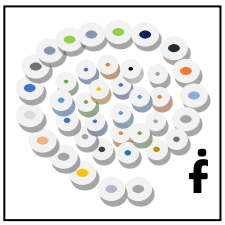

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# Modelling and analysis of a model for Chlamydia Trachomatis transmission dynamics

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**Abstract**

Chlamydia infection, one of the commonest sexually transmitted infections (STIs), remain a public health challenge in both underdeveloped and developed countries of the world. Chlamydia trachomatis has been observed to have negative health consequences hence much research work is needed to be done to curb the spread of the disease in the population. In this paper, a mathematical model for studying the impact of condom usage and treatment on the transmission dynamics and control of Chlamydia in the population is presented. Qualitative analysis of the model shows that it undergoes the phenomenon of backward bifurcation. In the absence of this phenomenon (which is shown to occur as a result of the reinfection of recovered individuals), the disease-free equilibrium of the model is globally asymptotically stable whenever the associated reproduction number is less than unity. Further, for the same scenario as above, it is shown that the unique endemic equilibrium of the model exists whenever the reproduction number is greater than unity. Numerical results show a relationship between the progression rate, treatment rate and the reproduction number. Results from the sensitivity analysis of the model, using the reproduction number, $R_c$ reveal that the top parameters that significantly drive the dynamics of Chlamydia in the population are the efficacy of condoms, condom compliance, a fraction of treated individuals who recover due to effective treatment and treatment rate. Numerical simulations of the model suggest that infected persons after treatment should wait for at least 7 days before engaging in any form of sexual activity or, if not possible use condoms correctly (to avoid reinfection) in order to effectively control the spread of the disease in the population.

*Keywords*:Chlamydia; reproduction number; reinfection; stability; bifurcation

## 1 Introduction

Chlamydia trachomatis, one of the commonest sexually transmitted infections (STIs) is primarily transmitted through vaginal, oral and anal sex, is a preventable and curable STIs caused by the bacterium Chlamydia trachomatis [8, 9, 34]. In 2020, an estimated 128.5 million new infections associated with Chlamydia trachomatis was reported worldwide amongst adults between 15 to 49 years of age and their prevalence globally was estimated to be 4.0 percent for women and 2.5 percent for men [34]. Furthermore, reports from Statista show that in 2020 about 25.5 million people in Africa had Chlamydia. The prevalence rate was higher among females with about 14.7 million people infected while 10.8 million males were infected with Chlamydia [31]. In Chlamydia infection, about 80 percent of infected people have no symptoms [23, 34]. If symptoms occur, they may not appear for about three weeks after having sex with someone who is infected with Chlamydia. In women, common symptoms include: a change in vaginal discharge, bleeding between menstrual period or after sex, itching or burning in and around the vagina, pain or discomfort in the lower abdomen, burning sensation when urinating while in men, common symptoms include: burning sensation when urinating, discharge from the penis and

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pain or discomfort in the testicles. Further, anal infection in women and men can equally cause pain, discharge and bleeding[8, 9, 34].

When someone is diagnosed with an uncomplicated Chlamydia infection, treatment can be administered using antibiotic tablets (mainly azithromycin or doxycycline) [9]. It is important to note that treatment may fail when individuals under treatment continue to have unprotected sex with infected partners. In pregnant women, untreated Chlamydia is associated with pre-term delivery and can even spread to the newborns thus causing an eye infection or pneumonia while in men, untreated Chlamydia can cause infection of the urethra which often spread to the tube that carries sperm from the testis. This may cause pain, fever and in most cases infertility [23, 28]. Chlamydia can be prevented through consistent and correct use of condoms when having vaginal or anal sex. At the moment, there are no vaccines for the prevention of Chlamydial infection [34].

Mathematical epidemiology is the study of diseases with the sole aim of identifying factors responsible for their occurrence. Over the last two decades, mathematical models have become veritable tools in epidemiology for gaining insights into the epidemiological patterns of diseases and predicting the consequences of the introduction of public health strategies in controlling the spread of such diseases [27]. Theoretical developments on numerous epidemic models are extensively discussed in [1, 3, 10, 11, 12, 16, 22, 27, 29, 30]. Research works on the use of mathematical models in studying the transmission dynamics, prevention and control of Chlamydia are very few [28]. For example, [18, 20] developed a discrete-time population-level models while [4, 25] designed a continuous-time models. A delayed differential equation model was formulated by [5] and was used to estimate the parameters associated with the intracellular development cycle of Chlamydia trachomatis. In the works of Wilson [35, 36], several differential equation models were developed for studying different aspects of Chlamydia dynamics in vivo. Further, [19, 32] designed other forms of Chlamydia models involving the use of an individually-based stochastic model and semi-parametric threshold regression analysis [37]. Also, [28, 29, 30] developed and rigorously analyzed a two group deterministic mathematical model for the spread of Chlamydia trachomatis in a population. [21] designed a deterministic model for assessing the epidemiological consequences of chlamydia gonorrhea co-infection and qualitatively analyzed it. In another research work by [26], a delayed chlamydia epidemic model with pulse vaccination was analyzed while [27], formulated and analyzed a Chlamydia epidemic model using mass action incidence function. Their model were compartmentalized into the susceptible $(S)$, exposed (infected but not yet infectious)$(E)$, infectives in the asymptomatic stage (showing no symptoms of Chlamydia)$(I_A)$, infectives in the symptomatic stage (showing symptoms of Chlamydia)$(I_S)$ and the recovered class (infectious people who have cleared or recovered from Chlamydia infection)$(R)$. Consequently, this model was extended to an optimal control model. The present study complements the aforementioned research works (particularly the one by [27]) by developing and analyzing an equivalent mathematical model for Chlamydia transmission dynamics by incorporating standard incidence function, condom usage and a class of treated individuals who failed treatment for the spread of Chlamydia in a population. This model on Chlamydia transmission dynamics is probably the first to incorporate the class of treated individuals who failed treatment. The model is based on the following assumptions:

i. The present study incorporates the class of treated individuals who failed treatment. This assumption is in line with the information obtained from [9, 34].

ii. This study incorporates the use of condoms in preventing Chlamydia infections as reported in [9, 34].

iii. This study adopts the use of standard incidence function since it is more appropriate for large population [1].

The rest of the paper is organized as follows. The model is developed in Section 2 and analyzed in Section 3. Sensitivity analysis and numerical simulations of the model are presented in Sections 4 and 5, respectively while the concluding remarks in Section 6.

