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A SENSITIVITY ANALYSIS OF A GONORRHOEA DYNAMICS AND CONTROL MODEL

LOUIS OMENYI^{1,3,*}, ALOYSIUS EZAKA², HENRY O. ADAGBA², GERALD OZOIGBO¹,
KAFAYAT ELEBUTE¹

¹Department of Mathematics and Statistics, Alex Ekwueme Federal University, Ndufu-Alike, Nigeria

²Department of Industrial Mathematics and Applied Statistics, Ebonyi State University, Abakaliki, Nigeria

³Department of Mathematical Sciences, Loughborough University, Leicestershire, United Kingdom

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Abstract. We formulate and analyse a robust mathematical model of the dynamics of gonorrhoea incorporating passive immunity and control. Our results show that the disease-free and endemic equilibria of the model are both locally and globally asymptotically stable. A sensitivity analysis of the model shows that the dynamics of the model is variable and dependent on waning rate, control parameters and interaction of the latent and infected classes. In particular, the lower the waning rate, the more the exponential decrease in the passive immunity but the susceptible population increases to the equilibrium and wanes asymptotically due to the presence of the control parameters and restricted interaction of the latent and infected classes.

Keywords: gonorrhoea; passive immunity; equilibria; reproduction number; sensitivity analysis; asymptotic stability.

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1. INTRODUCTION

Gonorrhoea, a sexually transmitted disease, is a major cause of infertility and other debilitating ailments among couples. Thus, it becomes necessary to undertake prompt prevention

*Corresponding author

E-mail addresses: omenyi.louis@funai.edu.ng; omenyilo@yahoo.com

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and control activities to tackle the ugly incidence of this sexually transmitted diseases, [6]. It is caused by a bacterium called *Neisseria gonorrhoeae*, [20]. According to Mushayabasa and Bhunu in [11], *neisseria gonorrhoea* is characterized by a very short period of latency, namely, 2 – 10 days and is commonly found in the glummer epithelium such as the urethra and endocervix 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, [19].

Studies by Usman and Adam [21] and Center for Disease Control Report in [3] 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 [14]. Other complications associated with this epidemic include arthritis, endocarditis, chronic pelvic pain, meningitis and ectopic pregnancy, [15].

Gonorrhoea confers temporal immunity on some individuals in the susceptible class while some others are not immuned, [19]. 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, [17]. 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, [17]. 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 hyper-sensitive reaction develops such as auto-immunity and allergy which may be injurious to the human 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. 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, 18, 16, 19, 20] and mostly recently

[1, 24, 13] and [4]. This present study continues the discussion by investigating the dynamics of the disease by carrying out the sensitivity analysis of the effective reproduction number of the model. Besides, the theory of epidemiology signifies the phenomenon of bifurcation at the equilibria. This is a classical requirement for the model's effective reproduction number, R_e . 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 paper, 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 , through sensitivity analysis to determine the most sensitive parameters to target by a way of intervention strategy.

2. MATERIALS AND METHODS

We formulate a modified SIR model by extending an existing one to incorporate passive immunity. 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 over time t . Let the parameters of the model be σ as level of recruitment, ν as waning rate of immunity, μ as rate of natural mortality, λ as contact rate between the susceptible and the latent classes, η as treatment rate of latent class, γ as induced death rate due to the infection, α as treatment rate of infected compartment, β as infectious rate of latent class, ω as recovery rate of treated class, δ as rate at which recovered class becomes susceptible again, θ as infectious rate from the susceptible class direct to the infectious class, k_1 as control measure given to latent class and k_2 as control measure given to infected class.

