

A Model of the Spread of Chlamydia Trachomatis

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Abstract

Chlamydia as sexually transmitted disease that has major occurrence in sub-Saharan African where Nigeria is predominant this create a necessity to be concern about its spread within the nation. The SEScITR model with six compartments (Susceptible, Exposed, Screened, Infectious, Treated, Recovered) of human population was formulated. The parameters in the model were obtained from literatures and some were assumed. The ordinary differential equations were obtained using Runge Kutta Fehlberg Method and analysis of our system of equations was done using Maple 2017. Simulated data obtained through RStudio were used and our analysis were done using deSolve package on Rstudio. The formulated model was further analyse to get the reproduction number which is 0.67. The local and global stability of SEScITR model was investigated using Jacobian matrix and Lyapunov function respectively and the results shows that Chlamydia disease free equilibrium is locally and globally asymptotically since $R_0 < 1$ i.e. $R_0 = 0.67$. Also, the endemic state of Chlamydia equilibrium is locally and globally stable when $R_0 > 1$.

Keywords: SEScITR model, Basic reproduction number, Infectious disease dynamics, Epidemiological modelling

INTRODUCTION

Chlamydia is one of the most common Sexually Transmitted Diseases (STD) known all over the world, Chukukere., Inyama .and Oname (2020). According to Chukwuma (2018) Chlamydia is a sexually transmitted disease caused by bacteria called Chlamydia and it is transmitted from infected persons to another through contact via vaginal, anal or oral sex.

Sexually Transmitted Diseases (STDs) are one of the health challenges all over the world. There is high incidence and prevalence rate of STDs in developing countries as against developed countries Adebowale, Titiloye, Fagbamigbe and Akinyemi(2013). Moreover, these diseases are prevalent in Sub-Saharan Africa where Nigeria is domicile. It is observed that across all the states of Nigeria as a nation, the pandemic is rampant among young people. One of the cause of pervasiveness of STDs among youth, was exposure to Western lifestyle which consequentially led to increasing sexual activity among young people Victor., Olawale, Gloria, Iche., and Kingsley(2015). According to Ananya Mandal (2014), STDs are infections that can be transmitted through sexual contact with an infected individual; however, it is evident that STDs are not only contract through sexual intercourse but also through blood transfusion, unsterilized sharp objects, mother to infant during pregnancy or childbirth, and many others. The spread of STD around the world is alarming as observed by Oluwole, Oyekanmi, Ogunyemi and Osanyin(2020), that over a million people acquired a sexually infection daily. Moreover, he went on to state that every year, there is an estimated 357 million new infections with one of the four STIs globally: Chlamydia (131 million), Gonorrhoea (78 million), Syphilis (5.6 million) or Trichomoniasis(143 million)

Chlymadia is one of STDs prevalent in Nigeria, Mawak, DasheAgabiand Panshak(2011) and observed that young people are more affected with women who started having sex early. Chlymadia have varied effects ranges from low sperm counts Okoro and Agbonlahor (2012), infertility in women, ectopic pregnancy, miscarriages Shah, Vaghela. Pandya and Shah(2022), urithrix, Pelvic Inflammatory diseases(PID) Herzog et. al (2012)etc on its victims. Mawak, DasheAgabiand Panshak(2011) noted that there are behaviours that put people at risk of contracting this disease such as the numbers of partners, an age under 25, years , cervical entropy, concurrent gonococcal infections, a history of sexually transmitted diseases, HIV seropositivity and seroconversion, the duration of prostitution, lack of condom use and age of onset of sexual activity.

This disease is often remain silent for a long time especially in females this heighten its spread and which has been observed to be on the increase in Nigeria Mawak, DasheAgabiand Panshak(2011)

MATERIALS AND METHODS

Chlamydia is one of public health challenges confronting Nigerians. In order to curb this problem, there is need to understand the mode of spread of this disease and this is achieved by developing a model. According to Garnett (2002) mathematical model serves several purposes in the understanding of Sexually Transmitted Diseases and their control. Mathematical modeling provides a framework to understand the transmission mechanism of these diseases and their spread. STDs modeling have enjoyed several approaches since the first model of infectious diseases was constructed by Kenmack and McKendrick (1927).

Literature review

Wilson(2004) presented a basic overview of the avenues available for fighting Chlamydia infection and described mathematical models developed to investigatchumoral immunity, cell-mediated immunity and within-host dynamics. Masoumeh, Dann,Ashkanand Graeme (2010) used partial differential equation model that allows spatio-temporal variation in the biological species. Chukukere, Inyama and Oname (2020) modelled Chlamydia trachomatis (CT) and Gonorrhea Codynamics, with optimal control analysis to study, analyze and assess the impact of targeted treatment for each of the diseases on their co-infection in a population.

Shah, Vaghela. Pandyaand Shah2(2022) proposed a model susceptible-exposed-infected – recover –susceptible (SEIRS)l among two population groups namely those who contact Chlamydia by sexual intercourse and those who were infected through unhygienic environment with five compartments using system of nonlinear ordinary differential equations

Formulation of a new model

The study formulates a model for the spread of Chlamydia among the people of reproductive age in Nigeria. With the objectives to determine its equilibrium point, the reproduction number and test for the stability of the model.

We present a new model that subdivides total human population at time t into Susceptible human at time t , ($S(t)$), Exposed human at time t , ($E(t)$), Screen human for the bacteria at time t , ($S_c(t)$), Infected humans at time t , ($I(t)$) Treatment of infected humans at time t , ($T(t)$), and Recovered at the end of treatment at time t , ($R(t)$). Hence, we have

$$N(t) = S(t) + E(t) + S_c(t) + I(t) + T(t) + R(t)$$

The definition of the parameters are as stated in the table 1 and the diagrammatic flow is in fig 1

