2013). But if $R_0 > 1$, it is expected that the disease will persist in the population. So for the disease to die out of the population, the associated reproduction number must be less than 1 (Hethcote and Yorke, 1984). When a control measure is given to a model, the reproduction number of the infectious disease becomes an effective reproduction number R_e (Hook and Handsfield, 2008).

Proposition 3.3: The controls in the model system (1) for the gonorrhoea dynamics extinct the pandemics from the population.

Proof: For the infectious classes are L, I, and T, let:

$$f_i = \begin{bmatrix} \theta IS \\ \theta(1-k_2)S \\ 0 \end{bmatrix}.$$

so that,

$$\frac{\partial f_i}{\partial x_j} E_0 = F = \begin{pmatrix} 0 & \theta S & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix}.$$

also,

$$v_i = \begin{bmatrix} \beta L + \mu L + \eta(1+k_1)L \\ \mu I + \gamma I + \alpha(1+k_2)I - \beta L - \theta S(1-k_2) \\ \mu T + \omega T - \eta(1+k_1)L - \alpha(1+k_2)I \end{bmatrix}.$$

so that:

$$\frac{\partial v_i}{\partial x_j}E_0 = V = \begin{pmatrix} (\beta+\mu+\eta(1+k_1)) & 0 & 0 \\ -\beta & (\mu+\gamma+\alpha(1+k_2)) & 0 \\ -\eta(1+k_1) & -\alpha(1+k_2) & (\mu+\omega) \end{pmatrix}$$

The matrix formed by the co-factors of the determinant is:

$$\begin{pmatrix} (\mu+\gamma+\alpha(1+k_2))(\mu+\omega) & -\beta(\mu+\omega) & \alpha\beta(1+k_2)+\eta(1+k_1)(\mu+\gamma+\alpha(1+k_2)) \\ 0 & (\beta+\mu+\eta(1+k_1))(\mu+\omega) & -\alpha(1+k_2)(\beta+\mu+\eta(1+k_1)) \\ 0 & 0 & (\beta+\mu+\eta(1+k_1)(\mu+\gamma+\alpha(1+k_2)) \end{pmatrix}$$

so that,

$$V^{-1} = \begin{pmatrix} \frac{1}{\beta+\mu+\eta(1+k_1)} & 0 & 0 \\ \frac{\beta}{(\beta+\mu+\eta(1+k_1))(\mu+\alpha(1+k_2)+\gamma)} & \frac{1}{\mu+\gamma+\alpha(1+k_2)} & 0 \\ \frac{\alpha\beta(1+k_2)+\eta(1+k_1)}{(\beta+\mu+\eta(1+k_1)(\mu+\omega)} & \frac{-\alpha(1+k_2)}{\mu+\gamma+\alpha(1+k_2)(\mu+\omega)} & \frac{1}{\mu+\omega} \end{pmatrix}$$

also,

$$|FV^{-1}-\lambda I| = \begin{vmatrix} \frac{\beta\theta S}{(\beta+\mu+\eta(1+k_1))(\mu+\gamma+\alpha(1+k_2))}-\lambda & \frac{\theta S(1-k_2)}{\mu+\gamma+\alpha(1+k_2)} & 0 \\ 0 & 0-\lambda & 0 \\ 0 & 0 & 0-\lambda \end{vmatrix} = 0.$$

Hence,

$$\lambda^2\left(\frac{(\beta\theta S)}{(\beta+\mu+\eta(1+k_1))(\mu+\gamma+\alpha(1+k_2))}-\lambda\right) = 0.$$

thus, either,

$$\lambda^2 = 0 \text{ or } \lambda = \frac{(\beta\theta S)}{(\beta+\mu+\eta(1+k_1))(\mu+\gamma+\alpha(1+k_2))}.$$

therefore, the effective reproduction number,

$$R_e = \frac{(\beta\theta S)}{(\beta+\mu+\eta(1+k_1))(\mu+\gamma+\alpha(1+k_2))}. \quad (6)$$

To illustrate this, let our variables and parameters be as in Table 1.

