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Numerical Solution of Fractional Order Chlamydia Model Via the Generalized Fractional Adams-Bashforth-Moulton Approach

[1,2]Ibrahim Abdulrazaq Abubakar, [1,2]David Omale, [1,2]Jeremiah Amos, *[1,2]William Atokolo, [1,2]Emmanuel Abah, [1,2]Joseph Achonu Omale, [1,2]Samson Ojima Makolo, [1,2]Samuel Kehinde Olayemi and [1,2]Bolarinwa Bolaji

[1]Department of Mathematical Sciences, Prince Abubakar Audu University, Anyigba, Kogi State, Nigeria.
[2]Laboratory of Mathematical Epidemiology, Prince Abubakar Audu University, Anyigba, Kogi State, Nigeria.

*Corresponding Author's email: williamsatokolo@gmail.com

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ABSTRACT
In the present paper we offer the epidemiological parameters of the Chlamydia infection and discuss its dynamics with the help of a fractional-order mathematical model and estimating the role of contact and vaccination rates in the development of interaction with this disease. The conditions of existence and uniqueness of solutions of the problem in the environment of a fractional order were determined. Numerical simulations are carried out to show how the model parameters and fractional-order influence the disease control and their propagation property through the use of the fractional Adams-Bashforth-Moulton method. Additional simulations show that a rise in contact rates and a subsequent reduction of the efficacy of vaccination are the involved factors that contribute to the increase of the prevalence of the Chlamydia. The findings indicate that a preventive strategy to reduce transmission of the infection is a verified method of valuing the low level of the infection transmission over the population.

INTRODUCTION

Chlamydia is a sexually transmitted or transferable disease that is most prevalent in the world today (WHO 2022). It is projected that there are 129 million cases of Chlamydia trachomatis infection in the year 2020 (WHO, 2021; 2022). This infection is caused by the same bacteria, Chlamydia trachomatis and it can be primarily transmitted through contact with an infected person or through sex-contact using vaginal, anal and oral sex methods. It can also be transferred via non-sexual means and this includes; touching another person with bare hands, using of bed linen, towels or clothes, and in flies that exposed themselves to eye or nasal discharge. In selected instances, the infection occurs through infected vaginal fluids or semen that come in contact with the eye leading to conjunctivitis. It is notable that there is a parasite known as Chlamydia trachomatis which is the most known cause of blindness in the world Thylefors et al. (1995).

It is known to affect both females and males but with prevalence rates of 4.2 and 2.7 respectively Newman et al. (2012) and WHO (2021). Sexually active women aged 15 to 24 CDC (2022) have the highest risk especially as younger persons. Severely infected women may develop throat, rectal, and cervical diseases, which in most cases result in pelvic inflammatory disease (PID), sterility, and ectopic pregnancies or abortions Paavonen and Lehtinen (1996), Paavonen and Eggert-Kruse (1999).

Mothers infected also can infect the newborns during childbirth CDC (2022). The effects on women comprise backup vaginal discharge, itching, burning, bleeding,

nausea and fever. In men, the problem is accompanied by painful urination, testicles swelling and seminal fluid discharge. The incubation period takes 7-20 days, and although the Chlamydia and infections may be cured after several days of taking the antibiotics such as azithromycin or doxycycline the infected individuals are advised to avoid sex during the drug treatment period. The possibility of Re-infection is always there after successful treatment Diethelm (2022).

Mathematics modeling has been of great help in the understanding of infectious disease transmission dynamics such as Chlamydia. These models examine sources of epidemics as well as assist in the development of control measures. Even though there has been vast application of traditional models, they tend to ignore memory effects or even long-term dependence embedded in biological processes. Fractional-order models are on the rise to overcome such a shortcoming. It accounts the non-local characteristics through which memory effects and abnormal diffusion in disease transmission can be included Podlubny (1998).

