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A Mathematical Model and Simulation of *Chlamydia trachomatis* in a Human Carrier

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Abstract

In this paper, a mathematical model describing the dynamics of *Chlamydia trachomatis* infection in a human carrier is presented. The model incorporated relevant feature such as recovery through drug administration. The existence and uniqueness of solutions of the model were examined by actual solution. We conduct local and global stability analysis for the model. The results show that it is stable under certain conditions. The system of equations were solved analytically using parameter-expanding method coupled with direct integration. The results are presented graphically and discussed. It is discovered that the influence of burst size per infected cell, rate of cell infection and recovery rate due to drug administration is quite significant.

Keywords: *Chlamydia*; *Chlamydia trachomatis*; sexually transmitted diseases (STDs); stability criteria.

1 Introduction

Chlamydia is an obligate intracellular bacterial pathogen that infects the genital and ocular mucosa of humans causing sexually transmitted disease (STD) and Trachoma. It is estimated that 70 – 75% of endocervical infections in women caused by bacterium. *Chlamydia trachomatis* are asymptomatic and may persist for months to years [1].

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According to Wilson [2], the disease due to invading *Chlamydia* commences as chlamydial particle infect epithelial cells of mucosal linings. However, the immune system responds via two mechanisms, such as humoral immunity and cell-mediated immunity. For more details on humoral immunity and cell-mediated immunity, see [2].

It has been suggested that cell-mediated immunity, not humoral immunity, plays the dominant role in protective immunity against *Chlamydia* [3]. The importance of cell-mediated immunity to chlamydial infection has been emphasized in a number of studies [4,5]. Wilson and McElwain [6] modelled humoral immunity against chlamydial challenge by tracking the antibody and host cell receptor aggregation over chlamydial particle. In another development, Wilson et al. [7] modelled the inter-conversion between infectious and replicating chlamydial particles to track the number of particles within a host cell with time over the developmental cycle.

It is a general belief that if a patient received treatment, he or she may recover and move to recovered class. This, we thought, may also be applicable to the infected epithelial cells of mucosal linings. As a result, we thought there are other factors and parameters which can be taken into consideration during the model development process of the disease. So, this present study investigates the criteria under which the rate of recovery of infected cells through drug administration could lead to the stability of equilibrium point.

2 Model Formulation

We modify the Wilson [2] model by incorporating recovery through drug administration. We let $C(t)$ be the concentration of free extracellular chlamydial particles, $E(t)$ be the number of uninfected mucosal epithelial cells (main host cell for *Chlamydia*), $I(t)$ be the number of *Chlamydia*-infected epithelial cells and $R(t)$ be the number of epithelial cells which recovered from *Chlamydia*-infection. Arising from the above, a simple mathematical description of the change in the interacting 'species' is:

$$\frac{dC}{dt} = P_C K_2 I - \mu_C C \quad (1)$$

$$\frac{dE}{dt} = P_E - \mu_E E - K_1 CE + \omega R \quad (2)$$

$$\frac{dI}{dt} = -K_2 I - \gamma I + K_1 CE - \mu_E I - \delta I \quad (3)$$

$$\frac{dR}{dt} = \delta I + \gamma I - \mu_E R - \omega R \quad (4)$$

As initial condition, we choose

$$C(0)=C_0, \quad E(0)=E_0, \quad I(0)=I_0, \quad R(0)=R_0, \quad (5)$$

where P_C is the number of Chlamydial particles released from infected cells, K_2 is the rate at which Chlamydia particles are released from infected cells, μ_C is the natural death rate of Chlamydial particles, P_E is the epithelial cells reproduction rate, μ_E is the natural death rate of epithelial cells, K_1 is the rate of Epithelial cell infection which may be reduced by antibodies, ω is the waning off Immunity, γ is the rate of clearance of infected cells due to cell-mediated immunity and δ is the recovery rate due to drug administration.

3 Method of Solution

3.1 Existence and Uniqueness of Solution

Theorem 1: Let $P_C = 1$, $\mu_E = \mu_C = \mu$. Then the equations (1) – (4) with initial conditions (5) has a unique solution for all $t \geq 0$.

Proof: Let $P_C = 1$, $\mu_E = \mu_C = \mu$ and $\phi(t) = C(t) + E(t) + I(t) + R(t)$, we obtain

$$\frac{d\phi}{dt} = P_E - \mu\phi, \quad \phi(0) = (C_0 + E_0 + I_0 + R_0) = \phi_0 \tag{6}$$

Using method of integrating factor, we obtain the solution of problem (6) as

$$\phi(t) = \frac{P_E}{\mu} (1 - e^{-\mu t}) + \phi_0 e^{-\mu t} \tag{7}$$

Then, we obtain

$$C(t) = \left(\frac{P_E}{\mu} + \left(\phi_0 - \frac{P_E}{\mu} \right) e^{-\mu t} \right) - (E(t) + I(t) + R(t)) \tag{8}$$

$$E(t) = \left(\frac{P_E}{\mu} + \left(\phi_0 - \frac{P_E}{\mu} \right) e^{-\mu t} \right) - (C(t) + I(t) + R(t)) \tag{9}$$

$$I(t) = \left(\frac{P_E}{\mu} + \left(\phi_0 - \frac{P_E}{\mu} \right) e^{-\mu t} \right) - (C(t) + E(t) + R(t)) \tag{10}$$

$$R(t) = \left(\frac{P_E}{\mu} + \left(\phi_0 - \frac{P_E}{\mu} \right) e^{-\mu t} \right) - (C(t) + E(t) + I(t)) \tag{11}$$

Hence, there exists a unique solution of problem (1) - (4). This completes the proof.