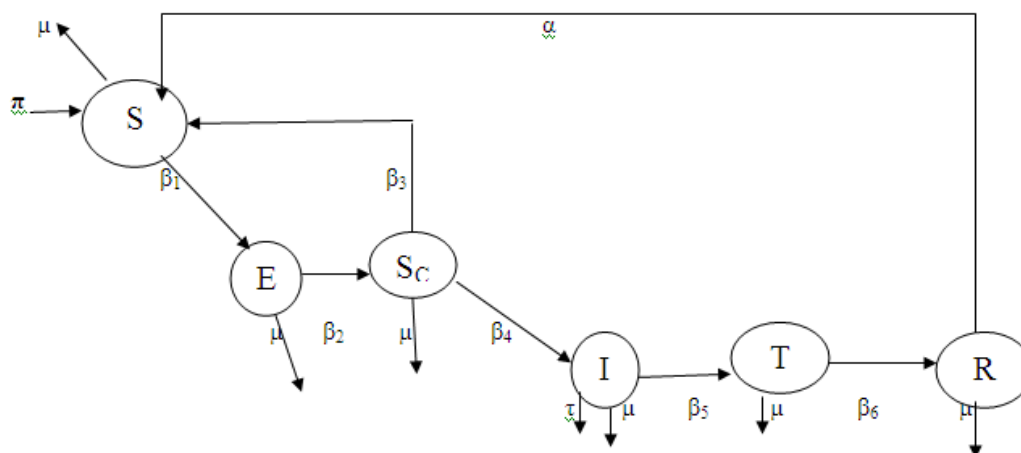


Figure: Flow diagram of SEScITR model

Table 1 Parameter symbols, descriptions, values and sources used in the model

Parameters	Descriptions	values	Source
π	Rate of entrance	0.018	Reference
β_1	Rate of transmission from Susceptible	0.4	Adjusted Reference
β_2	Rate of transmission from exposed to screening	0.01	Assumed
β_3	Rate of retuning to susceptible when discovered not infected	0.02	Assumed
β_4	Rate of transmission from screeningto infected	0.02	Assumed
β_5	Rate of transmission from infected to treated	0-01	Assumed
β_6	Rate of recoveryfrom treated to recovered	0.02	Assumed
τ	Rate of disease induced death	0.1	Assumed
α	Rate of retuning to susceptible	0.5	Assumed
μ	Escape rate	0.01	Reference

The model nonlinear equations are as stated below

$$S'(t) = \pi - \beta_1 SI - \mu S + \alpha R + \beta_3 S_C$$

$$E'(t) = \beta_1 SI - \mu E - \beta_2 E$$

$$S'_C(t) = E\beta_2 - \beta_3 S_C - \beta_4 S_C - \mu S_C$$

$$I'(t) = \beta_4 S_C - \beta_5 I - \mu I - \tau I \quad (1)$$

$$T'(t) = \beta_5 I - \beta_6 T_T - \mu T_T$$

$$R(t) = \beta_6 T_T - \mu R - \alpha R$$

The Equilibrium Points

The six equations in (1) are non-linear so the existence of equilibrium points can be found by equating our rate of change with respect to time to zero and solve simultaneously.

$$\text{Set } \mathbf{S'(t)} = E'(t) = S'_C(t) = I'(t) = T'(t) = R'(t) = 0$$

However, in finding the Disease free equilibrium point (DFE) all compartment are zero except S(t)

Then,

$$S = \frac{\pi}{\lambda + \mu}$$

$$\text{Therefore, the DFE} = \left(\frac{\pi}{\lambda + \mu}, 0, 0, 0, 0 \right)$$

and the endemic equilibrium are as follows

$$S =$$

$$\frac{\pi + \alpha R + \beta_3 S_C (\mu + \beta_2) (\beta_3 + \beta_4 + \mu) (\mu + \beta_2) (\beta_3 + \beta_4 + \mu) (\beta_5 + \mu + \tau) (\beta_5 + \mu) (\mu + \alpha) (\mu + \beta_2) (\beta_3 + \beta_4 + \mu) (\mu + \beta_2) (\beta_3 + \beta_4 + \mu) (\beta_5 + \mu + \tau) (\beta_5 + \mu) (\mu + \alpha) [\mu \beta_2 \beta_4 \beta_5 + \beta_3 \mu \beta_2]}{(\mu + \mu) (\mu + \beta_2) (\mu + \mu) (\beta_3 + \beta_4 + \mu) (\mu + \beta_2) (\beta_3 + \beta_4 + \mu) (\beta_5 + \mu + \tau) (\beta_5 + \mu) (\mu + \alpha) (\mu + \beta_2) (\beta_3 + \beta_4 + \mu) (\beta_5 + \mu + \tau) (\beta_5 + \mu) (\mu + \alpha) (\mu + \mu)}$$

$$E = \frac{\lambda \mu}{(\mu + \beta_2) (\lambda + \mu)}$$

$$S_C = \frac{\beta_2 E}{(\beta_3 + \beta_4 + \mu)} = \frac{\lambda \mu \beta_2}{(\mu + \beta_2) (\lambda + \mu) (\beta_3 + \beta_4 + \mu)}$$

$$I = \frac{\beta_4 S_C}{(\beta_5 - \mu - \tau)} = \frac{\lambda \mu \beta_2 \beta_4}{(\mu + \beta_2) (\beta_3 + \beta_4 + \mu) (\beta_5 + \mu + \tau)}$$

$$T_T = \frac{\beta_5 I}{(\beta_6 + \mu)} = \frac{\lambda \mu \beta_2 \beta_4 \beta_5}{(\mu + \beta_2)(\beta_3 + \beta_4 + \mu)(\beta_5 + \mu + \tau)(\beta_6 + \mu)}$$

$$R_0 = \frac{\beta_6 T_T}{(\mu + \alpha)} = \frac{\lambda \mu \beta_2 \beta_4 \beta_5 \beta_6}{(\mu + \beta_2)(\beta_3 + \beta_4 + \mu)(\beta_5 + \mu + \tau)(\beta_6 + \mu)(\mu + \alpha)}$$

Calculating The Reproduction ratio R_0

The Reproductive ratio (R_0) is the number of possible infection that can be produced by single infection in a susceptible population. There are a number of methods to calculate reproductive ratio but we are adopting next generation matrix Martcheva (2015). Thus,

the infected compartment $n =$

$$x_1 = E'(t) = \beta_1 SI - \mu E - \beta_2 E$$

$$x_2 = S'_C(t) = E\beta_2 - \beta_3 S_C - \beta_4 S_C - \mu S_C$$

$$x_3 = I'(t) = \beta_4 S_C - \beta_5 I - \mu I - \tau I$$

$$x_4 = T'(t) = \beta_5 I - \beta_6 T_T - \mu T_T$$

split the right hand side of the infected compartment

$$f_1 = \beta_1 SI \quad v_1 = -\mu E - \beta_2 E = -(\mu + \beta_2)E$$

$$f_2 = 0 \quad v_2 = E\beta_2 - \beta_3 S_C - \beta_4 S_C - \mu S_C = E\beta_2 - (\beta_3 + \beta_4 + \mu) S_C$$

$$f_3 = 0 \quad v_3 = \beta_4 S_C - \beta_5 I - \mu I - \tau I = \beta_4 S_C - (\beta_5 + \mu + \tau)I$$

$$f_4 = 0 \quad v_4 = \beta_5 I - \beta_6 T_T - \mu T_T = \beta_5 I - (\beta_6 + \mu)T_T$$