Fractional differential equations (FDEs) are a generalization of the conventional integer-order representations, which lead to a broader landscape of complexity modelling. This is the paper that offers a fractional-order mathematical model that would model the transmission dynamics of Chlamydia, including elements of control, that is; treatment strategies (control through treatment) and prevention strategies to model transmission. The model proves to be more sufficient in detailing the spread of Chlamydia as it incorporates the merits of applying the memory effect phenomenon present in fractional calculus. The proposed study will also strive to identify the most effective way of reducing the level of Chlamydia infections and achieving sustainable control of the Chlamydia infection by playing simulation games in which various intervention situations will be simulated.

The fractional derivative, capable of capturing effects of memory and heredity in biological systems, is particularly well suited to the modeling of diseases such as Chlamydia. They can contribute to the better understanding of the spread of the infection over the course of time and the impact of the infection history of individuals and their treatment regimens on the dynamics of the transmission. This broader focus helps to create more realistic and effective control measures over and above persistent issues of drug resistance, re-infection and poor healthcare resources.

The current developments in fractional calculus, as indicated by Atokolo et al. (2022), have attracted a lot of attention due to the fact that it is used to explain the dynamic character of any given system. With respect to classical integer-order systems, which can only account local properties, the fraction order models represent memory effects exhibited by the global behavior of a system. Not only are these models more realistic, but also more applicable to the real-world setting, and thus make a good impact as a result of how infectious diseases like had Chlamydia spread.

In the application of biology, the types of fractional derivatives being adopted are Routh fractional derivatives, Caputo and Riemann-Liouville derivative of singular kernels. Moreover, non-singular terms of the kernel derivations, like Mittag-Leffler and Atangana-Baleanu are, also, in the spotlight.

Atokolo et al. (2022), demonstrated that the fractional-order Sterile Insect Technology (SIT) model could be employed to regulate the onset of the Zika virus where they employed LADM technique to obtain infinite series expansions of solutions to this model that converged to a correct answer.

Lassa fever, similar to Ebola, African swine fever and cholera, was also used by Atokolo et al. (2024)., to study with a fractional-order mathematical model where they used power-law fractional derivative to characterize the effects of the vaccination and treatment on the dynamics of the disease transmission process.

Yunus et al. (2023) considered a Caputo fractional-order derivative of the control of COVID-19 in Nigeria and LADM and observed a high rate of recovery in the integer-order case owing to various factors including vaccination and treatment.

Omede et al. (2024) were the first authors to propose a fractional-order compartmental model, in terms of Caputo derivatives, which was used to explain infections of soil-transmitted helminths. On LADM, they demonstrated that the solutions to their model of the non-rectilinear bodies using the infinite series converged to perfect values, thus, one seeing more flexibility in the model than integer-order models originating in the past.

The transmission dynamics of the hepatitis C were modeled as a fractional model using Adams-Bashforth-Moulton method by Amos et al. (2024). They have demonstrated moderate contact rate and treatment improvement can serve long distance in avertive multiple infection spread with the fractional-order model being more versatile than the traditional models.

James et al. (2024) also numerically demonstrated the dynamics of the HIV/AIDS infection process by means of a fractional order model and the Adams-Bashforth-Moulton method. Their findings underscored the need to decrease the probability of contact and improved treatment options which can be used to control the disease since the stochasticity of fractional models was higher compared to the conventional models.

A fractional transmission model was introduced by Abah et al. (2024) who used the Adams-Bashforth-Moulton discretization. Their findings reflected the reduction of contact rates and the improvement of treatment efficacy reduced the spread of the disease further demonstrating