3.2 Stability Analysis

Our system of equations (1) – (4) has a trivial steady state:

$$\bar{C} = 0, \quad \bar{E} = \frac{P_E}{\mu_E}, \quad \bar{I} = 0, \quad \bar{R} = 0 \tag{12}$$

and a non-trivial steady state:

$$\left. \begin{aligned} \bar{C} &= \frac{\sigma(\alpha\mu_C\mu_E - K_1K_2P_C P_E)}{K_1\mu_C(\beta\omega - \sigma\alpha)} \\ \bar{E} &= \frac{\alpha\mu_C}{K_1K_2P_C} \\ \bar{I} &= \frac{\sigma(\alpha\mu_C\mu_E - K_1K_2P_C P_E)}{K_1K_2P_C(\beta\omega - \sigma\alpha)} \\ \bar{R} &= \frac{\beta(\alpha\mu_C\mu_E - K_1K_2P_C P_E)}{K_1K_2P_C(\beta\omega - \sigma\alpha)} \end{aligned} \right\}, \tag{13}$$

Corresponding to clearance of infection and active disease respectively,

where $\alpha = (K_2 + \gamma + \mu_E + \delta)$, $\beta = \delta + \gamma$, $\sigma = \mu_E + \omega$

Theorem 2: If $\frac{P_E}{\mu_E} \neq \frac{\alpha\mu_C}{K_1K_2P_C}$ there exist two equilibria.

Proof: The infection-free equilibrium is given by $P_1 = \left(0, \frac{P_E}{\mu_E}, 0, 0 \right)$

If $C \neq 0$, $I \neq 0$, $R \neq 0$, then $E = \frac{\alpha\mu_C}{K_1K_2P_C}$.

Hence the other equilibrium is

$$\begin{aligned} P_2 &= \left(\frac{\sigma(\alpha\mu_C\mu_E - K_1K_2P_C P_E)}{K_1\mu_C(\beta\omega - \sigma\alpha)}, \frac{\alpha\mu_C}{K_1K_2P_C}, \frac{\sigma(\alpha\mu_C\mu_E - K_1K_2P_C P_E)}{K_1K_2P_C(\beta\omega - \sigma\alpha)}, \frac{\beta(\alpha\mu_C\mu_E - K_1K_2P_C P_E)}{K_1K_2P_C(\beta\omega - \sigma\alpha)} \right) \\ &= (\phi_1, \phi_2, \phi_3, \phi_4) \end{aligned}$$

This completes the proof.

Next, we shall conduct stability analysis of the critical points.

Then, the Jacobian matrix of our system of equations (1) – (4) is

$$Df(C, E, I, R) = \begin{pmatrix} -\mu_C & 0 & P_C K_2 & 0 \\ -K_1 E & -\mu_E - K_1 C & 0 & \omega \\ K_1 E & K_1 C & -\alpha & 0 \\ 0 & 0 & \beta & -\sigma \end{pmatrix} \quad (14)$$

The linearization of (14) at $P_1 = \left(0, \frac{P_E}{\mu_E}, 0, 0\right)$ is

$$Df\left(0, \frac{P_E}{\mu_E}, 0, 0\right) = \begin{pmatrix} -\mu_C & 0 & q & 0 \\ -r & -\mu_E & 0 & \omega \\ r & 0 & -\alpha & 0 \\ 0 & 0 & \beta & -\sigma \end{pmatrix} \quad (15)$$

with eigenvalues:

$$\lambda_1 = -\sigma, \quad \lambda_2 = -\mu_E, \quad \lambda_{3,4} = \frac{-(\alpha + \mu_C) \pm \sqrt{(\alpha + \mu_C)^2 - 4(\alpha\mu_C - qr)}}{2} \quad (16)$$

Where

$$q = P_C K_2, \quad r = \frac{K_1 P_E}{\mu_E}$$

By definition, all the parameters are non-negative, hence q and r are non-negative.

1. If $\alpha\mu_C > qr$ and $(\alpha + \mu_C)^2 - 4(\alpha\mu_C - qr) > 0$ the eigenvalues are real, unequal and negative.
2. If $(\alpha + \mu_C)^2 - 4(\alpha\mu_C - qr) = 0$ the eigenvalues are real, equal and negative.
3. If $\alpha\mu_C > qr$ and $(\alpha + \mu_C)^2 - 4(\alpha\mu_C - qr) < 0$ the eigenvalues are complex with negative real part.

So in either case the disease-free equilibrium (DFE) is locally asymptotically stable.

Now, let us denote the endemic equilibrium (EE) points $(\phi_1, \phi_2, \phi_3, \phi_4)$ where each component corresponds to an earlier specified value.