Next, assume the 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, λ , rate is due to the movement of the infected population. Consequently, the total population at time t is $N(t) = Q(t) + S(t) + L(t) + I(t) + T(t) + R(t)$. The existing schematic model diagram is given as Figure 1 and that of the extended model as 2:

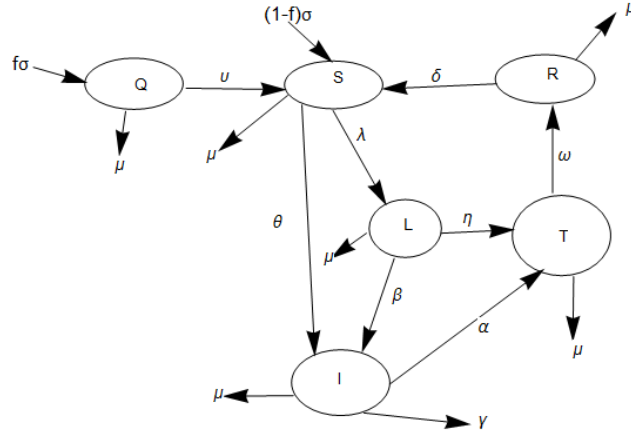


FIGURE 1. The diagram of the model; see [19].

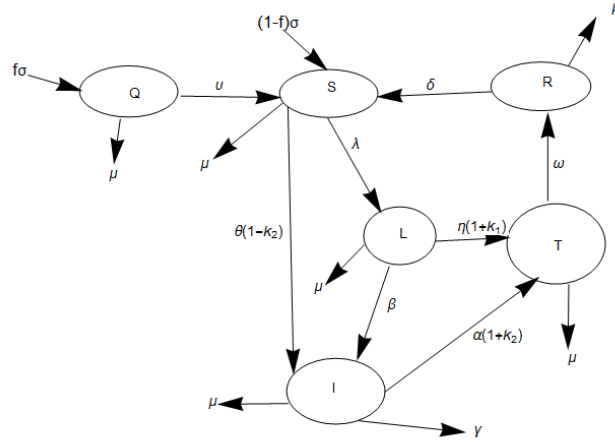


FIGURE 2. The model with control

So, the system of equations for the model without control is (1):

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

and those with control is (2):

$$(2) \quad \left. \begin{aligned} \frac{dQ}{dt} &= f\sigma - vQ - \mu Q \\ \frac{dS}{dt} &= vQ + (1-f)\sigma - \theta S(1-k_2) + \delta R - \mu S - \theta SI \\ \frac{dL}{dt} &= \theta SI - \beta L - \mu L - \eta(1+k_1)L \\ \frac{dI}{dt} &= \beta L + \theta S(1-k_2) - ((\mu + \gamma) + \alpha(1+k_2))I \\ \frac{dR}{dt} &= \omega T - \mu R - \delta R \\ \frac{dT}{dt} &= \eta(1+k_1)L + \alpha(1+k_2)I - \mu T - \omega T \end{aligned} \right\}$$

By sensitivity theory, see e.g. [2, 5, 9, 18] and [12], it is expected that a significant perturbation in the model parameters leads to a change in the behaviour of the equilibrium solution of the model. We proceed to study the qualitative properties of the model through the variation of the model parameters such as in [6] and [8].

3. MAIN RESULTS

We firstly prove the positivity of the solution set of the model.

Lemma 3.1. *The extended gonorrhoea model equations admit non-negative solution set.*

Proof. A direct computation of the model equations (2) gives

$$\begin{aligned} Q(t) &= \frac{f\sigma}{\mu + v} + c_1 e^{-(\mu+v)t} > 0; \\ S(t) &= \frac{vQ + (1-f)\sigma + \delta R}{\mu + \theta(1-k_2) + \theta I} + c_2 e^{-(\mu+\theta(1-k_2)+\theta I)t} > 0 \Rightarrow S(t) > 0; \\ L(t) &= \frac{\theta IS}{\mu + \beta + \eta(1+k_1)} + c_3 e^{-(\mu+\beta+\eta(1+k_1))t} > 0 \Rightarrow L(t) > 0; \\ I(t) &= \frac{\beta L + \theta S(1-k_2)}{\mu + \gamma + \alpha(1+k_2)} + c_4 e^{-(\mu+\gamma+\alpha(1+k_2))t} > 0; \\ R(t) &= \frac{\omega T}{\mu + \delta} + c_5 e^{-(\mu+\delta)t} > 0 \Rightarrow R(t) > 0 \text{ and} \\ T(t) &= \frac{\eta(1+k_1)L + \alpha(1+k_2)I}{\mu + \omega} + c_6 e^{-(\mu+\omega)t} > 0 \Rightarrow T(t) > 0 \end{aligned}$$

for arbitrary positive constants c_1, c_2, \dots, c_6 . □

Next, we show that there exists a feasible solutions region for the model.