the benefits of using fractional-order models to model complicated dynamics as compared to classical models.
An ABC-fractional order derivative model which was used to predict the co-epidemic dynamics of HIV and COVID-19 model was developed by Ahmed et al. (2021).
On the same note, Smith et al. (2023) performed an extensive analysis of co-infection patterns of hepatitis C and COVID-19. Their analysis was a synthesis equivalent of recent cadre of mathematical modeling studies, where the most frequently used techniques, key insights, and open holes left to be filled have been identified.
Fractional-order models have clear benefits, since they are flexible and are able to describe non-local effects. Fractional derivatives are less approximate than classical derivatives and better able to approximate phenomena in the real world and can attain some extra flexibility. They can include non-local interactions that are essential features not present in integer-order derivatives and can take account of memory effects that are not readily tractable in models that only include integer-order derivatives. Such properties have motivated researchers to use fractional differential equations to solve complex equations. As a case in point, Das et al. (2024), quoted Ullah et al. (2020) and solved fuzzy Volterra integral equations that had degenerate kernels using a combination of Laplace transform and Adomian Decomposition Method. Such innovative treatment has earned attention to the theoretical aspect of fuzzy analytical dynamic equations.
Ali et al. (2017) investigated the stability and the existence of solutions to a three-point boundary value problem with focus on the various types of Ulam stability. Their study used the classical non-linear fractional techniques to investigate the stability of the problem which adds valuable information to the area. The aim of this study includes to find conditions that give the existence and uniqueness of solution to the proposed fractional-order model; stability analysis of the endemic equilibrium point on the basis of the Lyapunov functions approach; solve numerically by the fractional Adams-Bashforth-Moulton method; and perform numerical experimentations to test the model performance.
A survey of the literature on Chlamydia and the mathematical modeling of their transmission also demonstrated deficiency of studies to consider application of fractional calculus in modeling Chlamydia transmission by employing the use of the Adams-Bashforth-Moulton method to model and study the transmission and control of Chlamydia.

Preliminary

In this section we present a short history of some of the most significant concepts and electrocalculi in the fractional calculus. This is realized in the context of the right and left fractional Caputo derivatives, on the grounds of the paradigms developed by Milici et al. (2018) and Bonyah et al. (2020). More importantly, the paper provides some kind of overview of the practical applications of fractional calculus towards solving challenges in the real world in various areas like in physics, engineering, bio-mathematics and other scientific activities.

Definition 1

Suppose that $f \in \Lambda^{\infty}(R)$, then the left-side and right-side Caputo fractional derivative of the function is given as follow:

$$CD_t^{\eta} f(t) = \left(t^0 D_t^{-(n-\eta)} \left(\frac{d}{dt}\right)^n f(t) \right)$$

$$CD_t^{\eta} f(t) = \frac{1}{\Gamma(n-\eta)} \int_0^t \left((t-\lambda)^{n-\eta-1} f^n(\lambda) \right) d\lambda \quad (1)$$

The same way

$$CD_t^{\eta} f(t) = \left(D_T^{-(n-\eta)} \left(\frac{-d}{dt}\right)^n \right) f(t)$$

$${}^{C}D_T^{\eta} f(t) = \frac{(-1)^n}{\Gamma(n-\eta)} \int_t^T (\lambda - t)^{n-\eta-1} f^n(\lambda) d\lambda$$

Definition 2

The generalized Mittag-Leffler function $E_{\alpha,\beta}(x)$ for $x \in R$ is given by

$$E_{\eta,\beta}(x) = \sum_{n=0}^{\infty} \frac{x^n}{\Gamma(\eta n+\psi)}, \eta, \psi > 0 \quad (2)$$

which can also be represented as

$$E_{\eta,\psi}(x) = xE_{\eta,\alpha+\eta(x)} + \frac{1}{\Gamma(\eta)} \quad (3)$$

$$E_{\eta,\psi}(x) = L\left[t^{\eta-1} E_{\alpha,\eta(\pm\omega t^{\alpha})}\right] = \frac{S^{\alpha-\eta}}{S^{\alpha} \pm \omega} \quad (4)$$

Proposition 1

Let $f \in \Lambda^{\infty}(R) \cap C(R)$and $\alpha \in R, n-1 < \eta < n$, therefore, the conditions given below holds:

1. ${}_{t_0}^{C}D_t^{\eta} I^{\eta} f(t) = f(t)$
2. ${}_{t_0}^{C}D_t^{\eta} I^{\eta} f(t) = f(t) - \sum_{k-0}^{n-k} \frac{t^k}{K!} f^k(t_0)$