We let

$$C^* = C - \phi_1, \quad E^* = E - \phi_2, \quad I^* = I - \phi_3, \quad R^* = R - \phi_4$$

Then

$$\frac{dC^*}{dt} = P_C K_2 I^* - \mu_C C^* \tag{17}$$

$$\frac{dE^*}{dt} = -\mu_E E^* - K_1 \phi_2 C^* - K_1 \phi_1 E^* + \omega R^* \tag{18}$$

$$\frac{dI^*}{dt} = K_1 \phi_2 C^* + K_1 \phi_1 E^* - \alpha I^* \tag{19}$$

$$\frac{dR^*}{dt} = \beta I^* - \sigma R^* \tag{20}$$

Thus

$$\begin{pmatrix} C^* \\ E^* \\ I^* \\ R^* \end{pmatrix}' = A \begin{pmatrix} C^* \\ E^* \\ I^* \\ R^* \end{pmatrix} \tag{21}$$

Where

$$A = \begin{pmatrix} -\mu_C & 0 & q & 0 \\ -r & -s & 0 & \omega \\ r & p & -\alpha & 0 \\ 0 & 0 & \beta & -\sigma \end{pmatrix}$$

and $q = P_C K_2, \quad r = K_1 \phi_2, \quad s = (\mu_E + K_1 \phi_1), \quad p = K_1 \phi_1$

Thus

$$|A - \lambda I| = 0$$

Implies

$$\lambda_1 = -\sigma$$

And

$$P(\lambda) = \lambda^3 + (\mu_C + s + \alpha)\lambda^2 + (\mu_C s + \mu_C \alpha + s\alpha - qr)\lambda + (\mu_C s\alpha + qr(p - s)) = 0 \tag{22}$$

Theorem 3: Let $K_1 = 0$. Then Equation (22) has three negative roots or one negative root and two complex roots.

Theorem 4: The infected (endemic) equilibrium is locally asymptotically stable if $K_1 = 0$.

Proof of theorems

The proof of the theorems 3 and 4 involved using the

(i) Descartes rule of signs:

The number of positive zeros of a polynomial with real coefficients is either equal to the number of variations in sign of the polynomial or less than this by an even number and

(ii) Routh-Hurwitz criteria [8]:

All zeros of $\lambda^3 + \alpha\lambda^2 + \beta\lambda + \gamma = 0$ have negative real parts if and only if $\alpha\beta - \gamma > 0$.

Therefore, all zeros of (22) have negative real parts if and only if

$$(\mu_c + s + \alpha)(\mu_c s + \mu_c \alpha + s\alpha - qr) - (\mu_c s\alpha + qr(p - s)) > 0$$

That is

$$(\mu_c + s + \alpha)(\mu_c s + \mu_c \alpha) + s\alpha(s + \alpha) - qr(\mu_c + \alpha + p) > 0 \text{ if } K_1 = 0.$$

Proof of theorem 3

From $P(\lambda)$ in (22), we obtain

$$P(-\lambda) = -\lambda^3 + (\mu_c + s + \alpha)\lambda^2 - (\mu_c s + \mu_c \alpha + s\alpha - qr)\lambda + (\mu_c s\alpha + qr(p - s)) = 0$$

So the number of change in sign is 3, if $K_1 = 0$. Hence by Descartes rule of signs, $P(\lambda)$ have either three negative roots or one negative root and two complex roots. This completes the proof.

Proof of theorem 4

Since the inequality holds if $K_1 = 0$. By theorem 3 and Routh-Hurwitz criteria, (22) has

- (i) Either three negative roots or
- (ii) One negative root and two complex roots whose real parts are equal and negative.

So in either case the equilibrium is locally asymptotically stable. This completes the proof.

Furthermore, we have the following result on the global stability of DFE.

Let N stands for the total epithelial cells of mucosal linings. Then we can write $R = N - E - I$.

We consider the model (1) - (3). Note that

$$C' = P_c K_2 I - \mu_c C \leq P_c K_2 N - \mu_c C$$

We have $0 \leq E$, $0 \leq I$ and $E + I \leq N$. The biological domain of this system (1) – (3) is the standard simplex.

The set $\Omega = \left\{ (C, E, I) : E \geq 0, I \geq 0, 0 \leq E + I \leq N, 0 \leq C \leq \frac{P_c K_2 N}{\mu_c} \right\}$ is a positively invariant compact set for (1) – (3). The system is well posed.

The basic reproduction number is given by

$$R_0 = \frac{P_c K_1 K_2}{\alpha \mu_c} E_0$$

Theorem 5: If $R_0 < 1$ then the DFE is globally asymptotically stable on Ω .

Proof: We define a Lyapunov function

$$V = P_c K_2 I + \alpha C$$

Clearly $V \geq 0$. Consider its derivatives:

$$\begin{aligned} \dot{V} &= P_c K_2 \dot{I} + \alpha \dot{C} = P_c K_1 K_2 C E - \alpha \mu_c C = \\ &P_c K_1 K_2 C E_0 \left(E - \frac{\alpha \mu_c}{P_c K_1 K_2 E_0} \right) \leq P_c K_1 K_2 C E_0 \left(1 - \frac{1}{R_0} \right) \leq 0 \end{aligned}$$

Since $R_0 < 1$. We see that $\dot{V} = 0$ if and only if $C = 0$ or $R_0 = 1$. Hence the largest invariant set in $\{(C, E, I) \in \Omega : \dot{V}(C, I) = 0\}$ is reduced to the DFE. Since we are in a compact positively invariant set, by the LaSalle's Invariance Principle [9], the DFE is globally asymptotically stable in Ω .

