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 introduction of treatment or control parameters leads to transcritical bifurcation. The backward bifurcation coefficients were calculated and their numerical perturbation results to 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.

Keywords: Gonorrhoea dynamics and control; passive immunity; reproduction number; stability; bifurcation; equilibria

1 Introduction

Due to increasing rate of infertility among the teeming 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 [6]. Gonorrhoea is one of such sexually transmitted infectious diseases caused by a bacterium called Neisseria gonorrhoeae [21]. The neisseria gonorrhoea is characterized by a very short period of latency, namely, 2 – 10 days [11] and is commonly found in the glummer epithelium such as the urethra and endo-cervix epithelia of the reproductive track [5]. Gonorrhoea is transmitted to a new born infant from the infected mother through the birth canal thereby causing inflammations and eye infection such as conjunctivitis. It is also spread through unprotected sexual intercourse, [20].

Studies by Usman and Adam [22] and Center for Disease Control Report in show that male patients of gonorrhoea have pains in the testicles (known as epididymitis), painful urination due to scaring inside the urethra while in female patients, the disease may ascend the genital tract and block the fallopean tube leading to pelvic inflammatory disease (PID) and infertility,
see also [15]. Other complications associated with this epidemic include arthritis, endocarditis, chronic pelvic pain, meningitis and ectopic pregnancy, [16].

Gonorrhoea confers temporal immunity on some individuals in the susceptible class while some others are not immuned, [20]. This immunity through the immune system plays an important role in protecting the body against the infection and other foreign substances, [3]. That is why an immuno-compromised patient has a reduced ability to fight infectious disease such as gonorrhoea due to certain diseases and genetic disorder, [18]. Such patient may be particularly vulnerable to opportunistic infection such as gonorrhoea. Hence, immune reaction can be stimulated by drug-induced immune system such as Thrombocytopenia, [18]. This helps to reduce the waning rate of passive immunity in the immune class, [2]. However, if the activity of immune system is excessive or over-reactive due to lack of cell mediated immunity, a hypersensitive reaction develops such as auto-immunity and allergy which may be injurious to body or may even cause death [25].

Statistically, gonorrhoea infection has spread worldwide with more than 360 million new cases witnessed globally in adults aged 15 − 49 years, [3]. In 1999, above over 120 million people in African countries were reported to have contracted the disease. While over 82 million people were reported in Nigeria, [3]. Researches abound on the modelling and control of this epidemic with various approaches and controls, see e.g. [3, 9, 10, 17, 20, 21] and mostly recently [1, 24, 14] and [4]. 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 analyse the stability of the system. This underscores the role of mathematical analysis of models to elicit desired results, see e.g. [12] and [13]. Education and enlightenment, use of condom and treatment of patients with ampicilin 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 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 \( \sigma \) as level of recruitment, \( \nu \) as waning rate of immunity, \( \mu \) as rate of natural mortality, \( \lambda \) as contact rate between the susceptible and the latent classes, \( \eta \) as treatment rate of latent class, \( \gamma \) as induced death rate due to the infection, \( \alpha \) as treatment rate of infected compartment, \( \beta \) as infectious rate of Latent class, \( \omega \) as recovery rate of treated class, \( \delta \) as rate at which recovered class become susceptible again, \( \theta \) as infectious rate from the susceptible class direct to the infectious class, \( k_1 \) as control measure given to latent class as \( k_2 \) as control measure given to 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 new birth are immunized against the infection; the immunity conferred on the new birth wanes after sometime, and that the rate of contact of the disease due to interaction \( \lambda \) 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 as figure (1). So, the model for the gonorrhoea

![Flow diagram of the model](image)

transmission dynamics is given by the following deterministic systems of non-linear differential equations (2.1):

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

We will use that bifurcation theory states that perturbation in the parameter of a model leads to a change in the behaviour of the equilibrium solution, [5]. 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 which has the same qualitative properties and can be studied in a relatively easier way, [2]. This leads to a result on endemic equilibrium and backward bifurcation for our model.

Besides, the theory of epidemiology signifies the phenomenon of backward bifurcation, that is the classical requirement 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, see e.g. [25]. Therefore, in this model we consider the nature of the equilibrium solution near the bifurcation point \( R_e = 1 \) in the neighbourhood 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, [6].