Model Formulation and Description

The rate at which individuals are recruited into the susceptible population is written as Λ_h , and therefore, β_1, β_2 are the effective rate of contact between the susceptible and infected humans and individuals under treatment due to Chlamydia respectively. We denote ψ_2 as the transition rates of exposed Chlamydia classes to infected class. The rate at which infected humans are treated is given by α_2 and σ_1 is the recovery rate of human due to Chlamydia. The natural rate of death among human beings is vested as μ_h . The Chlamydia and human's death rate due to the disease in the infected and treated individuals is expressed as δ_1, δ_5. The proportion of susceptible humans vaccinated against Chlamydia is taken to be ϕ_1 and , ϕ_2 where is the rate of vaccine failure., Recovered human population become susceptible again at the rate of τ_2.

Model Assumptions

1. We assumed that the vaccine is imperfect in the sense that there is probability of vaccine failure.
2. We assume that even once humans recovered of Chlamydia, they can still be vulnerable to it in the aftermath of the pathogen encounter.
3. We assume that humans die due to disease in the population.

Model Flow Diagram of Chlamydia

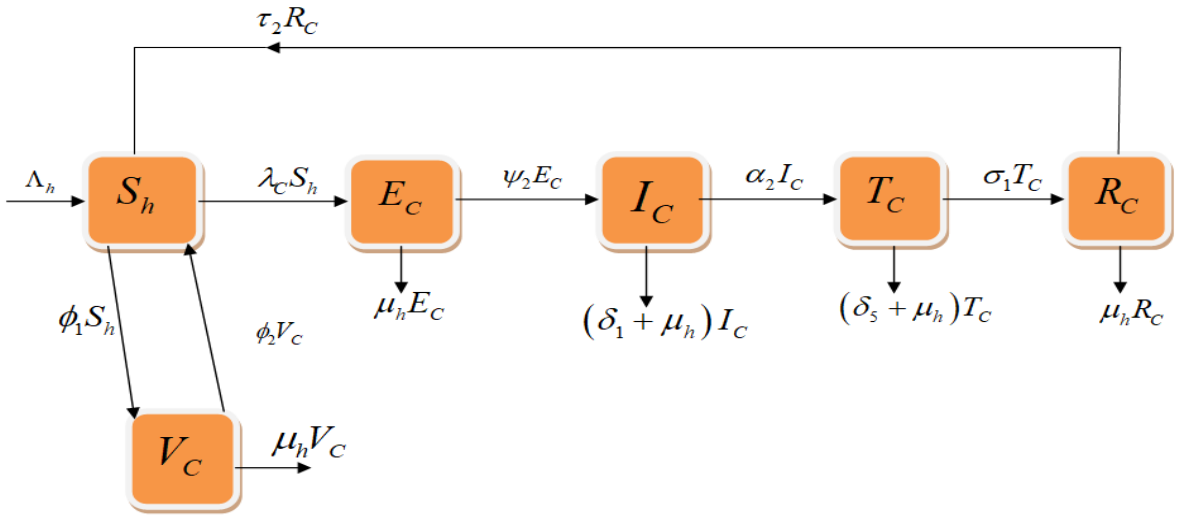

Figure 1: Chlamydia Model Flow Diagram

Chlamydia Model Equation

$$\frac{dS_h}{dt} = \boldsymbol{\Lambda}_h + \tau_2 R_C + \phi_2 V_C - \lambda_C S_h - (\phi_1 + \mu_h) S_h,$$
$$\frac{dE_C}{dt} = \lambda_C S_h - (\psi_2 + \mu_h) E_C,$$
$$\frac{dI_C}{dt} = \psi_2 E_C - (\alpha_2 + \delta_1 + \mu_h) I_C,$$
$$\frac{dT_C}{dt} = \alpha_2 I_C - (\sigma_1 + \delta_5 + \mu_h) T_C,$$
$$\frac{dV_C}{dt} = \phi_1 S_h - (\phi_2 + \mu_h) V_C,$$
$$\frac{dR_C}{dt} = \sigma_1 T_C - (\tau_2 + \mu_h) R_C.$$

Where $\lambda_C = \frac{(\beta_1 I_C + \beta_2 T_C)}{N_h}$.