## 2 Model Formulation

The total sexually active population at time $t$, denoted by $N(t)$, is divided into the mutually exclusive compartments of the susceptible individuals $(S(t))$, exposed individuals $(E(t))$, infected individuals in the asymptomatic stage $(I_a(t))$, infected individuals in the symptomatic stage $(I_s(t))$, treated individuals who failed treatment $(Q(t))$ and recovered individuals $(R(t))$.
Infectious interactions in the population are modelled with a standard incidence function. Let $\Lambda$ represent the recruitment rate of newborns and sexually active individuals (assumed susceptible) into the population while $\varepsilon$ and $\psi$ $(0 < \varepsilon, \psi < 1)$ represents the efficacy of condoms in preventing chlamydia infection in susceptible individuals and comdom compliance, respectively. Also, we further assume that $\beta_1$ is the coefficient of transmission of chlamydia infection from infectives in the asymptomatic stage to the susceptibles, $\beta_2$ is the coefficient of transmission of chlamydia infection from infectives in the symptomatic stage to the susceptibles, $\beta_3$ is the coefficient of transmission of chlamydia infection from individuals who failed treatment to the susceptibles, $\mu$ is the natural death rate associated to all the epidemiological classes. Let $\rho\eta$ be the proportion of exposed individuals who are asymptomatic while $(1-\rho)\eta$ is the proportion of exposed individuals who are symptomatic. Let $\eta$ be the progression rate of individuals in the exposed class to the asymptomatic and symptomatic stages of infection, respectively.

A fraction, $\phi$, of the treated individuals in the symptomatic stage of infection, will recover and move to class R, while the remaining fraction $(1-\phi)$, will fail treatment and move to the class Q. Individuals who have failed treatment will eventually br treated succesfully at a rate $\sigma\tau$ and move to class R. The parameter $\sigma < 1$ accounts for the assumed decrease in the recovery rate of individuals in class Q in comparison to the successfully treated individuals. Further, recovered individuals are reinfected at a rate $\theta\lambda$ $(\theta > 0)$. Recovered individuals can relapse and become infectious again, at a rate $\gamma$. Putting together these definitions and assumptioms, it follows that the model for the transmission dynamics of chlamydia in a sexually active population is given by the following system of non-linear ordinary differential equations (a flow diagrame of the model is shown in Figure 1, and the state variables and parameters of the model are presented in Table 1):

$$\begin{aligned}
\frac{dS(t)}{dt} &= \Lambda - \lambda S - \mu S,\\
\frac{dE(t)}{dt} &= \lambda S - [\rho\eta + (1-\rho)\eta + \mu]E,\\
\frac{dI_a(t)}{dt} &= \rho\eta E + \theta\lambda R + \gamma R - [(1-k)m + km + \mu]I_a,\\
\frac{dI_s(t)}{dt} &= (1-\rho)\eta E + (1-k)mI_a - [(1-\phi)\tau + \phi\tau + \mu]I_s,\\
\frac{dQ(t)}{dt} &= (1-\phi)\tau I_s - (\sigma\tau + \mu)Q,\\
\frac{dR(t)}{dt} &= \phi\tau I_s + \sigma\tau Q + kmI_a - [\theta\lambda + (\mu+\gamma)]R
\end{aligned} \tag{1}$$

where

$$\lambda = (1-\varepsilon\psi)\Big(\frac{\beta_1 I_a + \beta_2 I_s + \beta_3 Q}{N}\Big), \tag{2}$$

and

$$N = S + E + I_a + I_s + Q + R. \tag{3}$$

Therefore, the model (1) adds to the existing models on chlamydia transmission dynamics as in the literature (see for example, [27])

i. Allowing for the reinfection of individuals who recover from the infection.

ii. Allowing for the relapse of individuals who recovered from the infection.

The flow diagram of the model is shown in Figure 1, and the state variables and parameters of the model are tabulated/interpreted in Table 1.

Table 1: Description of state variables/parameters in the chlamydia model (1).

| Variables/parameters | Interpretation |
|---|---|
| $S$ | Susceptible individuals |
| $E$ | Exposed individuals |
| $I_a$ | Infected individuals in the asymptomatic stage |
| $I_s$ | Infected individuals in the symptomatic stage |
| $Q$ | Treated individuals who failed treatment |
| $R$ | Recovered individuals |
| $\Lambda$ | Recruitment rate |
| $\beta_1$ | The coefficient of transmission of chlamydia infection from infectives in the asymptomatic stage to the susceptibles |
| $\beta_2$ | The coefficient of transmission of chlamydia infection from infectives in the symptomatic stage to the susceptibles |
| $\beta_3$ | The coefficient of transmission of chlamydia infection from individuals who failed treatment to the susceptibles |
| $\mu$ | Natural death rate |
| $\varepsilon$ | Efficacy of condoms in preventing chlamydia infection in susceptible individuals |
| $\psi$ | Condom compliance rate |
| $\rho\eta$ | Proportion of exposed individuals who are asymptomatic |
| $(1-\rho)\eta$ | Proportion of exposed individuals who are symptomatic |
| $\theta$ | Re-infection rate |
| $\gamma$ | Relapse rate for individuals |
| $\sigma$ | Accounts for the assumed decrease in the recovery rate of individuals in class Q in comparison to the successfully treated individuals |
| $\phi$ | A fraction of treated individuals who recover due to effective treatment and move to R |
| $\tau$ | Treatment rate |
| $km$ | Accounts for the proportion of the asymptomatic individuals who recover and moved to R |
| $\lambda$ | Force of infection |
| $\eta, m$ | Progression rates |

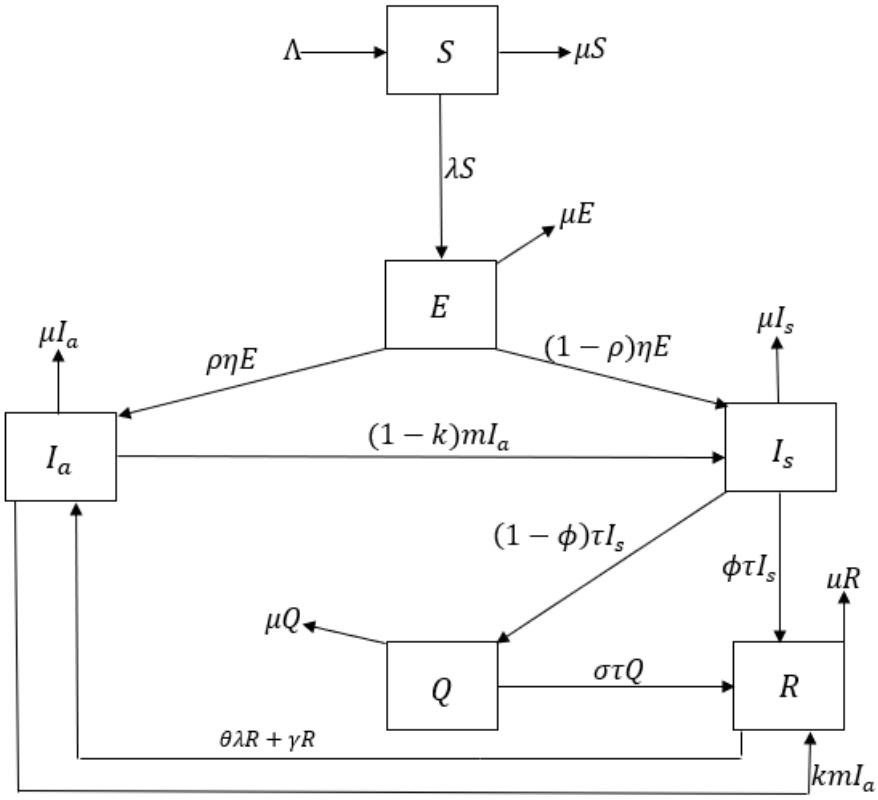


Figure 1: Schematic diagram for model (1)