Proposition 3.2. *Let $\dot{x} = f(x)$ in $D \subset \mathbb{R}_+^n$ be a system of equations in the feasible solutions region of the model, (2). Then the solutions $x(t)$ are feasible for all $t \geq 0$ if $x(0) \in D \subset \mathbb{R}_+^6$.*

Proof. It suffices to prove that the solution set $x(t) = \{(Q(t), S(t), L(t), I(t), T(t), R(t))\}$ enters the invariant region $\mathbb{D} \subset \mathbb{R}_+^6$. Since $N = Q + S + L + I + R + T$, it follows that

$$\frac{dN}{dt} = \frac{dQ}{dt} + \frac{dS}{dt} + \frac{dL}{dt} + \frac{dI}{dt} + \frac{dR}{dt} + \frac{dT}{dt}.$$

This implies that

$$\frac{dN}{dt} = \sigma - \mu N - \gamma I \leq \sigma - \mu N \Rightarrow \frac{dN}{dt} \leq \sigma - \mu N.$$

Solving gives

$$N \leq \frac{\sigma - qe^{-\mu t}}{\mu} \text{ with } N(0) = N_0 \leq \frac{\sigma - qe^{-\mu t}}{\mu}$$

where q is arbitrary constant and $q = \sigma - \mu N(0)$. Therefore,

$$N(t) \leq \frac{\sigma}{\mu} - \frac{(\sigma - \mu N(0))e^{-\mu t}}{\mu}.$$

Observe that as $t \rightarrow \infty$, the population size approaches the carrying capacity $\frac{\sigma}{\mu}$. That is,

$$0 < N \leq \frac{\sigma}{\mu} \Rightarrow N \rightarrow \frac{\sigma}{\mu}.$$

Hence, the feasible solution set of the system enters the invariant region $D \subset \mathbb{R}_+^6$, and

$$S > 0, Q > 0, L \geq 0, I \geq 0, T \geq 0, R \geq 0.$$

Whenever $N \leq \frac{\sigma}{\mu}$, every solution with initial condition in D remains in that region for $t > 0$.

Hence, the region D is positively invariant or bounded with $\frac{\sigma}{\mu}$ as the upper bound, $x(t) \in D$. \square

Lemma 3.3. *The extended model admits disease-free and endemic equilibria.*

Proof. Setting the right hand side of the system (2) equal to zero supposes there is no gonorrhoea infection in the population, i.e. $L = I = R = T = 0$ which gives the disease-free equilibria. On the other hand, suppose $L \neq 0$, $I \neq 0$, $R \neq 0$ and $T \neq 0$, one solves to obtain the endemic

equilibrium state of the model to be

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

□

Now, we recall that the basic reproduction number R_0 is the expected number of secondary infection produced in a completely susceptible population by a typical or one infected individual. It determines how long an infectious disease prevails in a given population, see e.g. [22]. When $R_0 < 1$, it indicates that with time the disease will die out of the population thereby giving it a clean health bill. But if R_0 is greater than one, we expect the disease will persist in the population. So for the infection to die out of the population, R_0 must be less than 1. We have the following result.

Theorem 3.4. *The basic reproduction number of the extended model $0 < R_0 \ll 1$.*

Proof. Following [7], we obtain the basic reproduction number R_0 from the model (1) without control measures, using next generation matrix method.