3.3 Solution by Parameter-expanding Method

Parameter-expanding method proposed by He and was successfully applied to various engineering problems [10]. We apply Parameter-expanding method to equations (1) – (4), where details can be found in [10].

For convenience, let $C = x$, $E = y$, $I = z$, $R = v$ and suppose the solution $x(t)$, $y(t)$, $z(t)$ and $v(t)$ in (1) - (4) can be expressed as

$$\left. \begin{aligned} x(t) &= x_0(t) + K_1 x_1(t) + K_1^2 x_2(t) + h.o.t \\ y(t) &= y_0(t) + K_1 y_1(t) + K_1^2 y_2(t) + h.o.t \\ z(t) &= z_0(t) + K_1 z_1(t) + K_1^2 z_2(t) + h.o.t \\ v(t) &= v_0(t) + K_1 v_1(t) + K_1^2 v_2(t) + h.o.t \end{aligned} \right\}, \tag{23}$$

where *h.o.t.* read “higher order terms in K_1 . In our analysis, we assume K_1 is small, so we are interested only in the first two terms.

Substituting (23) into (1) - (4), and processing, we obtain:

$$\frac{dx_0}{dt} = P_C K_2 z_0 - \mu_C x_0, \quad x_0(0) = x_0 \tag{24}$$

$$\frac{dy_0}{dt} = P_E - \mu_E y_0 + \omega v_0, \quad y_0(0) = y_0 \tag{25}$$

$$\frac{dz_0}{dt} = -\alpha z_0, \quad z_0(0) = z_0 \tag{26}$$

$$\frac{dv_0}{dt} = \beta z_0 - \sigma v_0, \quad v_0(0) = v_0 \tag{27}$$

$$\frac{dx_1}{dt} = P_C K_2 z_1 - \mu_C x_1, \quad x_1(0) = 0 \tag{28}$$

$$\frac{dy_1}{dt} = -\mu_E y_1 - x_0 y_0 + \omega v_1, \quad y_1(0) = 0 \tag{29}$$

$$\frac{dz_1}{dt} = x_0 y_0 - \alpha z_1, \quad z_1(0) = 0 \tag{30}$$

$$\frac{dv_1}{dt} = \beta z_1 - \sigma v_1, \quad v_1(0) = 0 \tag{31}$$

Solving equations (24) – (31) by direct integration, we obtain

$$x_0(t) = a_2 e^{-\alpha t} + a_3 e^{-\mu_C t} \tag{32}$$

$$y_0(t) = a_6 (1 - e^{-\mu_E t}) + a_4 (e^{-\alpha t} - e^{-\mu_E t}) + a_5 (e^{-\sigma t} - e^{-\mu_E t}) + y_0 e^{-\mu_E t} \tag{33}$$

$$z_0(t) = z_0 e^{-\alpha t} \tag{34}$$

$$v_0(t) = a_0 e^{-\alpha t} + a_1 e^{-\sigma t} \tag{35}$$

$$x_1(t) = d_0 e^{-\mu_C t} - d_1 e^{-\alpha t} - c_3 t e^{-\alpha t} + c_6 e^{-2\alpha t} + c_8 t e^{-\mu_C t} + c_{10} e^{-(\sigma+\alpha)t} + c_{11} e^{-(\mu_E+\mu_C)t} - c_9 e^{-(\mu_E+\alpha)t} + c_{12} e^{-(\mu_C+\alpha)t} + c_{13} e^{-(\mu_C+\sigma)t} \tag{36}$$

$$y_1(t) = n_{11}e^{-\mu_E t} + n_{12}e^{-\alpha t} - n_0 e^{-\sigma t} + n_2 t e^{-\alpha t} - e_4 e^{-2\alpha t} - n_5 e^{-\mu_C t} - n_7 e^{-(\sigma+\alpha)t} - n_8 e^{-(\mu_E+\mu_C)t} - n_6 e^{-(\mu_C+\alpha)t} - n_9 e^{-(\mu_C+\sigma)t} + n_{10} e^{-(\mu_E+\alpha)t} \quad (37)$$

$$z_1(t) = b_0 t e^{-\alpha t} - a_7 e^{-2\alpha t} + b_8 e^{-\alpha t} - a_8 e^{-\mu_C t} + a_9 e^{-(\mu_E+\alpha)t} - a_{10} e^{-(\sigma+\alpha)t} - a_{11} e^{-(\mu_E+\mu_C)t} - a_{12} e^{-(\mu_C+\alpha)t} - a_{13} e^{-(\mu_C+\sigma)t} \quad (38)$$

$$v_1(t) = c_2 e^{-\sigma t} - c_3 e^{-\alpha t} - b_{10} t e^{-\alpha t} + b_{11} e^{-2\alpha t} + b_{13} e^{-\mu_C t} + b_{15} e^{-(\sigma+\alpha)t} + b_{16} e^{-(\mu_E+\mu_C)t} + (b_{17} - b_{14}) e^{-(\mu_C+\alpha)t} + b_{18} e^{-(\mu_C+\sigma)t} \quad (39)$$