Using the Jacobian rule

$$F = \begin{pmatrix} \frac{df_1}{dE} & \frac{df_1}{dS_C} & \frac{df_1}{dI} & \frac{df_1}{dT_T} \\ \frac{df_2}{dE} & \frac{df_2}{dS_C} & \frac{df_2}{dI} & \frac{df_2}{dT_T} \\ \frac{df_3}{dE} & \frac{df_3}{dS_C} & \frac{df_3}{dI} & \frac{df_3}{dT_T} \\ \frac{df_4}{dE} & \frac{df_4}{dS_C} & \frac{df_4}{dI} & \frac{df_4}{dT_T} \end{pmatrix} = \begin{pmatrix} 0 & 0 & \beta_1 S & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}$$

$$V = \begin{pmatrix} \frac{dv1}{dE} & \frac{dv1}{dSC} & \frac{dv1}{dI} & \frac{dv1}{dT} \\ \frac{dv2}{dE} & \frac{dv2}{dSC} & \frac{dv2}{dI} & \frac{dv2}{dT} \\ \frac{dv3}{dE} & \frac{dv3}{dSC} & \frac{dv3}{dI} & \frac{dv3}{dT} \\ \frac{dv4}{dE} & \frac{dv4}{dSC} & \frac{dv4}{dI} & \frac{dv4}{dT} \end{pmatrix}$$

$$= \begin{pmatrix} (\mu + \beta_2) & 0 & 0 & 0 \\ -\beta_2 & (\beta_3 + \beta_4 + \mu) & 0 & 0 \\ 0 & -\beta_4 & (\beta_5 + \mu + \tau) & 0 \\ 0 & 0 & -\beta_5 & (\beta_6 + \mu) \end{pmatrix}$$

Then using maple 2017 the inverse is obtained as follows

$$V^{-1} = \begin{pmatrix} \frac{1}{\mu + \beta_2} & 0 & 0 & 0 \\ \frac{\beta_2}{(\mu + \beta_2)(\beta_3 + \beta_4 + \mu)} & \frac{1}{(\beta_3 + \beta_4 + \mu)} & 0 & 0 \\ \frac{\beta_2\beta_4}{(\mu + \beta_2)(\beta_3 + \beta_4 + \mu)(\beta_5 + \mu + \tau)} & \frac{\beta_4}{(\beta_3 + \beta_4 + \mu)(\beta_5 + \mu + \tau)} & \frac{1}{(\beta_5 + \mu + \tau)} & 0 \\ \frac{\beta_5\beta_4\beta_2}{(\mu + \beta_2)(\beta_3 + \beta_4 + \mu)(\beta_5 + \mu + \tau)(\beta_6 + \mu)} & \frac{\beta_5\beta_4}{(\beta_3 + \beta_4 + \mu)(\beta_5 + \mu + \tau)(\beta_6 + \mu)} & \frac{\beta_5}{(\beta_5 + \mu + \tau)(\beta_6 + \mu)} & \frac{1}{(\beta_6 + \mu)} \end{pmatrix}$$

Hence,

$$K = FV^{-1}$$

$$= \begin{pmatrix} \frac{\beta_1\beta_2\beta_4}{(\mu + \beta_2)(\beta_3 + \beta_4 + \mu)(\beta_5 + \mu + \tau)} & \frac{\beta_1\beta_4}{(\beta_3 + \beta_4 + \mu)(\beta_5 + \mu + \tau)} & \frac{\beta_1\beta_5}{(\beta_5 + \mu + \tau)} & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}$$

and $R_0 = \rho(K)$, which is the dominant eigen value of K , hence eigen values of K_s gotten using maple 2017 which is stated below

$$\begin{pmatrix} 0 \\ 0 \\ 0 \\ \frac{\beta_1\beta_2\beta_4}{(\tau\mu_2 + \tau\mu\beta_2 + \tau\mu\beta_3 + \tau\mu\beta_4 + \tau\beta_2\beta_3 + \tau\beta_2\beta_4 + \mu_3 + \mu_2\beta_2 + \mu_2\beta_3 + \mu_2\beta_4 + \mu_2\beta_5 + \mu\beta_2\beta_3 + \mu\beta_2\beta_4 + \mu\beta_2\beta_5 + \mu\beta_3\beta_5 + \mu\beta_4\beta_5 + \beta_2\beta_3\beta_5 + \beta_2\beta_4\beta_5)} \end{pmatrix}$$

Thus, the reproduction ratio is the largest of the eigen values and is stated below.

$$R_0 = \frac{\beta_1 \beta_2 \beta_4}{(\tau\mu_2 + \tau\mu\beta_2 + \tau\mu\beta_3 + \tau\mu\beta_4 + \tau\beta_2\beta_3 + \tau\beta_2\beta_4 + \mu_2 + \mu_2\beta_2 + \mu_2\beta_3 + \mu_2\beta_4 + \mu_2\beta_5 + \mu\beta_2\beta_3 + \mu\beta_2\beta_4 + \mu\beta_2\beta_5 + \mu\beta_3\beta_5 + \mu\beta_4\beta_5 + \beta_2\beta_3\beta_5 + \beta_2\beta_4\beta_5)}$$

We can simplify the reproduction ratio to be

$$R_0 = \frac{\beta_1 \beta_2 \beta_4}{(\mu + \beta_2)(\beta_3 + \beta_4 + \mu)(\beta_5 + \mu + \tau)}$$

Local stability Test of Chlamydia free equilibrium

The Chlamydia free equilibrium of the model equations (1) using basic reproduction number R_0 in the theorem below:

Theorem 1. The stability of equations (1) to be locally asymptotically stable at disease free

equilibrium (DFE) = $(\frac{\pi}{\lambda + \mu}, 0, 0, 0, 0, 0)$ if $R_0 < 1$ otherwise it is not stable.

Proof

The local stability of the disease free equilibrium of the systems of equations (1) will be analysed using the Jacobian matrix (J) is given by $|J - \xi I| = 0$, where I is an identity matrix

The Jacobian of equation (1) is

$$J = \begin{pmatrix} -(\lambda + \mu) & 0 & 0 & 0 & 0 & 0 \\ \lambda & -(\beta_2 + \mu) & 0 & 0 & 0 & 0 \\ 0 & \beta_2 & -(\beta_3 + \beta_4 + \mu) & 0 & 0 & 0 \\ 0 & 0 & \beta_4 & -(\beta_5 + \tau + \mu) & 0 & 0 \\ 0 & 0 & 0 & \beta_5 & -(\beta_6 + \mu) & 0 \\ 0 & 0 & 0 & 0 & \beta_6 & -(\alpha + \mu) \end{pmatrix}$$

$$I = \begin{pmatrix} 1 & 0 & 0 & 0 & 0 & 0 \\ 0 & 1 & 0 & 0 & 0 & 0 \\ 0 & 0 & 1 & 0 & 0 & 0 \\ 0 & 0 & 0 & 1 & 0 & 0 \\ 0 & 0 & 0 & 0 & 1 & 0 \\ 0 & 0 & 0 & 0 & 0 & 1 \end{pmatrix}$$

$$|J - \xi I| =$$

$$\begin{vmatrix} -(\lambda + \mu) - \xi & 0 & 0 & 0 & 0 & 0 \\ \lambda & -(\beta_2 + \mu) - \xi & 0 & 0 & 0 & 0 \\ 0 & \beta_2 & -(\beta_3 + \beta_4 + \mu) - \xi & 0 & 0 & 0 \\ 0 & 0 & \beta_4 & -(\beta_5 + \tau + \mu) - \xi & 0 & 0 \\ 0 & 0 & 0 & \beta_5 & -(\beta_6 + \mu) - \xi & 0 \\ 0 & 0 & 0 & 0 & \beta_6 & -(\alpha + \mu) - \xi \end{vmatrix}$$