So, let

$$f_i = \begin{bmatrix} \theta IS \\ \theta S \\ 0 \end{bmatrix}$$

so that

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

and let

$$v_i = \begin{pmatrix} \beta L + \mu L + \eta L \\ \mu I + \gamma I + \alpha I - \beta L - \theta S \\ \mu T + \omega T - \eta L - \alpha I \end{pmatrix}$$

so that

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

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

$$V = \begin{pmatrix} (\mu + \gamma + \alpha)(\mu + \omega) & \beta(\mu + \omega) & \alpha\beta + \eta(\mu + \gamma + \alpha) \\ 0 & (\beta + \mu + \eta)(\mu + \omega) & \alpha(\beta + \mu + \eta) \\ 0 & 0 & (\beta + \mu + \eta)(\mu + \gamma + \alpha) \end{pmatrix}$$

with inverse

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

Thus,

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

implies

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

Therefore, either $\lambda^2 = 0$ or $\lambda = \frac{(\beta\theta S)}{(\beta + \mu + \eta)(\mu + \gamma + \alpha)}$. So for $\lambda \neq 0$ we obtain the R_0 to be

$$(3) \quad R_0 = \frac{(\beta\theta S)}{(\beta + \mu + \eta)(\mu + \gamma + \alpha)} = \frac{\sigma\beta\theta(\mu + \nu) - \mu f}{\mu(\mu + \alpha + \gamma)(\mu + \beta + \eta)(\mu + \nu)}.$$

Parameter/Variable	β	θ	μ	η	γ	α	δ	v	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	

TABLE 1. Parameters/variables and values.

Using the data Table 1, we quickly observe that

$$R_0 = 0.1566070960 \ll 1.$$

When control measure is given to a model, we compute the effective reproduction number, R_e , similarly. From (2) let

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

So that

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

and 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 co-factors matrix is then

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

So, following the same procedure for the computation of reproduction number, we get the eigenvalues:

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

Therefore, the effective reproduction number

$$(4) \quad R_e = \frac{(\beta\theta S)}{(\beta + \mu + \eta(1+k_1))(\mu + \gamma + \alpha(1+k_2))}.$$

It can be observed here that the effective reproduction number is far less than the basic reproduction number i.e. $R_e \ll R_0$ since

$$\frac{(\beta\theta S)}{(\beta + \mu + \eta(1+k_1))(\mu + \gamma + \alpha(1+k_2))} < \frac{(\beta\theta S)}{(\beta + \mu + \eta)(\mu + \gamma + \alpha)}.$$

Using the data in Table 1, we see that

$$\begin{aligned} R_e &= \frac{\sigma\beta\theta((\mu + \nu) - \mu f)}{\mu(\mu + \alpha + \gamma)(\mu + \beta + \eta)(\mu + \nu)} < \frac{(1 - k_2)(\alpha + \gamma + \mu)(\beta + \mu + \eta)}{(\alpha(1 + k_2) + \gamma + \mu)(\eta(1 + k_1) + \beta + \mu)} \\ &= 0.09700176367 < 0.1566070960 \ll 1. \end{aligned}$$

This gives the result. □

One can as well use the Routh-Hurwitz criteria, see [23], to assess the local stability of the model. In this technique, an equilibrium point is called asymptotically stable if the trace of the Jacobian of the next generation matrix of the model is less than zero. This means that all the roots of the characteristic polynomial have negative real parts. We have the next result.

Theorem 3.5. *The extended model, (2), is asymptotically stable.*

Proof. From the model system (1) we set

$$\begin{aligned} f_1 &= f\sigma - (\nu + \mu)Q \\ f_2 &= \nu Q + (1 - f)\sigma - \theta(1 - k_2)S + \delta R - \mu S - \theta IS \\ f_3 &= \theta SI - (\beta + \mu + \eta(1 + k_1))L \\ f_4 &= \beta L + \theta S(1 - k_2) - (\mu + \gamma + \alpha(1 + k_2))I \\ f_5 &= \omega T - (\mu + \delta)R \\ f_6 &= \eta(1 + k_1)L + \alpha(1 + k_2)I - \mu T - \omega T. \end{aligned}$$