## 2.1 Basic properties

For model (1) to be epidemiologically meaningful, it is vital to show that all its state variables are non-negative for all time, $t$. In order words, the solutions of model (1) with positive initial data will remain positive for all $t \geq 0$.

### 2.1.1 Positivity and boundedness of solution.

Since the model (1) monitors the human population, the state variables and parameters of the model are non-negative. Consider the biologically feasible region.

$$\Gamma = \{(S, E, I_a, I_s, Q, R) \in \mathbb{R}^6_+ : N \leq \frac{\Lambda}{\mu}\}. \tag{4}$$

It can be shown that the set $\Gamma$ is a positively invariant set and a global attractor of this system. This implies that, any phase trajectory initiated anywhere in the non-negative region $\mathbb{R}^6_+$ of the phase space will eventually enter the region $\Gamma$ and remains in $\Gamma$ thereafter.

**Lemma 2.1.** *The region $\Gamma$ is positively invariant for model (1).*

*Proof.* The rate of change of the total population with time is given by

$$\frac{dN}{dt} = \Lambda - \mu N. \tag{5}$$

Since the right hand side of (5) is bounded by $\Lambda - \mu N$, we can show using a standard comparison theorem [17] that

$$N(t) \leq N(0)exp^{-\mu t} + \frac{\Lambda}{\mu}\big(1 - exp^{-\mu t}\big). \tag{6}$$

In particular, if $N(0) \leq \frac{\Lambda}{\mu}$, then $N(t) \leq \frac{\Lambda}{\mu}$. Therefore, $\Gamma$ is positively invariant. Hence, no solution path leaves through any boundary of $\Gamma$ and it is sufficient to consider the dynamics of model (1) in $\Gamma$. Hence, in this region, the model can be thus considered as being mathematically and epidemiologically well posed [15].

# 3 Mathematical Analysis

## 3.1 Asymptotic Stability of Disease-free Equilibrium (DFE)

The DFE of the model (1) is given by $E_0 = (S^*, E^*, I_a^*, I_s^*, Q^*, R^*) = \left(\frac{\Lambda}{\mu}, 0, 0, 0, 0, 0\right)$. The local stability of $E_0$ will be obtained using the next generation operator method [33]. The notations as used in [33] is adopted, the non-negative matrix, $F$, of new infection terms and the $M$-matrix, $V$, of transition terms associated with the model (1) are

$$F = \begin{pmatrix} 0 & q\beta_1 & q\beta_2 & q\beta_3 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix} \text{ and } \quad V = \begin{pmatrix} d_1 & 0 & 0 & 0 \\ -\rho\eta & d_2 & 0 & 0 \\ -d_5 & -d_6 & d_3 & 0 \\ 0 & 0 & -d_7 & d_4 \end{pmatrix},$$

where

$$\begin{aligned} q = (1-\varepsilon\psi), d_1 = (\rho\eta + d_5 + \mu), d_2 = (d_6 + km + \mu), d_3 = (d_7 + \phi\tau + \mu), \\ d_4 = (\sigma\tau + \mu), d_5 = (1-\rho)\eta, d_6 = (1-k)m, d_7 = (1-\phi)\tau, d_8 = (\mu + \gamma). \end{aligned} \tag{7}$$

It follows that the effective reproduction number of the model (1), denoted by $R_c$, is given by

$$R_c = \rho(FV^{-1}) = \frac{q\left[\beta_1\rho\eta d_3 d_4 + (\eta\rho d_6 + d_2 d_5)(\beta_2 d_4 + \beta_3 d_7)\right]}{d_1 d_2 d_3 d_4} \tag{8}$$

where $\rho(FV^{-1})$ is the spectral radius of the matrix $(FV^{-1})$.
The result below follows from Theorem 2 in [33].

**Lemma 3.1.** *The DFE ($E_0$) of the model (1) is locally asymptotically stable if $R_c < 1$ and unstable if $R_c > 1$.*

The threshold quantity, $R_c$ is the effective reproduction number of chlamydia [33]. By definition, it represents the average number of secondary chlamydia infections generated by a typical infected person in a completely susceptible population [33]. The implication of Lemma 3.1 epidemiologically is that when $R_c < 1$ is less than one, chlamydia can be eradicated from the population if the initial sizes of the subpopulation of the model are in the basin of attraction of the DFE ($E_0$). Hence, a small inflow of chlamydia-infected person into the community will not generate large chlamydia outbreaks, and the disease will die out as time progresses.

## 3.2 Backward bifurcation analysis

### 3.2.1 Existence of backward bifurcation analysis.

It is extremely important to categorize the type of bifurcation model (1) may undergo. We claim the following result.

**Theorem 3.2.** *Model (1) exhibits backward bifurcation at $R_c = 1$ whenever a bifurcation coefficient, denoted by $a$ is positive.*

*Proof.* Suppose $E_1 = (S^{**}, E^{**}, I_a^{**}, I_s^{**}, Q^{**}, R^{**})$ represents any arbitrary endemic equilibrium of model (1) (that is an equilibrium in which at least one of the infected components is non-zero). The existence of backward bifurcation will be determined using the centre manifold theory [6, 7]. To apply

this theory, we perform the following change of variables. Let $S = x_1, E = x_2, I_a = x_3, I_s = x_4, Q = x_5$ and $R = x_6$. Also, by using the vector notation $X = (x_1, x_2, x_3, x_4, x_5, x_6)^T$. The model (1) can be written in the form $\frac{dX}{dt} = F(X)$ where $F = (f_1, f_2, f_3, f_4, f_5, f_6)^T$ as follows