3 Results

We first observe that setting the right hand side of the system \((2.1)\) to zero gives the disease-Free Equilibrium (DFE) of the model as the equilibria:

\[
(Q^0, S^0, L^0, I^0, R^0, T^0) = \frac{f \sigma}{\mu + v}, \frac{vf \sigma + (\mu + v)(1 - f)\sigma}{(\mu + v)(\theta + \mu)}.
\]

Now suppose

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

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

\[
Q^* = \frac{f \sigma}{\mu + v}; \\
S^* = \frac{(\mu + \delta)(\mu + \omega)f \sigma + (\mu + v)(\mu + \delta)(\mu + \omega)\sigma(1 - f) + (\mu + v)\delta \omega(\alpha + \eta)}{(\mu + v)(\mu + \delta)(\mu + \omega)}; \\
L^* = \frac{\lambda(\mu + \delta)(\mu + \omega)f \sigma + (\mu + v)(\mu + \delta)(\mu + \omega)\sigma(1 - f) + (\mu + v)\delta \omega(\alpha + \eta)}{(\mu + \beta + \eta)(\mu + v)(\mu + \delta)(\mu + \omega)}; \\
I^* = \frac{(\mu + \delta)(\mu + \omega)f \sigma + (\mu + v)(\mu + \delta)(\mu + \omega)\sigma(1 - f) + (\mu + v)\delta \omega(\alpha + \eta)(\beta \lambda + (\mu + \beta + \eta)\theta)}{(\mu + \alpha + \gamma)(\mu + \beta + \eta)(\mu + v)(\mu + \delta)(\mu + \omega)}; \\
R^* = \frac{\omega(\alpha + \eta)}{(\mu + \delta)(\mu + \omega)}; \\
T^* = \frac{\alpha + \eta}{\mu + \omega}.
\]

Lemma 3.1. A qualitative change in the behaviour 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 steady state:

\[
\frac{dQ}{dt} = 0, \quad \frac{dS}{dt} = 0, \quad \frac{dL}{dt} = 0, \quad \frac{dI}{dt} = 0, \quad \frac{dR}{dt} = 0 \text{ and } \frac{dT}{dt} = 0.
\]
Thus,

\[
\frac{dQ}{dt} = f\sigma - \mu_2 Q \\
\frac{dS}{dt} = (1 - f)\sigma + vQ + \delta R - \theta S(1 - k_2) - \mu S - \theta IS \\
\frac{dL}{dt} = \theta IS - \mu_1 L \\
\frac{dI}{dt} = \beta L + \theta S(1 - k_2) - \mu_0 I \\
\frac{dR}{dt} = \omega T - \mu_2 R \\
\frac{dT}{dt} = \eta(1 + k_1)L + \alpha(1 + k_2)I - \mu_3 T.
\] (3.1)

So letting

\[
\mu_0 = \mu + \alpha + \gamma, \\
\mu_1 = \mu + \beta + \eta, \\
\mu_2 = \mu + v, \\
\mu_3 = \mu + \delta.
\]

At steady state, the equilibrium points of (3.1) 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 equation (3.2) 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 [9] and [6].

We have the next result.

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

**Proof.** As shown above, the introduction of 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
\[
L(t) = B e^{-\mu_1 \varepsilon t} \tag{3.3}
\]
where \( B \) is an arbitrary constant. Clearly, \(|L| \to 0\) as \(|t| \to \infty\).

Observe that equation \([3.3]\) 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
\[
I(t) = A e^{-\mu_0 \varepsilon t} \tag{3.4}
\]
for an arbitrary constant \( A \). So, there is linear stability for all \( \mu_0 > 0 \). Moreover,
\[
|I| \to 0 \text{ as } |t| \to \infty.
\]

On the addition of treatment or control parameters, we have the bifurcation shown in graphically in Figure 2.

![Figure 2: The Transcritical Bifurcation of the gonorrhoea model with passive immunity.](image)

When one considers the basic reproduction number \( R_0 \), which is the expected number of secondary infection produced in a completely susceptible population by a typical or one infected individual [23], 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 \[5\]. 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 \[7\]. When control measure is given to a model, the reproduction number of the infectious disease becomes effective reproduction number \( R_e \), \[8\].