At the disease-free equilibrium, the Jacobian matrix, $J|_{(DFE)}$, of the model and the associated characteristics determinant $|J - \lambda I| = 0$ implies

$$\begin{aligned} & (-(\mu + \nu) - \lambda)(-(\mu + \theta(1 - k_2)) - \lambda)(-(\beta + \mu + \eta(1 + k_1) - \lambda)(-\alpha(1 + k_2) \\ & + \gamma + \mu) - \lambda)(-(\mu + \delta) - \lambda)(-(\mu + \omega) - \lambda) = 0. \end{aligned}$$

Therefore,

$$\begin{aligned} \lambda_1 &= -(\mu + \nu), \\ \lambda_2 &= -(\mu + \theta(1 - k_2)), \\ \lambda_3 &= -(\beta + \eta(1 + k_1) + \mu), \\ \lambda_4 &= -(\gamma + \alpha(1 + k_2) + \mu), \\ \lambda_5 &= -(\mu + \delta), \\ \lambda_6 &= -(\mu + \omega); \end{aligned}$$

where $\lambda_1, \lambda_2, \lambda_3, \lambda_4, \lambda_5$ and λ_6 corresponding to $\lambda_j, j = 1, 2, \dots, 6$ are the eigenvalues. Since all the eigenvalues are negative, we conclude that the model is asymptotically stable. \square

We observe that this result indicates that the control interventions in the model such as the use of condom, education enlightenment programme and treatment are effective enough to almost wipe out the disease in a limited time.

One can also discuss the global stability of the Model. Here, we use Lyapunov direct method, (see e.g. [23]), to derive sufficient condition for the global stability of the system.

Theorem 3.6. [23]. *Let D be an open subset of \mathbb{R}^6 and $\dot{x} = f(x)$ a system of differential equations for $x \in D$. Let $V : D \rightarrow \mathbb{R}^6$ be a smooth function. Suppose V is positive definite around a critical point $x^* \in D$, i.e., $V(x^*) = 0$ and $V(x) > 0$, for all $x \in D$ with $x \neq x^*$. Then, the critical solution $x = x^*$ is asymptotically stable in the sense of Lyapunov if the time derivative $\dot{V} < 0$.*

For Proof, see e.g. [23]. We can now present one of the main results of this study.

Theorem 3.7. *The equilibrium point solution is globally asymptotically stable.*

Proof. In $R_0 = \frac{\beta\theta S}{(\alpha + \gamma + \mu)(\beta + \eta + \mu)}$, let $\mu_0 = \alpha + \gamma + \mu$ and $\mu_1 = \beta + \eta + \mu$.

Then $R_0 = \frac{\beta\theta S}{\mu_0\mu_1}$ so that $\frac{\mu_0\mu_1 R_0}{\beta} = \theta S$. Since $R_0 < 1$, then $R_0 - 1 < 0$ and so $\frac{\mu_0\mu_1}{\beta}(R_0 - 1) < 0$.

If we choose $\varepsilon > 0$ sufficiently small such that $\sigma = \frac{\mu_1}{\beta} - \varepsilon$ where $\frac{\beta + \eta + \mu}{\beta}$ is the mean number of susceptible people that are infected by one infectious individual during the infectious time and σ is the recruitment rate direct to susceptible class.

Now, define a Lyapunov function $V(L, I)$ by

$$(5) \quad V = L + \sigma I.$$

Clearly, V is a positive definite function and so we can show that its time derivative \dot{V} is negative definite. That is,

$$\begin{aligned} \frac{dV}{dt} &= \frac{dL}{dt} + \frac{dI}{dt}\sigma \\ &= \theta IS - (\beta + \eta + \mu)L + \sigma(\beta L - (\alpha + \mu + \gamma)I) \\ &= \theta IS - \mu_1 L + \sigma\beta L - \sigma\mu_0 I < 0. \end{aligned}$$

Take co-efficients of L and I to have that

$$\begin{aligned} L_c &= \sigma\beta - \mu_1 \\ I_c &= \theta S - \mu_0\sigma. \end{aligned}$$