$$\begin{aligned}
\frac{dx_1}{dt} &\equiv f_1 = \Lambda - (\lambda + \mu)x_1, \\
\frac{dx_2}{dt} &\equiv f_2 = \lambda x_1 - d_1 x_2, \\
\frac{dx_3}{dt} &\equiv f_3 = \rho\eta x_2 + \theta\lambda x_6 + \gamma x_6 - d_2 x_3, \\
\frac{dx_4}{dt} &\equiv f_4 = d_5 x_2 + d_6 x_3 - d_3 x_4, \\
\frac{dx_5}{dt} &\equiv f_5 = d_7 x_4 - d_4 x_5, \\
\frac{dx_6}{dt} &\equiv f_6 = \phi\tau x_4 + \sigma\tau x_5 + kmx_3 - \theta\lambda x_6 - d_8 x_6.
\end{aligned} \tag{9}$$

where (as defined earlier),

$$\begin{aligned}
&d_1 = (\rho\eta + d_5 + \mu), d_2 = (d_6 + km + \mu), d_3 = (d_7 + \phi\tau + \mu), \\
&d_4 = (\sigma\tau + \mu), d_5 = (1-\rho)\eta, d_6 = (1-k)m, d_7 = (1-\phi)\tau, d_8 = (\mu + \gamma),
\end{aligned} \tag{10}$$

and, the force of infection is given by

$$\lambda = \frac{q(\beta_1 x_3 + \beta_2 x_4 + \beta_3 x_5)}{N} \tag{11}$$

with $q = (1 - \varepsilon\psi)$ and $N = x_1 + x_2 + x_3 + x_4 + x_5 + x_6$.
We consider the scenario where $\beta_1 = \beta_2 = \beta_3 = \beta^*$ is choosen as the bifurcation parameter. Solving for $\beta_1 = \beta_2 = \beta_3 = \beta^*$ from $R_c = 1$, yields

$$\beta_1 = \beta_2 = \beta_3 = \beta^* = \frac{d_1 d_2 d_3 d_4}{q[\rho\eta d_3 d_4 + (\eta\rho d_6 + d_2 d_5)(d_4 + d_7)]} \tag{12}$$

The Jacobian matrix of the transformed system (9), evaluated at the DFE $(E^0)$ with $\beta_1 = \beta_2 = \beta_3 = \beta^*$, is given by

$$J^* = \begin{pmatrix}
-\mu & 0 & -q\beta^* & -q\beta^* & -q\beta^* & 0 \\
0 & -d_1 & q\beta^* & q\beta^* & q\beta^* & 0 \\
0 & \rho\eta & -d_2 & 0 & 0 & \gamma \\
0 & d_5 & d_6 & -d_3 & 0 & 0 \\
0 & 0 & 0 & d_7 & -d_4 & 0 \\
0 & 0 & km & \phi\tau & \sigma\tau & -d_8
\end{pmatrix}.$$

Matrix $J(E_0)$ has a right eigenvector (associated with the zero eigenvalue) given by $w = [w_1, w_2, w_3, w_4, w_5, w_6]^T$, where

$$\begin{aligned}
w_1 &= -\frac{q\beta^*}{\mu}(w_3 + w_4 + w_5), \\
w_2 &= -\frac{q\beta^*}{d_1}(w_3 + w_4 + w_5), \\
w_3 &= -\frac{b_1}{d_4(b_2 - b_3)}w_4, \\
w_4 &= w_4 > 0, \\
w_5 &= \frac{d_7}{d_4}w_4 > 0, \\
w_6 &= \frac{1}{d_8}(kmw_3 + \phi\tau w_4 + \sigma\tau w_5).
\end{aligned} \tag{13}$$

where $b_1 = \beta^*\eta q\rho d_8(d_4+d_7)+\gamma d_1\tau(\phi d_4+\sigma d_7)$, $b_2 = (\beta^*\eta q\rho d_8+\gamma k m d_1)$ and $b_3 = d_1d_2d_3$. Furthermore, $J^*$ has a left eigenvector, $v=(v_1,v_2,v_3,v_4,v_5,v_6)$, satisfying $v.w=1$, with

$$\begin{aligned} &v_1=0, v_2=\frac{(\rho\eta v_3+d_5v_4)}{d_1}, v_3=v_3>0, v_4=-\frac{b_1}{d_8(d_1-c_1)}\\ &v_5=\frac{(q\beta^*v_2+\sigma\tau v_6))}{d_4}, v_6=\frac{d_8}{\gamma}v_3, \end{aligned} \tag{14}$$

with $c_1=\beta^*qd_5(d_4+d_7)>0$ and $c_2=d_1d_2d_3>0$. From Theorem 4.1 in [7], it can be shown, by computing the non-zero partial derivatives of $F$ at the DFE $(E_0)$ and simplifying, that

$$\begin{aligned} a&=\sum_{k,i,j=1}^{6} v_kw_iw_j\frac{\partial^2 f_k}{\partial x_i\partial x_j}(E_0)\\ &=-\frac{2\mu\beta^*(w_3+w_4+w_5)[qv_2(w_2+w_3+w_4+w_5+w_6)+\theta w_6(qv_6-v_3)]}{\Lambda} \end{aligned} \tag{15}$$

Thus, the bifurcation coefficient, $a$, is positive whenever
$(w_3+w_4+w_5)[qv_2(w_2+w_3+w_4+w_5+w_6)+\theta w_6(qv_6-v_3)]<0$. Furthermore, it can be shown that

$$b=\sum_{k,i=1}^{6} v_kw_i\frac{\partial^2 f_k}{\partial x_i\partial\beta^*}(E_0)=v_2qp(w_3+w_4+w_5) \tag{16}$$

The bifurcation coefficient, b, is positive if $w_4+w_5>w_3$ and $v_2>0$ if $\rho\eta v_3>d_5v_4$. Therefore, it follows from Theorem 4.1 in [7], that the model (1), exhibits a backward bifurcation at $R_c=1$.
The phenomenon of backward bifurcation, which has been seen in several disease transmission dynamics (see for example, [7, 13]) is mainly characterized by the coexistence of a stable DFE and a stable endemic equilibrium when the associated reproduction number of the model is less than one. The implication of the phenomenon of backward bifurcation of model (1) is that the classical epidemiological requirement of having the reproduction number $(R_c)$ less than one, although necessary, is no longer sufficient for the effective control of the disease.

### 3.2.2 Nonexistence of backward bifurcation

It is instructive to note that mathematical models that incorporate reinfection of recovered individuals are known to lose their backward bifurcation property when the reinfection parameters are set to zero (see for example, [28, 29] and [15]). Here, the role of the reinfection of recovered individuals $(\theta)$ on the phenomenon of backward bifurcation for model (1) will be investigated. For ease of reference, we denote $\widehat{R_c}=R_c|\theta=\gamma=0$.