**Proposition 3.3.** The controls in the model system \( (2.1) \) 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

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

Also,

\[
|FV^{-1} - \lambda I| = \begin{vmatrix} \frac{\theta S}{(\beta + \mu + \eta(1+k_1))(\mu + \gamma + \alpha(1+k_2))} - \lambda & \frac{\theta S(1-k_2)}{\mu + \gamma + \alpha(1+k_2)} & 0 \\ 0 & 0 - \lambda & 0 \\ 0 & 0 & 0 - \lambda \end{vmatrix} = 0.
\]
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
\[ \lambda^2 = 0 \quad \text{or} \quad \lambda = \frac{(\beta \theta S)}{(\beta + \mu + \eta(1 + k_1))(\mu + \gamma + \alpha(1 + k_2))}. \]

Therefore, the effective reproduction number
\[ R_e = \frac{(\beta \theta S)}{(\beta + \mu + \eta(1 + k_1))(\mu + \gamma + \alpha(1 + k_2))}. \quad (3.5) \]

To illustrate this, let our variables and parameters be as in Table 3: then,

| Parameter/Variable | \( \beta \) | \( \theta \) | \( \mu \) | \( \eta \) | \( \gamma \) | \( \alpha \) | \( \delta \) | \( v \) | \( \omega \) | \( \sigma \) |
|-------------------|----------|----------|----------|----------|----------|----------|--------|--------|--------|--------|
| 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    |

Table 1: Parameters/variables and values.

\[ R_e = \frac{\sigma \beta \theta((\mu + \nu) - \mu f)}{\mu(\mu + \alpha + \gamma)(\mu + \beta + \eta)\mu + \nu} = 0.09700176367 < 1. \quad (3.6) \]

We have the following main result.

**Theorem 3.4.** The gonorrhoea model undergoes backward bifurcation at \( R_e = 1 \) whenever the bifurcation co-efficient \( a \) and \( b \) are positive.

**Proof.** Now, recall the effective reproduction number \( R_e \) of the gonorrhoea infection as shown by equation (3.7)

\[ R_e = \frac{S \beta \theta}{\mu(\mu + \alpha + \gamma)(\mu + \beta + \eta)\mu + \nu} \quad (3.7) \]

Or

\[ R_e = \frac{\sigma \beta \theta((\mu + \nu) - \mu f)}{\mu(\mu + \alpha + \gamma)(\mu + \beta + \eta)\mu + \nu} = 0.09700176367 < 1. \quad (3.8) \]

Let \( \psi = \theta s \) be the parameter by which the bifurcation occurs at \( R_e = 1 \). Equation (3.7) becomes

\[ 1 = \frac{\psi \beta}{\mu + \gamma + \alpha(1 + k_2)\mu + \beta + \eta(1 + k_1)} \]

\[ \psi = \frac{\mu + \gamma + \alpha(1 + k_2)}{\beta}; \quad \beta \neq 0. \]
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 equations (2.1) become
\[
\begin{align*}
  f_1 &= f_\sigma - (\mu + \nu)x_1 \\
  f_2 &= (1 - f_\sigma + \nu x_1 + \delta x_5 - \psi(1 - k_2) - \psi x_4 - \mu x_2 \\
  f_3 &= \psi x_4 - \mu x_3 - \beta x_3 - \eta(1 + k_1) x_3 \\
  f_4 &= \beta x_3 + \psi(1 - k_2) - \mu x_4 - \alpha(1 + k_2)x_4 - \gamma x_4 \\
  f_5 &= \eta(1 + k_1)x_3 + \alpha(1 + k_2)x_4 - \mu x_5 \omega x_5 \\
  f_6 &= \omega x_5 - \mu x_6 - \delta x_5.
\end{align*}
\]
Here \( \mu_2 = \mu + \nu, \mu_3 = \mu + \delta \) and \( \mu_4 = \mu + \omega. \)