Where

$$a_0 = \frac{\beta z_0}{\sigma - \alpha}, \quad a_1 = \left(v_0 - \frac{\beta z_0}{\sigma - \alpha} \right), \quad a_2 = \frac{P_C K_2 z_0}{\sigma - \alpha}, \quad a_3 = \left(x_0 - \frac{P_C K_2 z_0}{\sigma - \alpha} \right),$$

$$a_4 = \frac{\omega \beta z_0}{(\sigma - \alpha)(\mu_E - \alpha)}, \quad a_5 = \frac{\omega}{(\mu_E - \alpha)} \left(v_0 - \frac{\beta z_0}{\sigma - \alpha} \right), \quad a_6 = \frac{P_E}{\mu_E},$$

$$b_0 = a_2 a_6, \quad b_1 = a_2 a_4, \quad b_2 = a_2 a_5, \quad b_3 = a_2 y_0, \quad b_4 = a_3 a_6, \quad b_5 = a_3 a_4,$$

$$b_6 = a_3 a_5, \quad b_7 = a_3 y_0, \quad c_0 = b_0 + b_1 + b_2 + b_3, \quad c_1 = b_7 - b_4 - b_5 - b_6,$$

$$a_7 = \frac{b_1}{\alpha}, \quad a_8 = \frac{b_4}{(\mu_C - \alpha)}, \quad a_9 = \frac{c_0}{\mu_E}, \quad a_{10} = \frac{b_2}{\sigma}, \quad a_{11} = \frac{c_1}{(\mu_E + \mu_C - \alpha)},$$

$$a_{12} = \frac{b_5}{\mu_C}, \quad a_{13} = \frac{b_6}{(\mu_C + \sigma - \alpha)}, \quad b_8 = (a_7 + a_8 - a_9 + a_{10} + a_{11} + a_{12} + a_{13}),$$

$$b_9 = \frac{\beta b_0}{(\alpha - \sigma)^2}, \quad b_{10} = \frac{\beta b_0}{(\alpha - \sigma)}, \quad b_{11} = \frac{\beta a_7}{(2\alpha - \sigma)}, \quad b_{12} = \frac{\beta b_8}{(\alpha - \sigma)},$$

$$b_{13} = \frac{\beta a_8}{(\mu_C - \sigma)}, \quad b_{14} = \frac{\beta a_9}{(\mu_E + \alpha - \sigma)}, \quad b_{15} = \frac{\beta a_{10}}{\alpha}, \quad b_{16} = \frac{\beta a_{11}}{(\mu_E + \mu_C - \sigma)},$$

$$b_{17} = \frac{\beta a_{12}}{(\mu_C + \alpha - \sigma)}, \quad b_{17} = \frac{\beta a_{12}}{(\mu_C + \alpha - \sigma)}, \quad b_{18} = \frac{\beta a_{13}}{\mu_C},$$

$$c_2 = (b_9 - b_{11} + b_{12} - b_{13} + b_{14} - b_{15} + b_{16} + b_{17} + b_{18}), \quad c_3 = (b_9 + b_{12})$$

$$c_4 = \frac{P_C K_2 b_0}{(\alpha - \mu_C)^2}, \quad c_5 = \frac{P_C K_2 b_0}{(\alpha - \mu_C)}, \quad c_6 = \frac{P_C K_2 a_7}{(2\alpha - \mu_C)}, \quad c_7 = \frac{P_C K_2 b_8}{(\alpha - \mu_C)},$$

$$c_8 = P_C K_2 a_8, \quad c_9 = \frac{P_C K_2 a_9}{(\mu_E + \alpha - \mu_C)}, \quad c_{10} = \frac{P_C K_2 a_{10}}{(\sigma + \alpha - \mu_C)}, \quad c_{11} = \frac{P_C K_2 a_{11}}{\mu_E},$$

$$c_{12} = \frac{P_C K_2 a_{12}}{\alpha}, \quad c_{13} = \frac{P_C K_2 a_{13}}{\sigma}, \quad d_0 = c_4 - c_6 + c_7 + c_9 - c_{10} - c_{11} - c_{12} - c_{13}, \quad d_1 = c_4 + c_7$$

$$d_2 = \omega c_2, \quad d_3 = \omega c_3 - b_0, \quad d_4 = \omega b_{11} - b_1, \quad d_5 = \omega b_{13} - b_4, \quad d_6 = \omega b_{17} - \omega b_{14} - b_5, \\ d_7 = \omega b_{15} - b_2, \quad d_8 = \omega b_{16} - c_1, \quad d_9 = \omega b_{18} - b_6, \quad d_{10} = \omega b_{10},$$

$$n_0 = \frac{d_2}{(\sigma - \mu_E)}, \quad n_1 = \frac{d_3}{(\alpha - \mu_E)}, \quad n_2 = \frac{d_{10}}{(\alpha - \mu_E)}, \quad n_3 = \frac{d_{10}}{(\alpha - \mu_E)^2},$$

$$n_4 = \frac{d_4}{(2\alpha - \mu_E)}, \quad n_5 = \frac{d_5}{(\mu_C - \mu_E)}, \quad n_6 = \frac{d_6}{(\mu_C + \alpha - \mu_E)}, \quad n_7 = \frac{d_7}{(\alpha + \sigma - \mu_E)},$$

$$n_8 = \frac{d_8}{\mu_C}, \quad n_9 = \frac{d_9}{(\mu_C + \sigma - \mu_E)}, \quad n_{10} = \frac{c_0}{\alpha},$$

$$n_{11} = n_0 + n_1 - n_3 + n_4 + n_5 + n_6 + n_7 + n_8 + n_9 - n_{10}, \quad n_{12} = n_3 - n_1$$

The computations were done using computer symbolic algebraic package MAPLE.