Table 1: Parameters/variables and values

Parameter/Variable	β	θ	μ	η	γ	α	δ	υ	ω	σ
Value	0.01	0.5	0.2	0.1	0.01	0.2	0.8	0.4	0.7	0.4
Parameter/Variable	$d_1 = k_1$	$d_2 = k_2$	f	S	Q	R	T	L	I	
Value	0.5	0.8	0.91	2000	1000	500	1000	1000	500	

Then,

$$R_e = \frac{\sigma\beta\theta((\mu+\upsilon)-\mu f)}{\mu(\mu+\alpha+\gamma)(\mu+\beta+\eta)(\mu+\upsilon)} = 0.09700176367 < 1. \quad (7)$$

We have the following main result:

Theorem 3.4: The gonorrhoea model undergoes backward bifurcation at $R_e = 1$ whenever the bifurcation co-efficienta and b are positive.

Proof: From Table 1, we have that the effective reproduction number R_e of the gonorrhoea infection:

$$R_e = \frac{S\beta\theta}{\mu+\gamma+\alpha(1+k_2)(\mu+\beta+\eta(1+k_1)} \quad (8)$$

or

$$R_e = \frac{\sigma\beta\theta((\mu+\upsilon)-\mu f)}{\mu(\mu+\alpha+\gamma)(\mu+\beta+\eta)(\mu+\upsilon)} = 0.09700176367 < 1. \quad (9)$$

let $\psi = \theta s$ be the parameter by which the bifurcation occurs at $R_e = 1$.

Eq. 8 becomes:

$$1 = \frac{\psi\beta}{\mu+\gamma+\alpha(1+k_2)\mu+\beta+\eta(1+k_1)}$$
$$\psi = \frac{\mu+\gamma+\alpha(1+k_2)}{\beta};\ \beta \neq 0.$$

let $x_1 = Q, x_2 = S, x_3 = L, x_4 = I, x_5 = R$, and $x_6 = T$. Furthermore, by using the vector notation,

$$X = (x_1, x_2, x_3, x_4, x_5, x_6)^T$$

the model can be written in the form,

$$\frac{dx}{dt} = (f_1, f_2, f_3, f_4, f_5, f_6)^T$$

then the model Eq. 1 becomes,

$$\begin{aligned} f_1 &= f\sigma - (\mu+\upsilon)x_1 \\ f_2 &= (1-f)\sigma + \upsilon x_1 + \delta x_5 - \psi(1-k_2) - \psi x_4 - \mu x_2 \\ f_3 &= \psi x_4 - \mu x_3 - \beta x_3 - \eta(1+k_1)x_3 \\ f_4 &= \beta x_3 + \psi(1-k_2) - \mu x_4 - \alpha(1+k_2)x_4 - \gamma x_4 \\ f_5 &= \eta(1+k_1)x_3 + \alpha(1+k_2)x_4 - \mu x_5 \omega x_5 \\ f_6 &= \omega x_5 - \mu x_6 - \delta x_5. \end{aligned}$$

here $\mu_2 = \mu+\upsilon, \mu_3 = \mu+\delta$ and $\mu_4 = \mu+\omega$. The Jacobian matrix at DFE is therefore given by,

$$J = \begin{pmatrix} -(\mu+\upsilon) & 0 & 0 & 0 & 0 & 0 \\ \upsilon & -\mu & 0 & -\psi & 0 & \delta \\ 0 & 0 & -(\mu+\beta+\eta(1+k_1)) & \psi & 0 & 0 \\ 0 & 0 & \beta & -(\mu+\gamma+\alpha(1+k_2) & 0 & 0 \\ 0 & 0 & \eta(1+k_1) & \alpha(1+k_2) & -(\mu+\omega) & 0 \\ 0 & 0 & 0 & 0 & \omega & -(\mu+\delta) \end{pmatrix}$$

The Jacobian of the linearised system has simple zero eigenvalues, with all other eigenvalues having negative real parts, hence the center manifold theory can be used to analyze the dynamics of the system around the bifurcation point ψ (Shaban and Mofi, 2014; Usman and Adamu, 2017). The Jacobian matrix has a right eigenvector (corresponding to the zero eigenvalues) given by,