**Theorem 3.3.** *The special case of the model (1) in the absence of reinfection of recovered individuals (that is, $\theta=\gamma=0$) does not undergo a backward bifurcation at $\widehat{R_c}=1$.*

*Proof.* Setting $\theta=\gamma=0$ in the expression for the bifurcation coefficients $a$ and $b$ given in equations (15) and (16) above yields (note here that $w_5>0$, $w_4>0$ but $w_2,w_6>0$ if $(w_4+w_5)>\widehat{w_3}$ and $v_2>0$ if $\rho\eta v_3>d_5v_4$)

$$\begin{aligned} a&=\sum_{k,i,j=1}^{6} v_kw_iw_j\frac{\partial^2 f_k}{\partial x_i\partial x_j}(E_0)\\ &=-\frac{2\mu\beta^*(\widehat{w_3}+w_4+w_5)[qv_2(w_2+\widehat{w_3}+w_4+w_5+w_6)}{\Lambda} \end{aligned} \tag{17}$$

and

$$b = \sum_{k,i=1}^{6} v_k w_i \frac{\partial^2 f_k}{\partial x_i \partial \beta^*}(E_0) = v_2 qp(\widehat{w_3} + w_4 + w_5) \tag{18}$$

Therefore, from Theorem 4.1 in [7], it follows that the model (1) with $\theta = \gamma = 0$ will not undergo a backward bifurcation at $\widehat{R_c} = 1$. The epidemiological consequence of Theorem 3.3 is that the present study confirms the fact that reinfection (of recovered individuals) causes backward bifurcation in chlamydia transmission dynamics. To confirm further the absence of backward bifurcation in the model (when $\widehat{R_c} < 1$) for the special case of the model (1) with $\theta = \gamma = 0$, a global asymptotic stability result is given for the DFE $E_0$.

**Theorem 3.4.** *The DFE for the special case of the model (1) with $\theta = \gamma = 0$ is globally asmptotically stable in $\Gamma$ whenever $\widehat{R_c} \leq 1$.*

*Proof.* Note here that the equations for the infected classes of the model (1) with $\theta = \gamma = 0$ can be written in the matrix-vector form as follows:

$$\frac{dY(t)}{dt} = \Big[(F - V) - \Big(1 - \frac{S}{N}\Big)M\Big]Y(t), \tag{19}$$

where $Y(t) = \big(E(t), I_a(t), I_s(t), Q(t), R(t)\big)^T$ and the matrices $F$ and $V$ are given in Section 3. Furthermore,

$$M = \begin{pmatrix} q\beta^* & q\beta^* & q\beta^* & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}.$$

Since $M$ is a nonnegative matrix and $S \leq N$ in $\Gamma$, it follows that

$$\frac{dY(t)}{dt} \leq \big[(F - V)\big]Y(t). \tag{20}$$

Using the fact that the eigenvalues of the matrix $F - V$ all have negative real parts (that is, $\rho(FV^{-1}) < 1$ if $\widehat{R_c} < 1$. ), it follows that the linearized differential inequality system (20) is stable whenever $\widehat{R_c} < 1$. Thus, by comparison Theorem [17],

$$\lim_{t\to\infty} \big(E(t), I_a(t), I_s(t), Q(t), R(t)\big) = \Big(0, 0, 0, 0, 0\Big). \tag{21}$$

It can be shown by substituting (21) into (1) that, $S(t) \to \dfrac{\Lambda}{\mu}$, as $t \to \infty$. Hence,

$$\lim_{t\to\infty} \big(S(t), E(t), I_a(t), I_s(t), Q(t), R(t)\big) = \Big(\frac{\Lambda}{\mu}, 0, 0, 0, 0, 0\Big) = E_0. \tag{22}$$

Thus, every solution to the equation of the model (1), with $\theta = \gamma = 0$ and initial conditions in $\Gamma$, approaches the DFE $(E_0)$ as $t \to \infty$ whenever $\widehat{R_c} < 1$.

### 3.3 Existence of unique endemic equilibrium: Special case

Here, the existence of a unique endemic equilibrium of the model (1) will be examined, for the special case with no reinfection of recovered individuals (i.e., $\theta = \gamma = 0$).
To establish the existence of endemic equilibria of model (1), for the special case $\theta = \gamma = 0$ , let

$E_2 = \{S^{**}, E^{**}, I_a^{**}, I_s^{**}, Q^{**}, R^{**}\}$ represent any arbitrary equilibrium of model (1). The equations in (1), with $\theta = \gamma = 0$, are solved in terms of the force of infection at the steady state to give

$$\begin{aligned} S^{**} &= \frac{\Lambda}{(\lambda^{**}+\mu)}, E^{**} = \frac{\lambda^{**}\Lambda}{d_1(\lambda^{**}+\mu)}, I_a^{**} = \frac{\lambda^{**}\rho\eta\Lambda}{d_1 d_2(\lambda^{**}+\mu)}, \\ I_s^{**} &= \frac{\lambda^{**}\Lambda(\eta\rho d_6 + d_2 d_5)}{d_1 d_2 d_3(\lambda^{**}+\mu)}, Q = \frac{\lambda^{**} d_7 \Lambda(\eta\rho d_6 + d_2 d_5)}{d_1 d_2 d_3 d_4(\lambda^{**}+\mu)}, \\ R^{**} &= \frac{\lambda^{**}\Lambda\big[km\rho\eta d_3 d_4 + \phi\tau d_4(\rho d_6 + d_2 d_5) + \sigma\tau d_7(\eta\rho d_6 + d_2 d_5)\big]}{\mu d_1 d_2 d_3 d_4(\lambda^{**}+\mu)}. \end{aligned} \tag{23}$$

where (as defined earlier),

$$\begin{aligned} &d_1 = (\rho\eta + d_5 + \mu), d_2 = (d_6 + km + \mu), d_3 = (d_7 + \phi\tau + \mu), \\ &d_4 = (\sigma\tau + \mu), d_5 = (1-\rho)\eta, d_6 = (1-k)m, d_7 = (1-\phi)\tau. \end{aligned} \tag{24}$$

The force of infection at steady state, $\lambda^{**}$ is expressed as

$$\lambda^{**} = q\frac{(\beta_1 I_a^{**} + \beta_2 I_s^{**} + \beta_3 Q^{**})}{N^{**}} \tag{25}$$

with $q = (1 - \varepsilon\psi)$. Substituting the expressions in (23) into (25) gives

$$(1 + \frac{\lambda^{**}}{d_1} H_1) = \frac{q\big[\beta_1\rho\eta d_3 d_4 + (\eta\rho d_6 + d_2 d_5)(\beta_2 d_4 + \beta_3 d_7)\big]}{d_1 d_2 d_3 d_4}$$

which yields

$$(1 + \frac{\lambda^{**}}{d_1} H_1) = R_c$$

$$\lambda^{**} = \frac{d_1}{H_1}(R_c - 1) > 0,$$

with

$$\begin{aligned} H_1 = \Big[1 + \frac{\rho\eta}{d_2} + \frac{(\eta\rho d_6 + d_2 d_5)}{d_2 d_3} + \frac{d_7(\eta\rho d_6 + d_2 d_5)}{d_2 d_3 d_4} \\ + \frac{\big[km\rho\eta d_3 d_4 + \phi\tau d_4(\rho d_6 + d_2 d_5) + \sigma\tau d_7(\eta\rho d_6 + d_2 d_5)\big]}{\mu d_2 d_3 d_4}\Big]. \end{aligned} \tag{26}$$

Hence, a unique endemic equilibrium (for the case $\theta = \gamma = 0$) exists when $\widehat{R_c} > 1$. The above result is summarized in the theorem below.