Using maple 2017 the Eigen values are as follows:

$$\begin{bmatrix} -\lambda - \mu - \xi \\ -\beta_6 - \mu - \xi \\ -\beta_3 - \beta_4 - \mu - \xi \\ -\mu - \beta_2 - \xi \\ -\alpha - \mu - \xi \\ -\beta_5 - \mu - \tau - \xi \end{bmatrix}$$

Each of the six eigen values have negative real part which implies that $R_0 < 1$

Moreover, the trace and determinant respectively are as follows

$$-\lambda - 6\mu - 6\xi - \beta_2 - \beta_3 - \beta_4 - \beta_5 - \tau - \beta_6 - \alpha < 0 \text{ and}$$

$$[(-\lambda - \mu - \xi)(-\mu - \beta_2 - \xi)(-\beta_3 - \beta_4 - \mu - \xi)(-\beta_5 - \mu - \tau - \xi)(-\beta_6 - \mu - \xi)(-\alpha - \mu - \xi)] > 0$$

Which also implies that $R_0 < 1$ and therefore indicates that the diseases free equilibrium is stable

Endemic equilibrium

The endemic equilibrium is the point where a disease persists in a given population for a certain time. This state depends on the term already defined called Reproduction ratio. (R_0) when it is greater than $R_0 > 1$. We approach this by obtain the Jacobian of equation (1), at the endemic equilibrium.

Proof

The local stability of the endemic equilibrium of the systems of equations (1) will be analysed using the Jacobian matrix (J) is given by $|J - \xi I| = 0$, where I is an identity matrix

The Jacobian of equation (1) is

$$J = \begin{pmatrix} -(\lambda + \mu) & 0 & \beta_3 & 0 & 0 & \alpha \\ \lambda & -(\beta_2 + \mu) & 0 & 0 & 0 & 0 \\ 0 & \beta_2 & -(\beta_3 + \beta_4 + \mu) & 0 & 0 & 0 \\ 0 & 0 & \beta_4 & -(\beta_5 + \tau + \mu) & 0 & 0 \\ 0 & 0 & 0 & \beta_5 & -(\beta_6 + \mu) & 0 \\ 0 & 0 & 0 & 0 & \beta_6 & -(\alpha + \mu) \end{pmatrix}$$

$$I = \begin{pmatrix} 1 & 0 & 0 & 0 & 0 & 0 \\ 0 & 1 & 0 & 0 & 0 & 0 \\ 0 & 0 & 1 & 0 & 0 & 0 \\ 0 & 0 & 0 & 1 & 0 & 0 \\ 0 & 0 & 0 & 0 & 1 & 0 \\ 0 & 0 & 0 & 0 & 0 & 1 \end{pmatrix}$$

$$|J - \xi I| =$$

$$\begin{pmatrix} -(\lambda + \mu) - \xi & 0 & \beta_3 & 0 & 0 & \alpha \\ \lambda & -(\beta_2 + \mu) - \xi & 0 & 0 & 0 & 0 \\ 0 & \beta_2 & -(\beta_3 + \beta_4 + \mu) - \xi & 0 & 0 & 0 \\ 0 & 0 & \beta_4 & -(\beta_5 + \tau + \mu) - \xi & 0 & 0 \\ 0 & 0 & 0 & \beta_5 & -(\beta_6 + \mu) - \xi & 0 \\ 0 & 0 & 0 & 0 & \beta_6 & -(\alpha + \mu) - \xi \end{pmatrix}$$

Expanding the first and last column we can see that the characteristics equation have two roots

$$\xi_1 = -\lambda - \mu; \xi_2 = -\alpha - \mu.$$

The remaining roots are solution to the equation

$$\begin{vmatrix} -(\beta_2 + \mu) - \xi & 0 & 0 & 0 \\ \beta_2 & -(\beta_3 + \beta_4 + \mu) - \xi & 0 & 0 \\ 0 & \beta_4 & -(\beta_5 + \tau + \mu) - \xi & 0 \\ 0 & 0 & \beta_5 & -(\beta_6 + \mu) - \xi \end{vmatrix} = 0$$

Using maple 2017 the remaining eigen values are as follows

$$\begin{bmatrix} -\beta_6 - \mu - \xi \\ -\beta_3 - \beta_4 - \mu - \xi \\ -\mu - \beta_2 - \xi \\ -\beta_5 - \mu - \tau - \xi \end{bmatrix}$$

The determinant is as follows

$$-(-\mu - \beta_2 - \xi) (\beta_3 + \beta_4 + \mu + \xi) (\beta_5 + \mu + \tau + \xi) (\beta_6 + \mu + \xi)$$

Our approach is having expanded these equations we can show that it has real parts we will do this by applying the Routh–Hurwitz criterion.

Theorem 2 (Routh–Hurwitz Criteria). *Consider the n th-degree polynomial with real constant coefficients*

$$P(\lambda) = \lambda^n + a_1 \lambda^{n-1} + \dots + a_{n-1} \lambda + a_n.$$

which states that all the roots of a polynomial will have negative real parts if and only if all the coefficients a_i are positive. The constant term a_n gives the reproduction number, the Routh

Hurwitz Conditions is as stated below:

1. Trace $J < 0$
2. Determinant > 0

The trace of matrix above is

$$-4\mu - \beta_2 - 4\xi - \beta_3 - \beta_4 - \beta_5 - \tau - \beta_6 < 0$$

And the determinant

$$-(-\mu - \beta_2 - \xi) (\beta_3 + \beta_4 + \mu + \xi) (\beta_5 + \mu + \tau + \xi) (\beta_6 + \mu + \xi) > 0$$