The Jacobian matrix at DFE is therefore given by
\[
J = \begin{pmatrix}
-\mu - v & 0 & 0 & 0 & 0 & 0 \\
0 & -\mu - v & \psi & 0 & 0 & \delta \\
0 & 0 & -\mu - \beta + \eta(1 + k_1) & 0 & \psi & 0 \\
0 & 0 & \beta & -\mu - \gamma + \alpha(1 + k_2) & 0 & 0 \\
0 & 0 & \eta(1 + k_1) & \alpha(1 + k_2) & -\mu - \omega & 0 \\
0 & 0 & 0 & 0 & \omega & -\mu - \delta
\end{pmatrix}
\]
The Jacobian of the linearised system has a simple zero eigenvalues, with all other eigenvalues having negative real parts, hence the center manifold theory can be used to analyse the dynamics of the system around the bifurcation point \( \psi, \) \([19]\) and \([23]\). The Jacobian matrix has a right eigenvectors (corresponding to the zero eigenvalues) given by
\[
h = (h_1, h_2, h_3, h_4, h_5, h_6)
\]
\[
\begin{align*}
  -(\mu + v)h_1 &= 0 \Rightarrow h_1 = 0 \\
  \nu h_1 - \mu h_2 - \psi h_4 + \delta h_6 &= 0 \Rightarrow h_2 = \frac{\delta h_6 - \psi h_4}{\mu} \\
  h_3 &= \frac{\psi h_4}{\mu + \beta + \alpha(1 + k_2)} \\
  \beta h_3 - (\mu + \gamma + \alpha(1 + k_2) h_4 &= 0 \Rightarrow h_4 = \frac{\beta h_3}{\mu + \gamma + \alpha(1 + k_2)} \\
  h_5 &= \frac{\eta(1 + k_1) h_3 + \alpha(1 + k_2) h_4}{\mu + \omega} \\
  \omega h_5 - (\mu + \delta) h_6 &= 0 \Rightarrow h_6 = \frac{\omega}{\mu + \delta}.
\end{align*}
\]
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

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

So that \( v \cdot h = 1 \) in line with [5].

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 \( b \). For instance, if \( a = 0 \), and \( b > 0 \) when \( \psi < 0 \), then, 0 is locally asymptotically stable and there exist a positive stable equilibrium [17]. 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,

\[ \frac{\partial^2 f_3}{\partial x_3 \partial x_4} = 0 \text{ and } \frac{\partial^2 f_4}{\partial x_3 \partial x_4} = 0. \]

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

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

with

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

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.

4 Discussion of Results

Graphical simulation buttress our results. These are the following:

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

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

Figure 3 suggests that when the waning rate $\nu$ is low (i.e., $\nu = 0.2$), the passive immune population decreases exponentially with time, while Figure 4 indicates that as the waning rate is high, (i.e., $\nu = 0.6$), 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 $\nu$ is low and increases faster as the waning rate $\nu$ is high as shown in Figures 3 and 4 respectively. In both cases, the susceptible class later decreases with time due to the interaction among the latent, infected and the susceptible classes coupled with the natural mortality rate $\mu$.

The impact of contact rate on Susceptible, Latent and Infected classes is shown in Figure 5. Figure 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 $\lambda$ becomes zero and hence, nobody suffers the disease.

5 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, addition of treatment or control measures such as condom and education enlightenment helped to reduce the infection in the population. However, addition of control parameters led to transcritical bifurcation.

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

References

[1] Adam II and Sulaiman U (2018). Mathematical Model for the dynamics of Neisseria Gonorrhea disease with Natural immunity and treatment effects. Journal of Mathematics Research 10(2), 2018: 151. https://doi.org/10.5539/jmr.v10n2p151

[2] Echeng BB and Adagba HO (2021). Global Stability Analysis of the Role of Antiretroviral Therapy (ART) Abuse in HIV/AIDS Treatment Dynamics. Pure and Applied Mathematics Journal, 10(1): 9-31. https://doi.org/10.11648/j.pamj.20211001.12
[3] Centers for Disease Control (CDC) (2016). *Antibiotic-Resistant gonorrhoea: Basic Information*. Center for Disease Control (CDC).

[4] Didelot X, Kendall M, Xu Y, White PJ and McCarthy N (2021). *Genomic epidemiology analysis of infectious disease outbreaks using TransPhylo*. Current protocols, 1(2). [https://doi.org/10.1002/cpz1.60](https://doi.org/10.1002/cpz1.60)

[5] Garba SM, Safi MA and Gumel AB (2013). *Cross immunity backward bifurcation for a model of transmission dynamics of two strains of influenza*. Nonlinear analysis B: Real World Applications 1384.

[6] Gregory Faye (2011). *An Introduction to bifurcation theory*. Neuro Mathematical Computer Laboratory, Sophia Antipolis Paris, France.

[7] Hethcoote HW and York JA (1984). *Lecture notes in biomathematics, vol 56: gonorrhoea transmission dynamics and controls*. Springer-Verlag, Heidelberg.

[8] Hook EW and Handsfield HH (2008). *Sexually transmitted disease 4th edition*. Mcraw-Hill Education, New York, 627-45.

[9] Jing F, Qixing H, Yuguo L and Daqing J (2015). *Asymptotic behavior of a multigroup SIS epidemic model with stochastic perturbation*. Advances in Difference Equations, 2015:1-9.