**Theorem 3.5.** *When there are no reinfection of recovered individuals (i.e., $\theta = \gamma = 0$), the model (1) has a unique endemic equilibrium whenever $\widehat{R}_c > 1$ and no endemic equilibrium if otherwise.*

**Theorem 3.6.** *The unique endemic equilibrium of the model (1) for the special case $\theta = \gamma = 0$, is locally asymptotically stable whenever $\widehat{R}_c > 1$ and $\widehat{R_c}$ near 1.*

Here, a contour plot of the reproduction number $\widehat{R_c}$, as a function of condom compliance $(\psi)$ and efficacy of condom $(\varepsilon)$ is shown in Figure 2 using the parameter values in Table 2. This graphical profile shows that no matter the level of condom compliance and its efficacy, the control or eradication of chlamydia in the population will be difficult (since $\widehat{R_c}$ is always greater than one). Furthermore, if the treatment rate is sufficiently increased, then the effective control of chlamydia in the population will be possible using condom compliance of 75 percent (based on the assumed efficacy of 80 percent)(see Figure 3). This result is similar to the one obtained by [10]. Though the result of their study was based on syphilis while the present study is based on chylamydia.

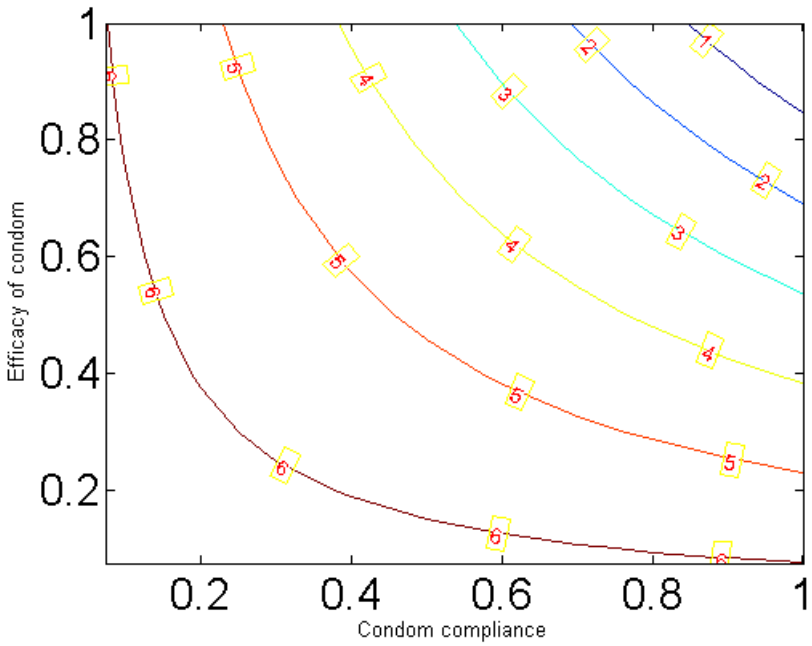


Figure 2: Contour plot of $\widehat{R}_c$ as a function of condom compliance $(\psi)$ and efficacy of condom $(\varepsilon)$. Parameter values used are as in Table 2.

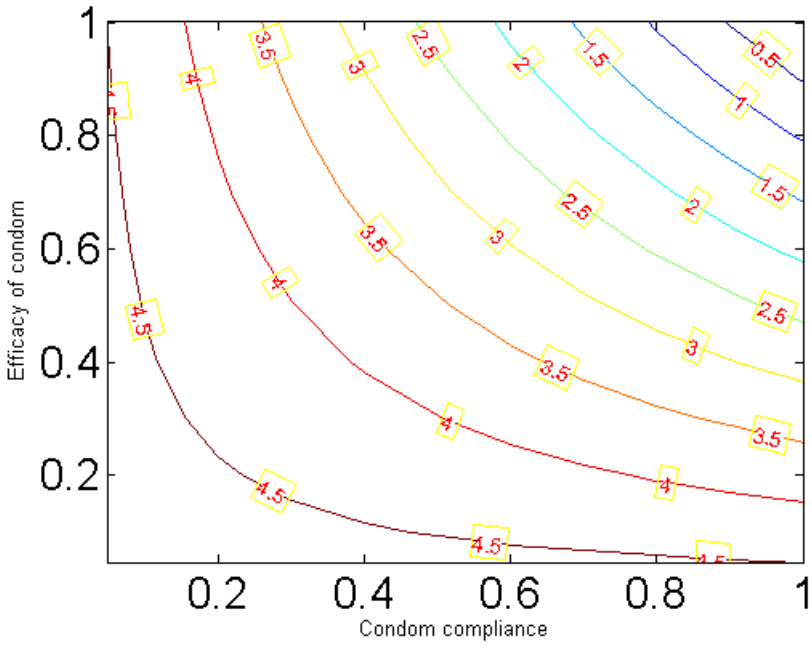


Figure 3: Contour plot of $\widehat{R}_c$ as a function of condom compliance $(\psi)$ and efficacy of condom $(\varepsilon)$. Parameter values used are as in Table 2.

## 4 Sensitivity Analysis

In this section, we conduct sensitivity analysis on the parameters of the model associated with the reproduction number, $R_c$ using the parameter values in Table 2. The essence of this analysis is to determine the relative importance of each parameter in the model that depicts Chlamydia transmission. A method similar to the ones oulined in the works of [2, 3, 24] were utilized to obtain the sensitivity index of all the parameters connected to the reproduction number using the formula in (26).

$$\chi_p^{R_c} = \frac{\partial R_c}{\partial \xi} \times \frac{\xi}{R_c}, \tag{27}$$

where $\xi$ denotes model parameters contained in $R_c$. We obtained from (26) the sensitivity indices of the parameters associated with the reproduction number as presented in Table 3. The parameters with positive sensitivity indices signify a high impact burden of Chlamydia in the population if their values keeps increasing. In a similar manner, parameters in which their sensitivity indices are negative have a great effect in reducing Chlamydia burden in the population as their values increase while the others remain constant. Hence, as their values increase, the reproduction number decreases thus reducing the endemicity of the disease in the population.