$h = (h_1, h_2, h_3, h_4, h_5, h_6) - (\mu+\upsilon)h_1 = 0 \Rightarrow h_1 = 0$
$\upsilon h_1 - \mu h_2 - \psi h_4 + \delta h_6 = 0 \Rightarrow h_2 = \frac{\delta h_6 - \psi h_4}{\mu}$
$h_3 = \frac{\psi h_4}{\mu+\beta+\alpha(1+k_2)}$
$\beta h_3 - (\mu+\gamma+\alpha(1+k_2)h_4 = 0 \Rightarrow h_4 = \frac{\beta h_3}{\mu+\gamma+\alpha(1+k_2)}$
$h_5 = \frac{\eta(1+k_1)h_3+\alpha(1+k_2)h_4}{\mu+\omega}$
$\omega h_5 - (\mu+\delta)h_6 0 \Rightarrow h_6 = \frac{\omega}{\mu+\delta}$.

Similarly, the left eigenvectors (corresponding to the zero eigenvalues) are given by

$v = (v_1, v_2, v_3, v_4, v_5, v_6)$

where,

$-\mu v_2 = 0 \Rightarrow v_2 = 0$
$-(\mu+\upsilon)v_1 + \upsilon v_2 = 0 \Rightarrow v_1 = 0$
$\delta v_2 - (\mu+\delta)v_6 = 0 \Rightarrow v_6 = 0$
$-(\mu+\omega)v_5 = 0 \Rightarrow v_5 = 0$
$-(\mu+\beta+\alpha(1+k_2)v_3 + \beta v_4 + \eta(1+k_1)v_5 = 0$
$\Rightarrow v_3 = \frac{\beta v_4}{\mu+\beta+\alpha(1+k_2)}$
$-\psi v_2 + \psi v_3 - (\mu+\gamma+\alpha(1+k_2))v_4 + \alpha(1+k_2))v_5 = 0 \Rightarrow v_4 = \frac{\psi v_3}{\mu+\gamma+\alpha(1+k_2)}$.

so that $v \cdot h = 1$ is in line with Garba et al. (2013). We are now left to consider f_k; $k = 3,4$ since $v_1 = v_2 = v_5 = v_6 = 0$.

The local dynamic of the system is totally governed by the signs of a and. For instance, if $a = 0$, and $b > 0$ when $\psi < 0$, then, 0 is locally asymptotically stable and there exists a positive stable equilibrium (Nana-Kyere et al., 2016). Hence, by computing the non-zero partial derivatives of the right-hand function $f_i, i = 1, 2, \cdots, 6$ the associated backward bifurcation coefficients a and b are given respectively by

$a = \sum_{i=j=k=1}^{n} v_k h_i h_j \frac{\partial^2 f_k}{\partial x_i x_j}(0,0)$.

So,

$\frac{\partial^2 f_3}{\partial x_3 \partial x_4} = 0$ and $\frac{\partial^2 f_4}{\partial x_3 \partial x_4} = 0$.

This implies

$v_3 h_4 \frac{\partial^2 f_3}{\partial x_3 \partial x_4} + v_4 h_3 \frac{\partial^2 f_4}{\partial x_3 \partial x_4} = 0 \Rightarrow a = 0$.

and

$b = \sum_{i=j=k=1}^{n} v_k h_i \frac{\partial^2 f_k}{\partial x_i x_\psi}(0,0)$.

with

$\frac{\partial^2 f_3}{\partial x_3 \partial \psi} = 1$ and $\frac{\partial^2 f_4}{\partial x_4 \partial \psi} = 0$.

so,

$v_3 h_3 \frac{\partial^2 f_3}{\partial x_3 \partial \psi} + v_4 h_4 \frac{\partial^2 f_4}{\partial x_4 \partial \psi} = 1 + 0 = 1 > 0 \Rightarrow b > 0$.

Since the backward bifurcation co-efficient b is positive, it follows that the gonorrhoea model will undergo backward bifurcation. This means that there is Endemic Equilibrium when $R_e > 1$, and when $R_e = 1$. But from equations of R_0 and R_e, they are both less than 1, showing that the disease will be controlled in the population in a limited time.