Expanding the determinant we have

$$\begin{aligned} & \tau \xi^2 + 3\tau \xi^2 \mu + \tau \xi^2 \beta_2 + \tau \xi^2 \beta_3 + \tau \xi^2 \beta_4 + 3\tau \xi \mu^2 + 2\tau \xi \mu \beta_2 + 2\tau \xi \mu \beta_3 + 2\tau \xi \mu \beta_4 + 2\tau \xi \mu \beta_6 + \tau \xi \beta_2 \beta_3 + \tau \xi \beta_2 \beta_4 \\ & + \tau \xi \beta_2 \beta_6 + \tau \xi \beta_3 \beta_6 + \tau \xi \beta_4 \beta_6 + \tau \mu^3 + \tau \mu^2 \beta_2 + \tau \mu^2 \beta_3 + \tau \mu^2 \beta_4 + \tau \mu^2 \beta_6 + \tau \mu \beta_2 \beta_3 + \tau \mu \beta_2 \beta_4 + \tau \mu \beta_2 \beta_6 + \\ & \tau \mu \beta_3 \beta_6 + \tau \mu \beta_4 \beta_6 + \tau \beta_2 \beta_3 \beta_6 + \tau \beta_2 \beta_4 \beta_6 + \xi^4 + 4\xi^3 \mu + \xi^3 \beta_2 + \xi^3 \beta_3 + \xi^3 \beta_4 + \xi^3 \beta_5 + \xi^3 \beta_6 + 6\xi^2 \mu^2 + 3\xi^2 \mu \beta_2 \\ & + 3\xi^2 \mu \beta_3 + 3\xi^2 \mu \beta_4 + 3\xi^2 \mu \beta_5 + 3\xi^2 \mu \beta_6 + \xi^2 \beta_2 \beta_3 + \xi^2 \beta_2 \beta_4 + \xi^2 \beta_2 \beta_5 + \xi^2 \beta_2 \beta_6 + \xi^2 \beta_3 \beta_5 + \xi^2 \beta_3 \beta_6 + \xi^2 \beta_4 \beta_5 \\ & + \xi^2 \beta_4 \beta_6 + \xi^2 \beta_5 \beta_6 + 4\xi \mu^3 + 3\xi \mu^2 \beta_2 + 3\xi \mu^2 \beta_3 + 3\xi \mu^2 \beta_4 + 3\xi \mu^2 \beta_5 + 3\xi \mu^2 \beta_6 + 2\xi \mu \beta_2 \beta_3 + 2\xi \mu \beta_2 \beta_4 + 2\xi \mu \beta_2 \beta_6 \end{aligned}$$

$$\begin{aligned} & \beta_5 + 2\xi\mu\beta_2 \beta_6 + 2\xi\mu\beta_3 \beta_5 + 2\xi\mu\beta_3 \beta_6 + 2\xi\mu\beta_4 \beta_5 + 2\xi\mu\beta_4 \beta_6 + 2\xi\mu\beta_5 \beta_6 + \xi\beta_2 \beta_3 \beta_5 + \xi\beta_2 \beta_3 \beta_6 + \xi\beta_2 \\ & \beta_4\beta_5 + \xi\beta_2 \beta_4\beta_6 + \xi\beta_2 \beta_5\beta_6 + \xi\beta_3 \beta_5\beta_6 + \xi\beta_4 \beta_5\beta_6 + \mu^4 + \mu^3 \beta_2 + \mu^3 \beta_3 + \mu^3 \beta_4 + \mu^3 \beta_5 + \mu^3 \beta_6 + \mu^2 \beta_2 + \mu^2 \beta_3\beta_3 \\ & + \mu^2 \beta_2\beta_4 + \mu^2 \beta_2\beta_5 + \mu^2 \beta_2\beta_6 + \mu^2 \beta_3\beta_5 + \mu^2 \beta_3\beta_6 + \mu^2 \beta_4\beta_5 + \mu^2 \beta_4\beta_6 + \mu^2 \beta_5\beta_6 + \mu\beta_2 \beta_3 \beta_5 + \mu\beta_2 \beta_3 \beta_6 + \mu\beta_2 \\ & \beta_4\beta_5 + \mu\beta_2 \beta_4\beta_6 + \mu\beta_2 \beta_5\beta_6 + \mu\beta_3 \beta_5\beta_6 + \mu\beta_4 \beta_5\beta_6 + \beta_2 \beta_3 \beta_5\beta_6 + \beta_2 \beta_4\beta_5\beta_6 \end{aligned}$$

=0

This leads to the following cubic equation in ξ

$$P(\xi) := \xi^4 + a_1\xi^3 + a_2\xi^2 + a_3\xi + a_4 = 0,$$

$$a_1 = \tau + 4\mu + \beta_2 + \beta_3 + \beta_4 + \beta_5 + \beta_6$$

$$a_2 = 3\tau\mu + \tau\beta_2 + \tau\beta_3 + \tau\beta_4 + 6\mu^2 + 3\mu\beta_2 + 3\mu\beta_3 + 3\mu\beta_4 + 3\mu\beta_5 + 3\mu\beta_6 + \beta_2\beta_3 + \beta_2\beta_4 + \beta_2\beta_5 + \beta_2\beta_6 + \beta_3\beta_5 + \beta_3\beta_6 + \beta_4\beta_5 + \beta_4\beta_6 + \beta_5\beta_6$$

$$a_3 = 3\tau\mu^2 + 2\tau\mu\beta_2 + 2\tau\mu\beta_3 + 2\tau\mu\beta_4 + 2\tau\mu\beta_5 + 2\tau\mu\beta_6 + \tau\beta_2\beta_3 + \tau\beta_2\beta_4 + \tau\beta_2\beta_5 + \tau\beta_2\beta_6 + \tau\beta_3\beta_5 + \tau\beta_3\beta_6 + 4\mu^3 + 3\mu^2\beta_2 + 3\mu^2\beta_3 + 3\mu^2\beta_4 + 3\mu^2\beta_5 + 3\mu^2\beta_6 + 2\mu\beta_2\beta_3 + 2\mu\beta_2\beta_4 + 2\mu\beta_2\beta_5 + 2\mu\beta_2\beta_6 + 2\mu\beta_3\beta_5 + 2\mu\beta_3\beta_6 + 2\mu\beta_4\beta_5 + 2\mu\beta_4\beta_6 + 2\mu\beta_5\beta_6 + \beta_2\beta_3\beta_5 + \beta_2\beta_3\beta_6 + \beta_2\beta_4\beta_5 + \beta_2\beta_4\beta_6 + \beta_2\beta_5\beta_6 + \beta_3\beta_5\beta_6 + \beta_4\beta_5\beta_6$$