4 Results and Discussion

Here the existence and uniqueness of solution of our system of equations (1) – (4) is proved by actual solutions. Also, under certain conditions, we have conducted local and global stability analysis of the disease-free and endemic equilibriums. The results showed that is stable. Analytical solutions of equations (1) - (4) are achieved via Parameter-expanding method and computed for the values of

$$P_C = 20, \quad P_E = 10 \text{ cells} / \text{mm}^3 / \text{day}, \quad K_1 = 0.02 \text{ mm}^3 / \text{day} / \text{cell}, \quad K_2 = 0.33 \text{ days}^{-1}, \\ \mu_C = 3 \text{ days}^{-1}, \quad \mu_E = 2 \text{ days}^{-1}, \quad \gamma = 2 \text{ days}^{-1}, \quad \delta = 0.2 \text{ days}^{-1}, \quad \omega = 0.04 \text{ days}^{-1}$$

The concentration of free extracellular chlamydial particles and number of uninfected mucosal epithelial cells, *Chlamydia*-infected epithelial cells and recovered epithelial cells are depicted graphically in Figs. 1 - 7.

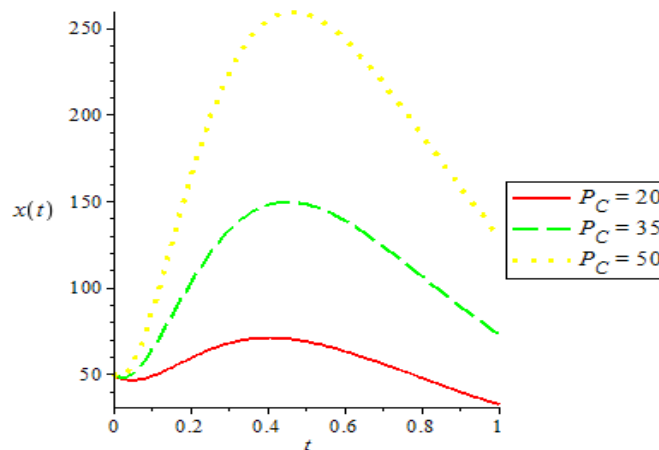


Figure 1: $x(t)$ versus time t for various values of P_C at $P_E = 10, K_1 = 0.02, K_2 = 0.33, \mu_C = 3, \mu_E = 2, \gamma = 2, \delta = 0.2, \omega = 0.04, v_0 = 0, x_0 = 50, y_0 = 300, z_0 = 10$

From Fig. 1, we can conclude that with the increase of number of infectious chlamydial particles released by an infected cell (P_C), concentration of free extracellular chlamydial particles increase.

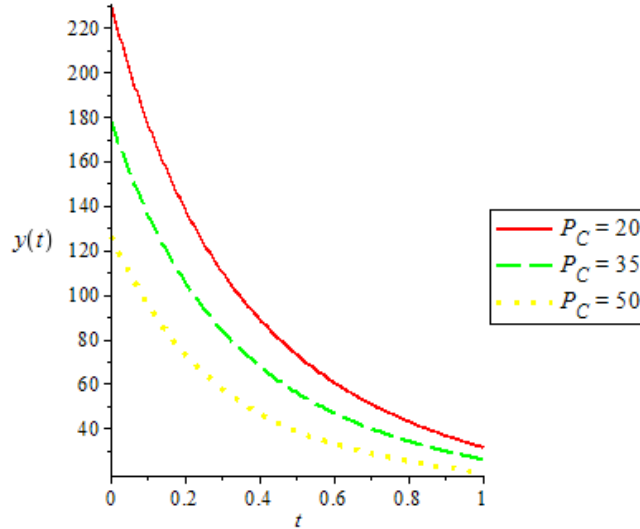


Figure 2: $y(t)$ versus time t for various values of P_C at $P_E = 10, K_1 = 0.02, K_2 = 0.33, \mu_C = 3, \mu_E = 2, \gamma = 2, \delta = 0.2, \omega = 0.04, v_0 = 0, x_0 = 50, y_0 = 300, z_0 = 10$

From Fig. 2, we can conclude that with the increase of number of infectious chlamydial particles released by an infected cell (P_C), number of uninfected mucosal epithelial cells decrease.