From the results of the sensitivity analysis presented in Table 3, some of the parameters with high negative indices are the efficacy of condoms in preventing Chlamydia infection in the susceptible individuals ($\varepsilon$), condom compliance ($\psi$), a fraction of treated individuals who recover due to effective treatment ($\phi$) and treatment rate ($\tau$). These are the top parameters that significantly drive the dynamics of Chlamydia in the population. Consequently, to control the spread of the disease, these top parameters must be effectively targetted by policy makers in the health sector so that the sensitivity indices of these parameters must be kept negative for effective control of the disease. This implies that the advocacy for the use of condoms by susceptible individuals and proper treatment of infected individuals must be higher if Chlamydia must be curtailed in the population.

Table 2: The parameter values of model (1)

| Parameter | Nominal value ($year^{-1}$) | Reference |
|---|---|---|
| $\Lambda$ | 1000 | [29] |
| $\beta_1$ | 0.1 | [27] |
| $\beta_2$ | 0.15 | [27] |
| $\beta_3$ | 0.14 | Implied from [27] |
| $\mu$ | 0.15 | [27] |
| $\varepsilon$ | 0.80 | [11] |
| $\psi$ | 0.75 | Assumed |
| $\rho$ | 0.70 | [27] |
| $\eta$ | 0.50 | [27] |
| $\theta$ | 0.90 | [27] |
| $\gamma$ | 0.80 | [27] |
| $\sigma$ | 0.20 | Assumed |
| $\phi$ | 0.85 | Assumed |
| $\tau$ | 0.36 | Assumed |
| $k$ | 0.70 | [27] |
| $m$ | 0.70 | [27] |

Table 3: Sensitivity indices of some parameters values

| Parameter | Sensitivity indices |
|---|---|
| $\Lambda$ | $+1.0000$ |
| $\beta_1$ | $+0.3255$ |
| $\beta_2$ | $+0.5493$ |
| $\beta_3$ | $+0.1248$ |
| $\mu$ | $-1.3837$ |
| $\varepsilon$ | $-1.5000$ |
| $\psi$ | $-1.5000$ |
| $\rho$ | $-0.4263$ |
| $\eta$ | $+1.4820 \times 10^{-10}$ |
| $\sigma$ | $-0.0405$ |
| $\phi$ | $-0.7072$ |
| $\tau$ | $-0.3918$ |
| $k$ | $-0.5755$ |
| $m$ | $-0.2245$ |

## 5 Numerical Simulations

In this section, numerical simulations are carried out on model (1) using the parameter values in Table 2. The model (1) is solved numerically using MATLAB ODE45 solver. Figure 4 shows the effect of the progression rate $(\eta)$ of individuals in the exposed stage to the asymptomatic and symptomatic stages of infection on the number of infected individuals in some of the stages of Chlamydia infection. An increase in the progression rate $(\eta)$ leads to an increase in the number of infected persons in the asymptomatic, symptomatic and individuals who failed treatment stages of Chlamydia infection. Unfortunately, increasing the treatment rate $(\tau)$ (as shown in Figure 5) does not lead to a decline rather an increase in the number of infected persons in the asymptomatic, symptomatic and treated individuals who failed treatment stages of infection. This result clearly show that treatment alone may not necessarily lead to a reduction in the number of infected persons in the population. As reported in WHO, infected persons after treatment should wait for at least 7 days before engaging in any form of sexual activity or, if not possible, use condoms correctly [34]. Violation of this report by WHO could be responsible for the increase in the number of asymptomatic, symptomatic and treated individuals who failed treatment despite the administration of drugs (azithromycin or doxycycline) (see Figure 5). Consequently, infected persons undergoing treatment or those who have successfully completed treatment are encouraged not to engage in any form of sexual activity until at least 7 days after treatment or correctly use condoms to avoid reinfection.

We demostrate numerically in Figures 6 and 7, the relationship between the progression rate $(\eta)$ and the treatment rate $(\tau)$. The reproduction number $(R_c)$ is plotted as a function of the parameters $\eta$ and $\tau$. From Figure 6, it is observed that an increase in the progression rate from the exposed class to the asymptomatic and symptomatic stages of infection leads to an increase in the reproduction number. This implies that if concerted efforts are not put in place by relevant stakeholders in the health sector, this will trigger the number of Chlamydia cases in the population. In Figure 7 (as expected), increasing the treatment rates for individuals in the symptomatic and treated individuals who failed treatment leads to a decrease in the reproduction number. This can only be achievable if infected persons complete their treatment regime and not engage in any form of sexual activity until at least 7 days after treatment or, if not possible, use condoms correctly.

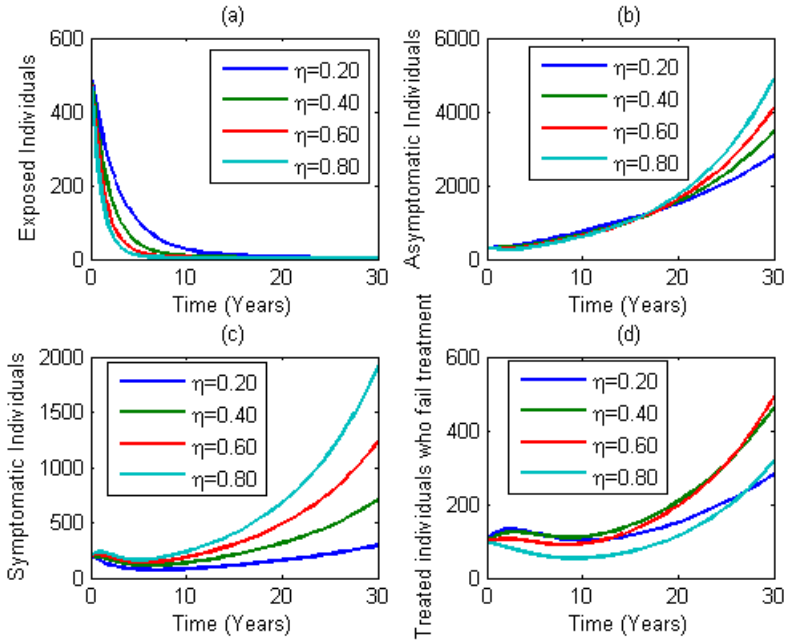


Figure 4: Simulation of model (1) showing the effect of progression rate from the exposed stage to the asymptomatic, symptomatic and treated individuals who failed treatment stages of infection. Here, $\eta$ is varied from 0.20 to 0.80.