$$a_4 = \mu^4 + \mu^3\beta_2 + \mu^3\beta_3 + \mu^3\beta_4 + \mu^3\beta_5 + \mu^3\beta_6 + \mu^2\beta_2 + \mu^2\beta_2\beta_3 + \mu^2\beta_2\beta_4 + \mu^2\beta_2\beta_5 + \mu^2\beta_2\beta_6 + \mu^2\beta_3\beta_5 + \mu^2\beta_3\beta_6 + \mu^2\beta_4\beta_5 + \mu^2\beta_4\beta_6 + \mu^2\beta_5\beta_6 + \mu\beta_2\beta_3\beta_5 + \mu\beta_2\beta_3\beta_6 + \mu\beta_2\beta_4\beta_5 + \mu\beta_2\beta_4\beta_6 + \mu\beta_2\beta_5\beta_6 + \mu\beta_3\beta_5\beta_6 + \mu\beta_3\beta_6 + \mu\beta_4\beta_5 + \mu\beta_4\beta_6 + \mu\beta_5\beta_6 + \tau\mu^3 + \tau\mu^2\beta_2 + \tau\mu^2\beta_3 + \tau\mu^2\beta_4 + \tau\mu^2\beta_5 + \tau\mu^2\beta_6 + \tau\mu\beta_2\beta_3 + \tau\mu\beta_2\beta_4 + \tau\mu\beta_2\beta_5 + \tau\mu\beta_2\beta_6 + \tau\mu\beta_3\beta_5 + \tau\mu\beta_3\beta_6 + \tau\mu\beta_4\beta_5 + \tau\mu\beta_4\beta_6 + \tau\mu\beta_5\beta_6 + \tau\beta_2\beta_3\beta_5 + \tau\beta_2\beta_3\beta_6 + \tau\beta_2\beta_4\beta_5 + \tau\beta_2\beta_4\beta_6 + \tau\beta_2\beta_5\beta_6 + \tau\beta_3\beta_5\beta_6 + \tau\beta_3\beta_6 + \tau\beta_4\beta_5 + \tau\beta_4\beta_6 + \tau\beta_5\beta_6$$

The sufficient condition for the roots of the polynomial $P(\xi)$ to be negative or have negative real part is that all coefficients be strictly positive. Therefore, since $a_1 > 0$, $a_2 > 0$, $a_3 > 0$, $a_4 > 0$ and implies that the Eigen values have negative real parts.

Thus, we conclude that Chlamydia endemic equilibrium is asymptotical stable

Global stability Test of Chlamydia Disease free equilibrium

The global stability analysis of Chlamydia Disease free equilibrium will be carried out using Lyapunov function. Lyapunov functions are scalar functions that may be used to prove the global stability of an equilibrium.

Theorem 3: if a function $L(t)$ is globally positively definite and radically unbounded and its time derivative globally negative $L'(t) < 0$ for all $t \neq t^*$ then the equilibrium t^* is globally stable

Proof;

Let's consider Lyapunov functions defined as follow;

$$L_1(t) = S(t) + E(t) + S_C(t) + I(t) + T(t) + R(t) = 0 \text{ at DFE}$$

Differentiating the above, we have

$$L'_1(t) = S'(t) + E'(t) + S'_C(t) + I'(t) + T'(t) + R'(t)$$

Substituting, we have

$$L'_1(t) = \pi - \beta_1 SI - \mu S + \alpha R + \beta_3 S_C + \beta_1 SI - \mu E - \beta_2 E + E\beta_2 - \beta_3 S_C - \beta_4 S_C - \mu S_C + \beta_4 S_C - \beta_5 I - \mu I - \tau I + \beta_5 I - \beta_6 T_T - \mu T_T + \beta_6 T_T - \mu R - a R$$

$$= \pi - \mu S - \mu E - \mu S_C - \mu I - \tau I - \mu T_T - \mu R$$

$$= \pi - \mu(S + E + S_C + T_T + R) - (\mu + \tau)I$$

$$L'_1(t) < 0$$

Therefore, by Lyapunov theorem the disease-free equilibrium is globally asymptotically stable.

Global stability Test of Chlamydia endemic equilibrium

The endemic equilibrium is unique globally stable whenever it exists where $R_0 > 1$ but unstable otherwise. The model solution will be analysed in the interior of the feasible region $\mathcal{A}$, which will converge at endemic equilibrium whenever $R_0 > 1$. The chlamydia bacteria will spread out and continue in the population.

Theorem 4 The endemic equilibrium point is globally asymptotically stable in the feasible region $\mathcal{A}$.

Proof

Consider the Lyapunov function defined as follows

$$L_2(t) = \frac{1}{2} [(S(t) - S^*) + (E(t) - E^*) + (S_C(t) - S_C^*) + (I(t) - I^*) + (T(t) - T^*) + (R(t) - R^*)]^2$$

Thus, differentiating the function above we have,

$$L'_2(t) = [(S(t) - S^*) + (E(t) - E^*) + (S_C(t) - S_C^*) + (I(t) - I^*) + (T(t) - T^*) + (R(t) - R^*)][S'(t) + E'(t) + S'_C(t) + I'(t) + T'(t) + R'(t)]$$

$$= [(S(t) - S^*) + (E(t) - E^*) + (S_C(t) - S_C^*) + (I(t) - I^*) + (T(t) - T^*) + (R(t) - R^*)][\pi - \mu(S + E + S_C + T_T + R) - (\mu + \tau)I]$$

$$\text{Then putting } \pi = \mu S^* + \mu E^* + \mu S_C^* + \mu I^* + \mu T_T^* + \mu R^*)$$

$$= [(S(t) - S^*) + (E(t) - E^*) + (S_c(t) - S_c^*) + (I(t) - I^*) + (T(t) - T^*) + (R(t) - R^*)] [-\mu(S(t) - S^*) + (E(t) - E^*) + (S_c(t) - S_c^*) + (I(t) - I^*) + (T(t) - T^*) + (R(t) - R^*)I]$$

$$= [-\mu(S(t) - S^*) + (E(t) - E^*) + (S_c(t) - S_c^*) + (I(t) - I^*) + (T(t) - T^*) + (R(t) - R^*)I]^2 \leq 0$$

Hence, the endemic equilibrium is globally asymptotically stable.

RESULTS AND DISCUSSION

Numerical Simulation

Using the values in Table 1 we carried out the numerical simulation for the model, R software was adopted to simulate our data and the results are presented in the following sections.