From L_c we have that

$$\begin{aligned} \left(\frac{\mu_1}{\beta} - \varepsilon\right)\beta - \mu_1 = -\varepsilon\beta < 0 &\Rightarrow \theta S - \mu_0\left(\frac{\mu_1}{\beta} - \varepsilon\right) = \theta S - \frac{\mu_0\mu_1}{\beta} + \varepsilon\mu_0 \\ \frac{\mu_0\mu_1 R_0}{\beta} - \frac{\mu_0\mu_1}{\beta} + \varepsilon\mu_0 &= \frac{\mu_0\mu_1}{\beta}(R_0 - 1) + \varepsilon\mu_0 < 0. \end{aligned}$$

This implies,

$$\frac{dV}{dt} = \frac{dL}{dt} + \sigma \frac{dI}{dt} < 0.$$

Hence, $\dot{V} < 0$ as required. □

Finally, we conduct the sensitivity analysis of the model on the effective reproduction number.

Theorem 3.8. *The dynamics of the model is variable and dependent on the waning rate, the control parameters and the interaction between the latent and infected classes.*

Proof. Following [5], we prove Theorem (3.8) using the sensitivity index function of the effective reproduction number given by

$$(6) \quad \gamma_{R_e}(\cdot) = \frac{\partial R_e}{\partial(\cdot)} \times \frac{(\cdot)}{R_e}.$$

So, the variation of the treatment rate of the infected class is

$$\gamma_{R_e}(\alpha) = \frac{\alpha}{R_e} \times \frac{\partial}{\partial \alpha} R_e = -\left(\frac{\alpha((\beta + \mu + \eta(1+c))(1+k))}{(\beta + \mu + \eta(1+c))(\mu + \Omega + \alpha(1+k))} \right).$$

Now taking $\Omega = 0.01, \alpha = 0.2, \beta = 0.01, c = 0.5, \eta = 0.1, k = 0.8, \mu = 0.2$, we have

$$\gamma_{R_e}(\alpha) = -0.6315789474.$$

Similarly, the variation of the rate at which the the recovered class becomes susceptible again is

$$\gamma_{R_e}(\delta) = \frac{\delta}{R_{[e]}} \times \frac{\partial}{\partial \delta} R_{[e]} = \lambda\left(\left(\frac{\alpha((\beta + \mu + v(1+f))(1+k))}{(\beta + \mu + v(1+f))(\mu + \delta + \theta(1+k))} \right) \right).$$

Take $\delta = 0.4, \theta = 0.5, \beta = 0.01, f = 0.91, v = 0.4, k = 0.8, \mu = 0.2$ to have

$$\gamma_{R_e}(\delta) = +1.000000000.$$

Continuing this way, we obtain the sensitivity index table of the model as Table 2:

Parameters	Sensitivity indices $\gamma_{R_e}(\cdot)$
θ	+1
σ	+1
β	+0.9722222218
v	+0.2902711325
γ	-0.01754385965
k_1	-0.1388888889
μ	-2.176703880
η	-0.4166666667
α	-0.6315789474
f	-0.4354066982
k_2	-0.4280701754

TABLE 2. Sensitive parameters and values.

Graphically, we have the effect of increasing waning rate on the susceptible and immune classes as Figure 3 while Figure 4 shows the effect of a higher waning rate.

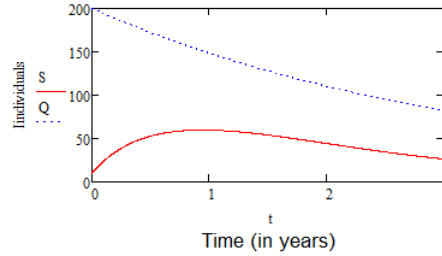


FIGURE 3. Waning rate $v = 0.2$.

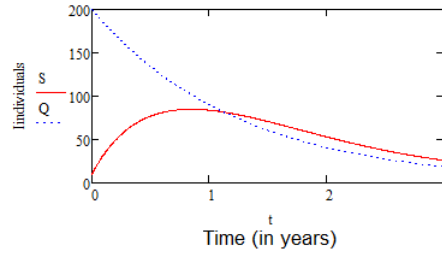


FIGURE 4. Waning rate $v = 0.6$.