Graphical simulations buttress our results. These are shown in Fig. 3 and Fig. 4.

Fig. 3 suggests that when the waning rate υ is low (i.e., $\upsilon = 0.2$), the passive immune population decreases exponentially with time, while Fig. 4 indicates that as the waning rate is high, (i.e., $\upsilon = 0.6$), the passive immune population decreases faster and varnishes with time. The continuous decay in the population of the immune class (Q) with time is due to the fact that the immunity conferred on the individuals in this class is temporal and hence, expires with time.

However, the susceptible population increases slower to the turning point at about one year and three months as the waning rate υ is low and increases faster as the waning rate υ is high as shown in Figs. 3 and 4 respectively. In both cases, the susceptible class later decreases with time due to the interaction among the latent, infected, and susceptible classes coupled with the natural mortality rate μ. The impact of contact rate on susceptible, latent, and infected classes is shown in Fig. 5.

Fig 5 indicates that when the interaction rate is low (i.e., $\theta = 0.3$), the latent and the infected classes decrease exponentially with time, and even varnishes in the long run since there will be almost nobody to contact and suffer the disease. It is also shown that when the interaction rate $\theta = 0$, the reproduction number of the disease becomes zero. That is,

$R_0 = \frac{(\beta\theta S)}{(\beta+\mu+\eta)(\mu+\gamma+\alpha)} = 0$.

Thus, at this point, the contact rate λ becomes zero and hence, nobody suffers the disease.

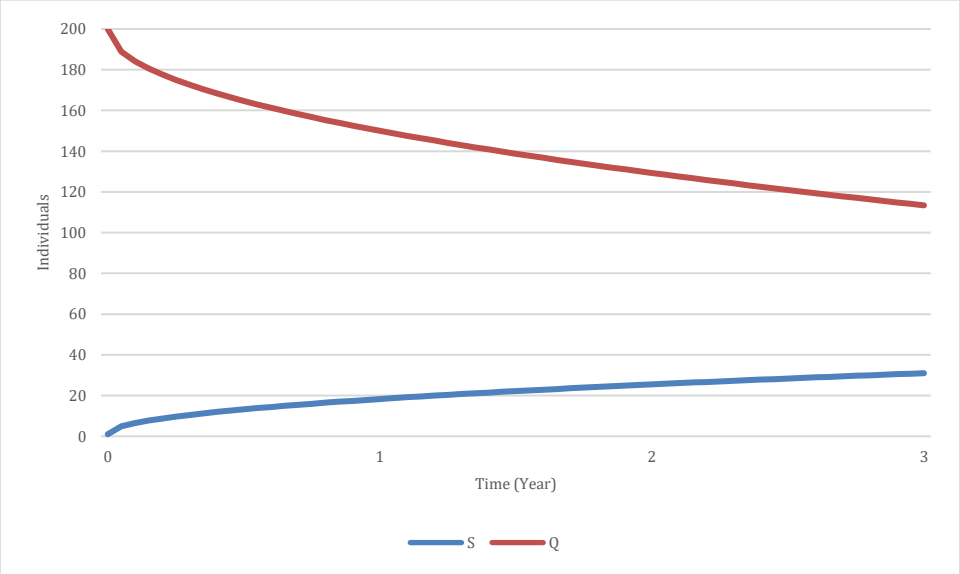

Fig. 3: Effect of decreasing waning rate on the susceptible and immune classes, i.e. $\upsilon = 0.2$