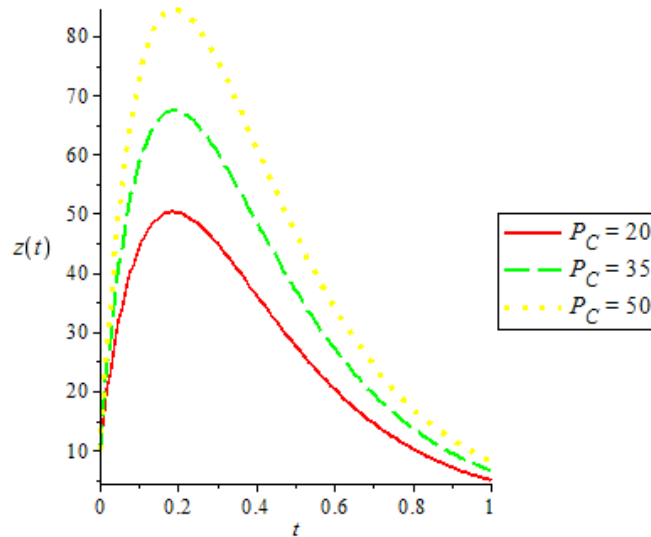


Figure 3: $z(t)$ versus time t for various values of P_C at $P_E = 10, K_1 = 0.02, K_2 = 0.33, \mu_C = 3, \mu_E = 2, \gamma = 2, \delta = 0.2, \omega = 0.04, v_0 = 0, x_0 = 50, y_0 = 300, z_0 = 10$

From Fig. 3, we can conclude that with the increase of number of infectious chlamydial particles released by an infected cell (P_C), number of *Chlamydia*-infected epithelial cells increase.

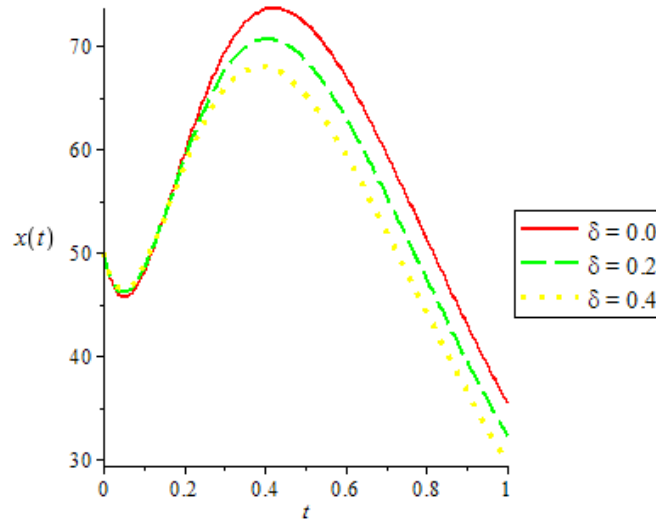


Figure 4: $x(t)$ versus time t for various values of δ at $P_C = 20, P_E = 10, K_1 = 0.02, K_2 = 0.33, \mu_C = 3, \mu_E = 2, \gamma = 2, \omega = 0.04, v_0 = 0, x_0 = 50, y_0 = 300, z_0 = 10$

From Fig. 4, we can conclude that with the increase of recovery rate due to drug administration (δ), concentration of free extracellular chlamydial particles decrease.

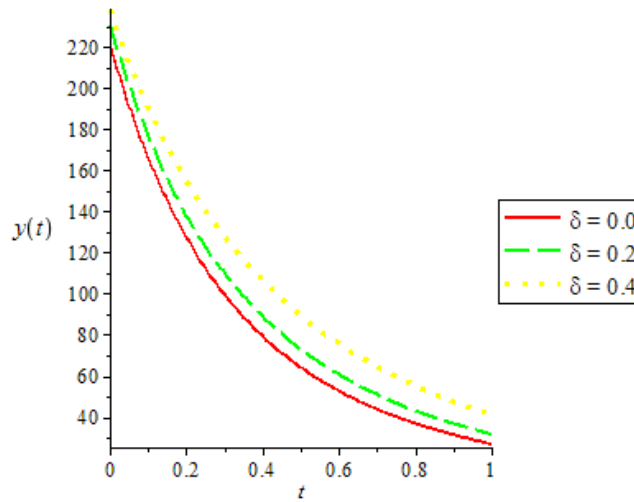


Figure 5: $y(t)$ versus time t for various values of δ at $P_C = 20, P_E = 10, K_1 = 0.02, K_2 = 0.33, \mu_C = 3, \mu_E = 2, \gamma = 2, \omega = 0.04, v_0 = 0, x_0 = 50, y_0 = 300, z_0 = 10$

From Fig. 5, we can conclude that with the increase of recovery rate due to drug administration (δ), number of uninfected mucosal epithelial cells increase.

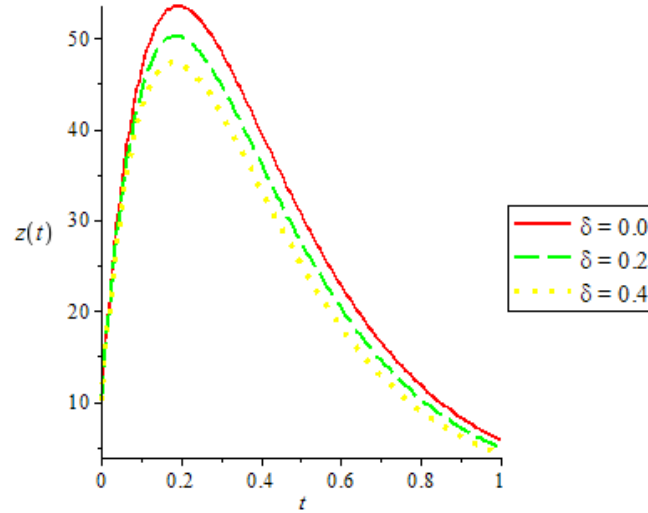


Figure 6: $z(t)$ versus time t for various values of δ at $P_C = 20, P_E = 10, K_1 = 0.02, K_2 = 0.33, \mu_C = 3, \mu_E = 2, \gamma = 2, \omega = 0.04, v_0 = 0, x_0 = 50, y_0 = 300, z_0 = 10$

From Fig. 6, we can conclude that with the increase of recovery rate due to drug administration (δ), number of *Chlamydia*-infected epithelial cells decrease.