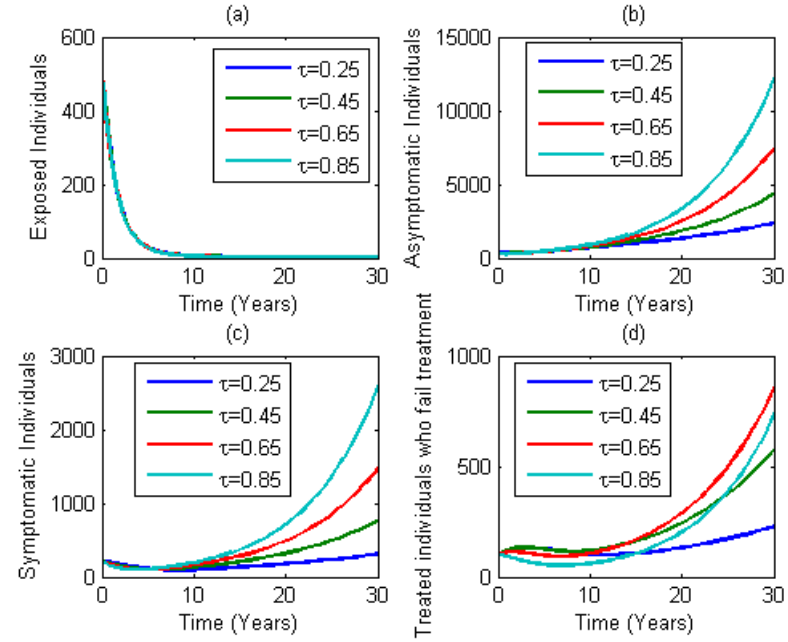


Figure 5: Simulation of model (1) showing the effect of treatment rate for individuals in the exposed, asymptomatic, symptomatic and treated individuals who failed treatment stages of infection. Here, $\tau$ is varied from 0.25 to 0.85.

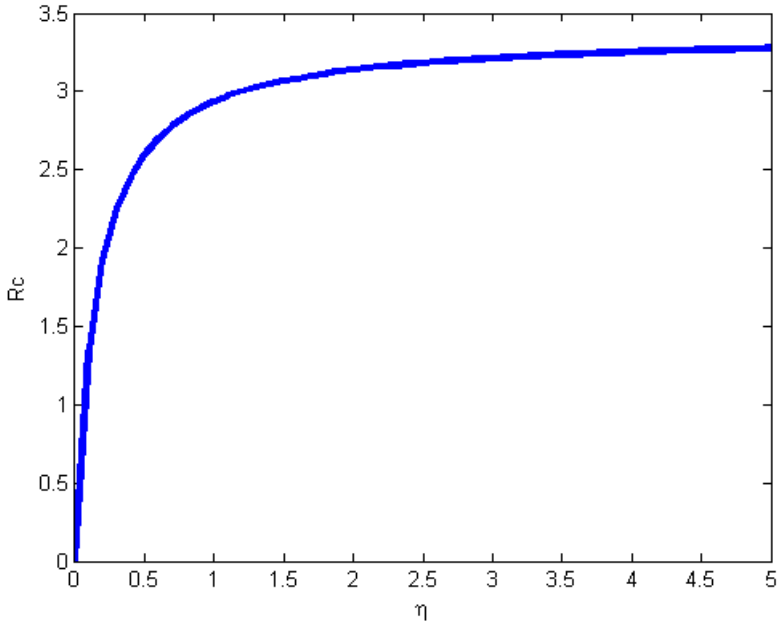


Figure 6: Plot of reproduction number, $R_c$ as a function of progression rate, $\eta$. Parameter values used are as in Table 2.

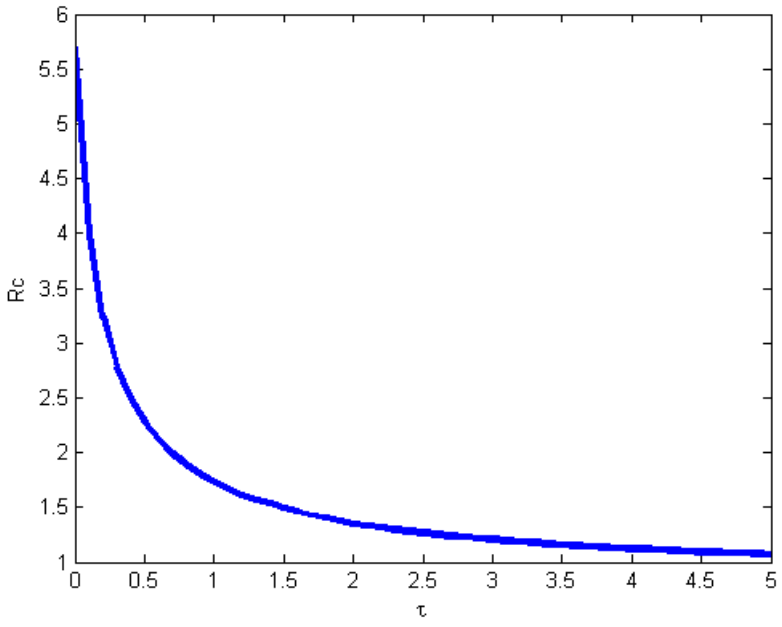


Figure 7: Plot of reproduction number, $R_c$ as a function of treatment rate, $\tau$. Parameter values used are as in Table 2.

## 6 Conclusion

This paper presents a deterministic mathematical model for gaining insights into the transmission dynamics of Chlamydia trachomatis in a population with condom usage and treatment as intervention strategies. The disease-free equilibrium (DFE) of the model (1) is locally asymptotically stable whenever the associated reproduction number is less than unity. Qualitative analysis of the model (1) show that it undergoes the phenomenon of backward bifurcation, where a stable DFE coexists with a stable endemic equilibrium. In a scenario of backward bifurcation, the effective control of Chlamydia is largely dependent on the initial sizes of the sub-population of the model. The epidemiological implication is that, the presence of backward bifurcation in the transmission dynamics of the disease makes its control extremely difficult. This phenomenon is shown to arise due to the reinfection of recovered individuals. In the absence of this phenomenon (i.e., where reinfection of recovered individuals does not occur), it is shown that the disease-free equilibrium of the model (1) is globally asymptotically stable whenever the associated reproduction number is than unity. Further, it is shown that the unique endemic equilibrium of the model exists whenever the reproduction number is greater than unity. Results from the sensitivity analysis of the model, using the reproduction number, $R_c$ show that the top parameters that largely drive the dynamics of Chlamydia in the population are the efficacy of condoms, condom compliance, a fraction of treated individuals who recover due to effective treatment and treatment rate. Numerical simulations of the model suggest that infected persons after treatment should wait for at least 7 days before engaging in any form of sexual activity or, if not possible use condoms correctly (to avoid reinfection) in order to effectively control the spread of the disease in the population.

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## Competing interests

The authors declare that they have no financial or competing interests.

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