Results of Simulation

This section simulation was achieved using R-studio software and a package known as deSolve package in R-studio was used to solve the ordinary differential equation (ODE) using the system of linear equations in (2) couple with the set of parameter values in table 1 with the following initial condition $S(0) = 9999, E(0) = 1, S_c(0) = 0, I(0) = 0, T(0) = 0, R(0) = 0$. The equilibrium point i.e ($R_0 < 1$) and endemic equilibrium point i.e ($R_0 > 1$) for SESCITR model was investigated using our results from the parameter and initial conditions

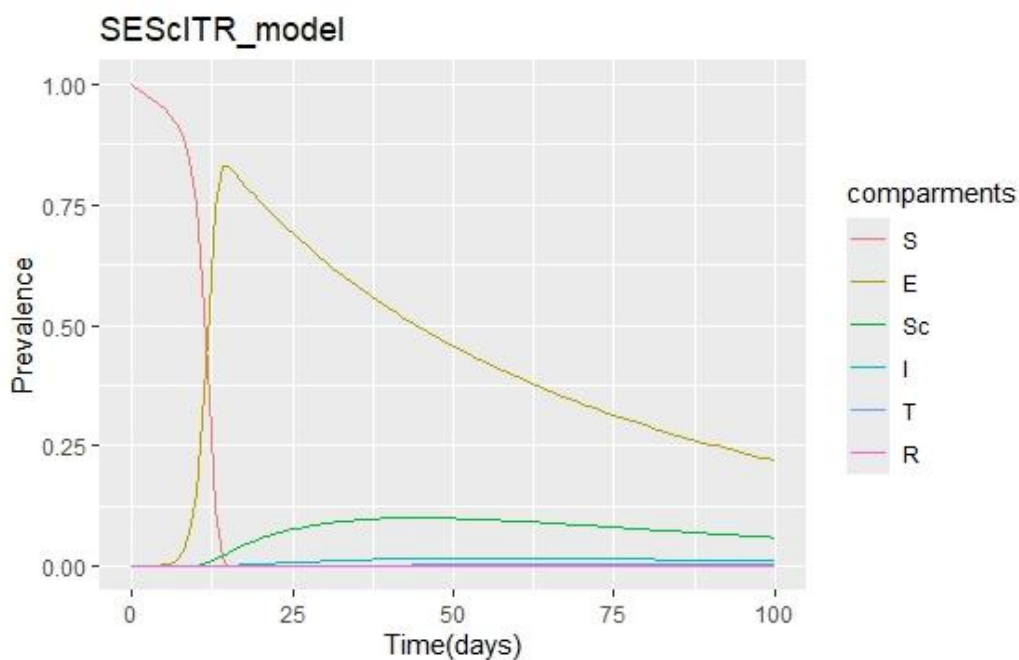


Fig .2 Showing general behaviour of SESCITR model

The graph in Fig 2 illustrates the dynamics of a Susceptible-Exposed-Screened-Infected-Treated-the graph shows the progression of an infection and the impact of screening and treatment over time. The rapid initial spread is brought under control through screening and treatment, leading to stabilization of the infection rates. The low levels of recovered individuals suggest that recovery is slow or that the duration of the model is insufficient to show significant recovery. Overall, the model highlights the importance of early intervention to control the spread of infection. It is also useful for understanding the temporal progression of disease and the effectiveness of intervention strategies in a population

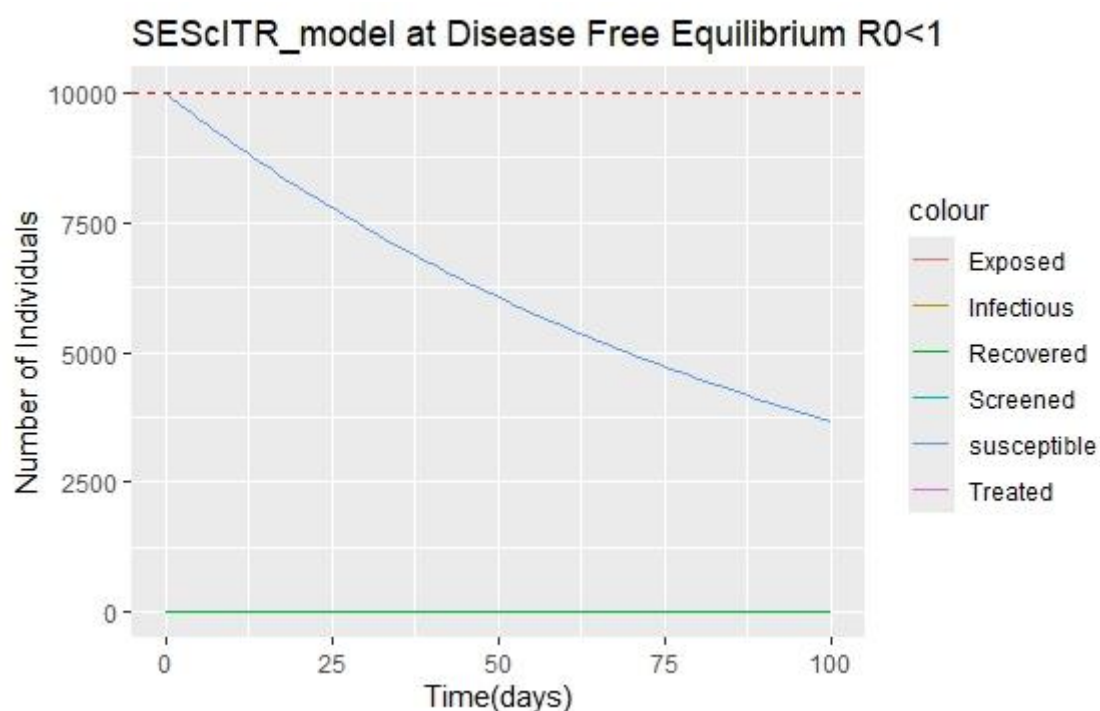


Fig. 3 Disease Free Equilibrium state of SEScITR

In fig 3, we saw that at the start (time = 0), the entire population is susceptible, with no individuals in the exposed, infectious, recovered, screened, or treated compartments. The susceptible population starts at the total population (around 10,000 individuals). Over the 100 days, the number of susceptible individuals steadily decreases. This decline indicates that individuals are being moved out of the susceptible compartment, likely due to infection, screening, or treatment interventions. The graph demonstrates that under the given conditions, the SEScITR model at disease-free equilibrium with $R_0 < 1$, successfully maintains the population largely free of the disease. The susceptible population decreases over time due to effective screening and treatment measures, ensuring that the exposed,

infectious, recovered, and treated compartments remain insignificant. This scenario illustrates successful disease control and eventual eradication within the simulated timeframe.