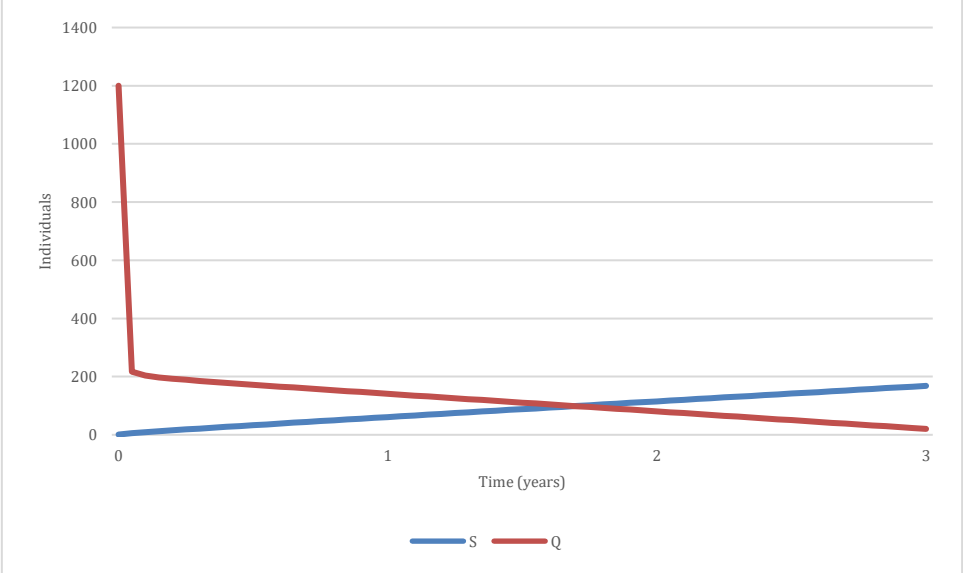

Fig. 4: Effect of increasing waning rate on the susceptible and immune classes, i.e., $\upsilon = 0.6$

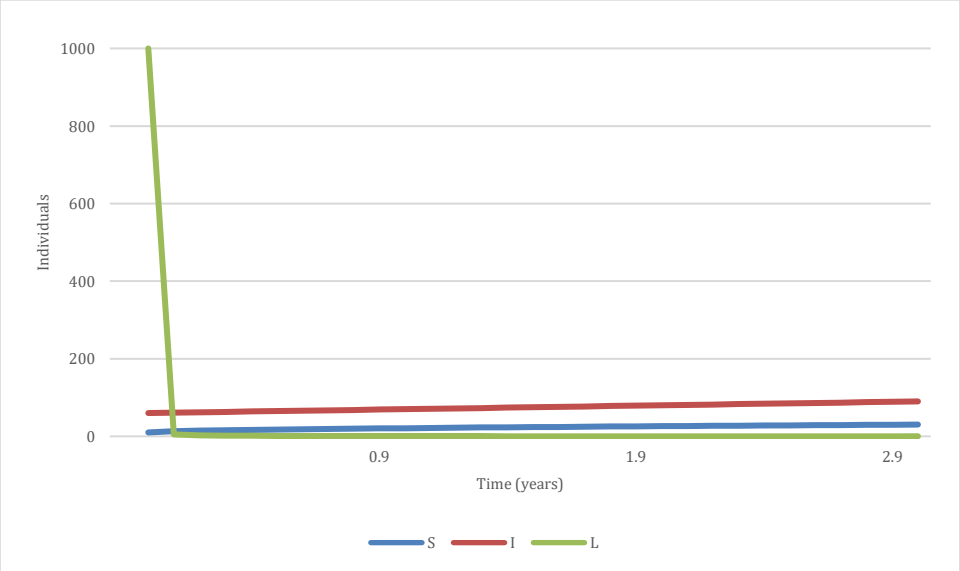

Fig. 5: The effect of reducing contact rate $\lambda = \theta I$

4. Conclusion

Based on the analysis and results of this work, we observed that the disease would be eradicated from the population since the effective reproduction number is less than 1. Again, the addition of treatment or control measures such as condoms and education enlightenment helped to reduce the infection in the population. However, the addition of control parameters led to transcritical bifurcation.

From the graphical illustrations, we concluded that the immune population continues to decay exponentially due to temporal immunity conferred on the individuals in the immune class. We also concluded that the reproduction number of the infection grows when there is no control measure in the model and decays when the control measure is applied in the model. Finally, we concluded that for the disease to be totally eliminated from the community, the interaction rate θ with the infective which leads to contact should be totally reduced to the barest minimum or zero.

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Compliance with ethical standards

Conflict of interest

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

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