[10] Mushayabasa S, Tchuenche JM, Bhunu CP and Ngarakana-Gwasira E (2011). *Modeling gonorrhoea and HIV co-interaction*. Biosystems, 103: 27-37.

[11] Mushayabasa S and Bhunu CP (2011). *Modelling the effect of heavy alcohol consumption on the transmission dynamics of gonorrhoea*. National University of Science and Technology Zimbabwe.

[12] Omenyi L and Uchenna M (2019). *Global analysis on Riemannian manifolds*. Australian Journal of Mathematical Analysis and Applications, 16(2):1-17. Online: [https://ajmaa.org/searchroot/files/pdf/v16n2/v16i2p11.pdf](https://ajmaa.org/searchroot/files/pdf/v16n2/v16i2p11.pdf)

[13] Omenyi L, Omaba M, and Nwaeze E, et al (2021). *Analysis of Gegenbauer kernel filtration on the hypersphere*. International Journal of Advanced and Applied Sciences, 8(11): 1-9.

[14] Osnes MN, Didelot X, Korne-Elenbaas J, Alfsnes K, Brynildsrud OB, Syversen G, Nilsen J, De Blasio BF, Caugant DA and Eldholm V (2020). *Sudden emergence of a Neisseria gonorrhoeae clade with reduced susceptibility to extended-spectrum cephalosporins, Norway*. Microbial genomics, 6(12). [https://doi.org/10.1099/mgen.0.000480](https://doi.org/10.1099/mgen.0.000480)

[15] Rama Kishore R and Pattabhiramacharyulu NC (2011). *A numerical approach for the spread of gonorrhoea in homosexuals*. ARPN Journal of Engineering and Applied Sciences, 6(6): 1-8.
[16] Riley S, Fraser C, Donnelly CA, Ghani AC, Abu-Raddad LJ, Hedley AJ, Leung GM, Ho LM, Lam TH, Thach TQ, Chau P, Chan KP, Lo SV, Leung PY, Tsang T, Ho W, Lee KH, Lau EM, Ferguson NM, Anderson RM (2003). Transmission dynamics of the etiological agent of SARS in Hong Kong: impact of public health interventions. Science. 2003 Jun 20;300(5627):1961-1966. https://doi:10.1126/science.1086478. Epub2003May23. PMID:12766206.

[17] Sacrifice NK, et al (2016). A qualitative Analysis of Neisseria gonorrhoea Disease with treatment effect. Applied Mathematics, 6(1), 6-15.

[18] Schiffert Health Center (2011). Patient Information: gonorrhea question and answers. Virginia Tech Division of Student Affairs. http://www.healthcenter.vt.edu/assets/docs/gonorrhea.pdf

[19] Shaban N and Hawa M (2014). Modeling the impact of vaccination and screening on the dynamics of human papillomavirus infection. International Journal of Mathematical Analysis, 8(9) 441-454. https://dx.doi.org

[20] Ugwu CS (2015). Mathematical model on gonorrhoea transmission. MSc dissertation Submitted to the Department of mathematics, University of Nigeria, Nsukka.

[21] Unemo M (2015). Current and future antimicrobial treatment of gonorrhoea: the rapidly evolving Neisseria gonorrhoeae continues to challenge. BMC Infectious Diseases, 15(364). https://doi.org/10.1186/s12879-015-1029-2

[22] Usman S and Adam II (2017). Modeling the transmission Dynamics of the monkey-pox virus Infection with treatment interventions. Journal of Applied Mathematics and Physics, 5, 2335-2353.

[23] Van den Driessche P and Watmough J (2002). Reproduction number and sub-threshold endemic equilibria for compartmental model of disease transmission. Mathematical Biosciences, 180, 29-18.

[24] Whittles LK, White PJ and Didelot X (2020). Assessment of the potential of vaccination to combat antibiotic resistance in gonorrhoea: A modeling analysis to determine preferred product characteristics. Clinical infectious diseases : an official publication of the Infectious Diseases Society of America, 71(8), 1912-1919. https://doi.org/10.1093/cid/ciz1241

[25] World Health Organization (2006). Prevention and control of sexually transmitted Infections. Draft global strategy, Report by the Secretariat.(Geneva: WHO). http://www.who.int/reproductivehealth/docs/stis

[26] Workowsk KA and Bolan GA (2015). Sexually transmitted diseases treatment guidelines. MMWR. Recommendations and Reports/CDC.64(RR-03): 1-137. PMID 26042815.