The graphs indicate that when the waning rate v is low (i.e., $v = 0.2$), the passive immune population decreases exponentially with time, while when the waning rate is high, (i.e., $v = 0.6$), the passive immune population decreases faster and vanishes 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 v is low and increases faster as the waning rate v is high as shown in Figure 3 and Figure 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 μ .

Next figures show the effect when the control measures are removed and when they are introduced. Figure 5 shows when $k_1 = k_2 = 0$ that the susceptible individuals first increased after 45 days due to recruitment into it, and the trajectory decreases with time as more people

are getting infected with the disease.

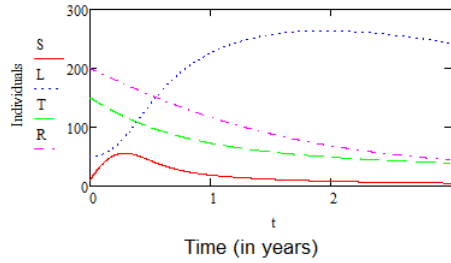


FIGURE 5. When $k_1 = 0 = k_2$.

In the same way the latent and the infected population show exponential increase because more people are getting infected without control measure. However, the treated and the recovered population show drastic exponential decay with time as a result of no control measure.

Figure (6) suggests that susceptible population increases exponentially with time since more people get treated, recovered and join the susceptible class again because of increase in control measure. Also the recovered class increases with time for about 7 months as the control measure is high (i.e., $k_1 = 0.7$ and $k_2 = 0.9$), and started decreasing again asymptotically with time because recovery from gonorrhoea is with temporal immunity. However, the trajectory of the latent and the infected classes decreased to zero with time due to increase in control measure.

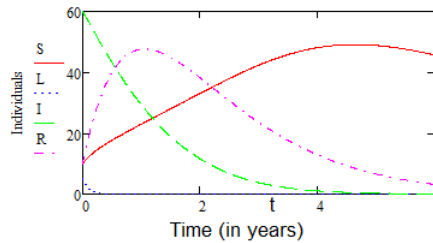


FIGURE 6. Effect when $k_1 = 0.7$ and $k_2 = 0.9$.

Figure (7) 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 vanishes 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 basic reproduction number of the disease becomes zero.

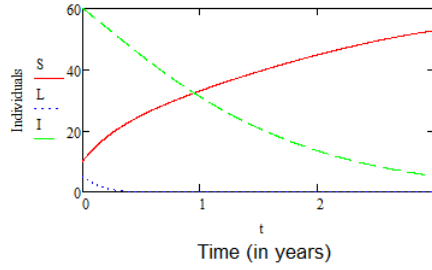


FIGURE 7. Effect when $\lambda = \theta I$, $\theta = 0$, and when $\theta = 0.3$.

We conclude that

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

At this point, the contact rate λ becomes zero and hence, nobody suffers the disease. \square

4. CONCLUSION

Based on the analysis and results of this work, we observed that the solution set of the state variables of the extended model are all positive for $t \geq 0$. This clearly showed that the population we are studying is not a zero population. We obtained an important threshold parameter called the effective reproduction number \mathbb{R}_e using the next generation matrix method. The result of the effective reproduction number is less than one. The local and global stability were investigated using Routh-Hurtz criteria and Lyapunov method respectively and both were asymptotically stable for $R_e < 1$.

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. Also from the analysis, it can be seen that the disease can be totally eliminated from the community, because the sensitivity index shows that the lower the waning rate ν , the more the exponential decrease in the passive immunity but the susceptible population increases to the equilibrium and wanes asymptotically due to the presence of the control parameters and restricted interaction of the latent and infected classes.

The sensitivity analysis of the effective reproduction number further shows that the rate of expiration of immunity ν , coupled with the interaction rate θ , recruitment rate σ and the infectious rate β are the most sensitive parameters to be targeted by way of intervention strategy.

CONFLICT OF INTERESTS

The author(s) declare that there is no conflict of interests.

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