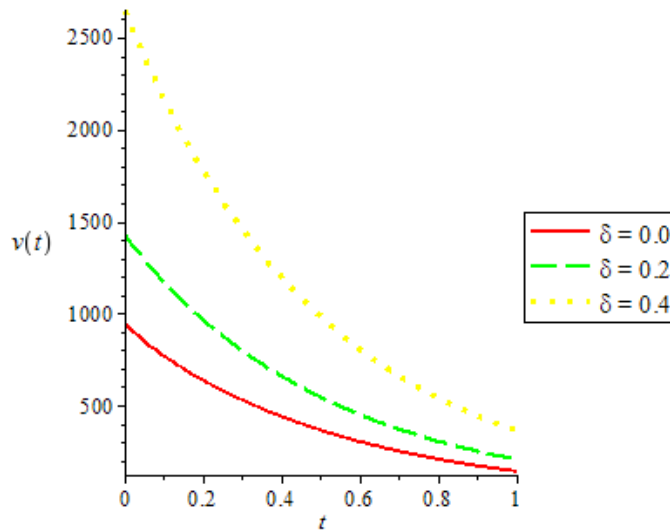


Figure 7: $v(t)$ versus time t for various values of δ at $P_C = 20, P_E = 10, K_1 = 0.02, K_2 = 0.33, \mu_C = 3, \mu_E = 2, \gamma = 2, \omega = 0.04, v_0 = 0, x_0 = 50, y_0 = 300, z_0 = 10$

From Fig. 7, we can conclude that with the increase of recovery rate due to drug administration (δ), number of recovered epithelial cells increase.

5 Conclusion

From the studies made on this paper we conclude as under.

1. Burst size per infected cell enhances the concentration of free extracellular chlamydial particles and reduces the number of uninfected mucosal epithelial cells.
2. Recovery rate due to drug administration increase the number of uninfected mucosal epithelial cells and recovered epithelial cells and decrease the concentration of free extracellular chlamydial particles and number of *Chlamydia*-infected epithelial cells.
3. The local stability of infected (endemic) equilibrium depends on the rate of cell infection.

Thus, increased ability to clear infection will be obtained if

1. There is a proper treatment of ailments
2. Number of infectious chlamydial particles released by an infected cell is reduced.
3. Contact between uninfected mucosal epithelial cells and free extracellular Chlamydia particles can be prevented.

Competing Interests

Authors have declared that no competing interests exist.

References

- [1] Dixon RE. Chlamydia infection causes loss of oviduct pacemaker cells and inhibits oocyte transport in the mouse oviduct. *Journal of Biology Reproduction*. 2009;80:665–673.
- [2] Wilson DP. Mathematical modeling of Chlamydia. *ANZIAM Journal*. 2004;45(E):C201-C214. Available: <http://www.journal.austms.org.au/ojs/index.php/ANZIAMJ/article/view/883/653>
- [3] Yang X, Brunham RC. T lymphocyte immunity in host defence against *Chlamydia trachomatis* and its implication for vaccine development. *Canadian J. Infect. Dis*. 1998;9:99-108.
- [4] Cain TK, Rank RG. Local Th1-like responses are induced by intravaginal infection of mice with the mouse pneumonitis biovar of *Chlamydia trachomatis*. *Infect and Immun*. 1995;63:1784-1789.
- [5] Magee DM, Williams DM, Smith JG, Bleicker CA, Grubbs BG, Schacter J, Rank RG. Role of CDS T cells in Primary *Chlamydia* infection. *Infect and Immun*. 1995;63:516-521.
- [6] Wilson DP, McElwain DLS. A Model of neutralization of *Chlamydia trachomatic* based on antibody and host cell aggregation on the elementary body surface. *Journal of Theoretical Biology*. 2004;226(3):321–330.
- [7] Wilson DP, Timms P, McElwain DLS. A mathematical model for the investigation of the Th1 immune response to *Chlamydia trachomatis*. *Mathematical Bioscience*. 2003;182:27–44. C206.

- [8] Ayeni RO, Popoola AO, Ogunmoyela JK. Some new results on affinity hemodialysis and T cell recovery. *Journal of Bacteriology Research*. 2010;2(1):001–004.
Available: <http://www.academicjournals.org/JBR>
- [9] LaSalle JP. *The stability of dynamical systems*, regional conference series in applied mathematics. SIAM, Philadelphia; 1976.
- [10] He JH. Some asymptotic methods for strongly nonlinear equations. *Int. J. Modern Phys. B*. 2006;20(10):1141-1199.

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