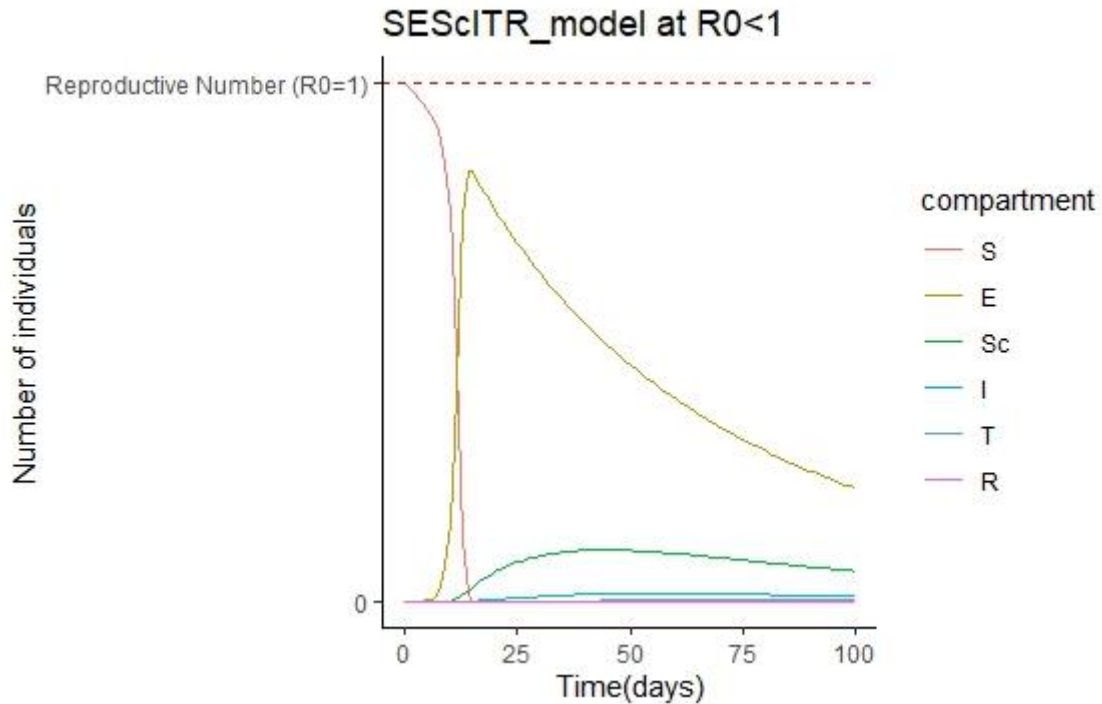


Fig 4 SESCOITR Model when $R_0 < 1$

The SESCOITR model (fig 4) simulation demonstrates that with $R_0 < 1$, the disease can be effectively controlled over time. The red dashed line at $R_0=1$ is a crucial reference indicating that the disease's reproductive number is below the critical threshold necessary for sustained transmission. Initial exposure occurs rapidly, but the number of infectious individuals remains low due to effective screening and treatment interventions. The susceptible population decreases as individuals are exposed or screened, leading to a gradual increase in recovered individuals. The model illustrates the successful control of the disease, preventing large-scale outbreaks and maintaining low levels of infection throughout the 100-day period.

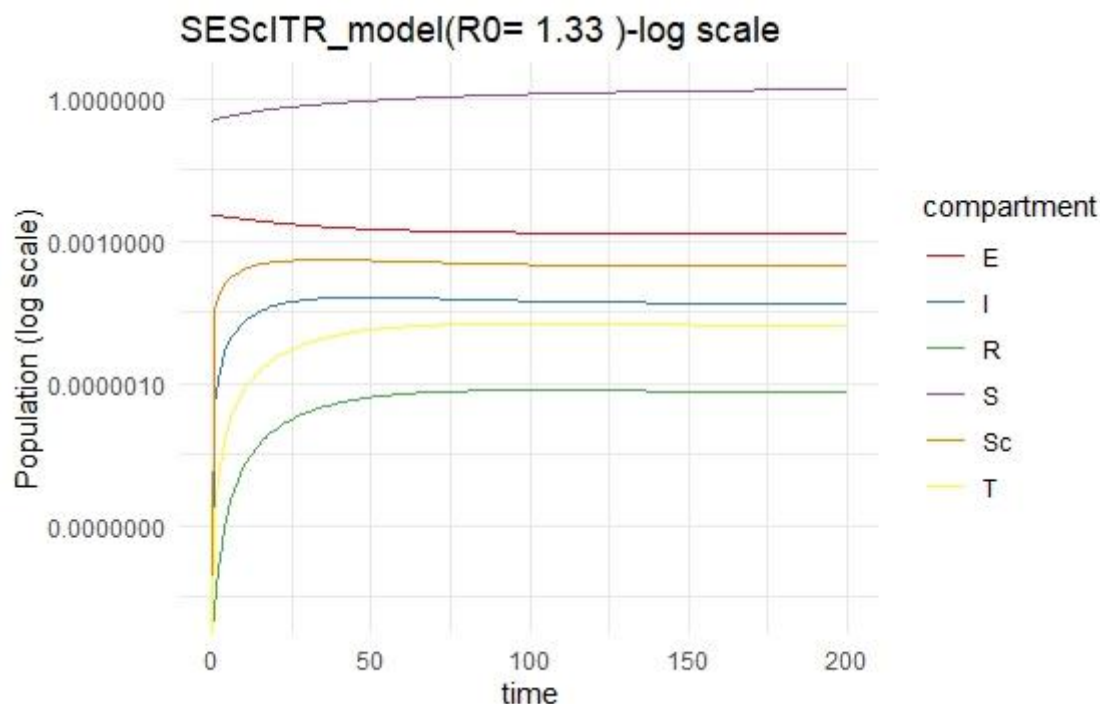


Fig 5 SEScITR Model when $R_0 > 1$

The graph in fig 5 shows that we adjusted the transmission rate β_1 equal to 0.8 which make the reproductive number $R_0=1.33$ for us to see how the model will be at endemic equilibrium. The SEScITR model with $R_0=1.33$ demonstrates the persistence of the disease in the population. The exposed and infectious compartments stabilize at relatively high levels, indicating ongoing transmission. Screening and treatment efforts are visible but insufficient to eradicate the disease. The susceptible population decreases, while the recovered population increases, showing that while recovery occurs, new infections continue to happen. The log scale allows for a detailed view of the dynamics, especially for smaller populations, highlighting the importance of sustained intervention to reduce R_0 below 1 for effective disease control.

CONCLUSION

This research work formulated a new model to study the spread of chlamydia among people of reproductive age in Nigeria by improving on existing model incorporating additional two compartments namely screened and treatment. The SEScITR model (Susceptible, Exposed, Screened, Infectious, Treated, Recovered) was analysed to determine the reproduction number and under various conditions to understand disease

dynamics in a population We examined scenarios where the basic reproduction number R_0 was less than 1 and greater than 1. The simulations ran over periods of 100 to 200 days and depicted the transition of individuals through different compartments of the model.

This study obtained the reproduction number for SEScITR model using the next generation matrix as follows:

$$R_0 = \frac{\beta_1 \beta_2 \beta_4}{(\mu + \beta_2)(\beta_3 + \beta_4 + \mu)(\beta_5 + \mu + \tau)}$$

The above, state the reproduction number of infections among human population that are susceptible which stand for secondary infection a human will produce in an entirely susceptible population in its life span.

The local and global stability of SEScITR model was investigated using Jacobian matrix and Lyapunov function respectively and the results shows that Chlamydia disease free equilibrium is locally and globally asymptotically since $R_0 < 1$ i.e. $R_0 = 0.67$. Also, the endemic state of Chlamydia equilibrium is locally and globally stable when $R_0 > 1$

Conflict of Interest: Not Applicable

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