Table 1: Model Variables and Parameters Descriptions

Variables	**Descriptions**
S_h	Humans who are susceptible to Chlamydia
E_C	Humans population who are Exposed to Chlamydia
I_C	Human population infected with Chlamydia
T_C	Human population on Chlamydia treatment
V_C	Vaccinated human population against Chlamydia
R_C	Recovered human population from Chlamydia
Parameters	**Descriptions**
Λ_h	Recruitment rate of Susceptibility of people to Chlamydia
β_1	The frequency of contact between the susceptible human beings and human infected being
β_2	
ψ_2	Rate of exposure of human subjects to infected human beings
α_2	Infected peoples, treatment rate
σ_1	The recoverability of the human being regarding the Chlamydia treatment
μ_h	Natural death rate of human being
δ_1	Mortality rate of infected human population of Chlamydia
δ_5	The mortality of human beings under Chlamydia drugs due to disease in mankind
ϕ_1	Vaccination in human population who are susceptible
ϕ_2	The decline in the rate of vaccine
τ_2	The rate of conversion of the susceptible human population back into susceptibility

Fractional Chlamydia Mathematical Model

In this part, we will extend the integer model of Chlamydia, in Equation (5), a Caputo fractional derivative operator. The latter variation is less constrained than the traditional one expressed in Equation (5) because the outcome of the fractional order model can be adjusted to take on various characteristics. The fractional Chlamydia model is thus developed as follows:

$$^{c}D_t^{\eta}S_h = \Lambda_h + \tau_2 R_C + \phi_2 V_C - \frac{(\beta_1 I_C + \beta_2 T_C)}{N_h} S_h - P_1 S_h, \quad (5)$$

$$^{c}D_t^{\eta}E_C = \frac{(\beta_1 I_C + \beta_2 T_C)}{N_h} S_h - P_2 E_C,$$

$$^{c}D_t^{\eta}I_C = \psi_2 E_C - P_3 I_C, \quad (6)$$

$$^{c}D_t^{\eta}T_C = \alpha_2 I_C - P_4 T_C,$$

$$^{c}D_t^{\eta}V_C = \phi_1 S_h - P_5 V_C,$$

$$^{c}D_t^{\eta}R_C = \sigma_1 T_C - P_6 R_C.$$

Where

$P_1 = (\phi_1 + \mu_h), P_2 = (\psi_2 + \mu_h), P_3 = (\alpha_2 + \delta_1 + \mu_h), P_4 = (\sigma_1 + \delta_5 + \mu_h), \quad P_5 = (\phi_2 + \mu_h), P_6 = (\tau_2 + \mu_h).$

Subject to positive initial conditions

$$S_h(0) = S_{h0}, E_C(0) = E_{C0}, I_C(0) = I_{C0}, T_C(0) = T_{C0}, V_C(0) = V_{C0}, R_C(0) = R_{C0}. \quad (7)$$

Positivity of Model Solution

We considered the non-negativity of the initial values

$N_h(t) \leq \frac{\Lambda_h}{\mu_h}$ as $t \to \infty$

Secondly, $if\ limsup\ N_{h0}(t) \leq \frac{\Lambda_h}{\mu_h}$, then our model feasible domain is given by:

$$\Omega_h = \left\{(S_h, E_C, I_C, T_C, V_C, R_C) \subset R_+^6 : (S_h + E_C + I_C + T_C + V_C + R_C) \leq \frac{\Lambda_h}{\mu_h}\right\},$$

hence Ω is positively invariant.

If $(S_{h0}, E_{C0}, I_{C0}, T_{C0}, V_{C0}, R_{C0})$ are non-negative, then the solution of model (6) will be non-negative for $t > 0$. From Eq. (6), selecting the first equation, we obtained:

$$^{c}D_t^{\eta}S_h = \Lambda_h + \tau_2 R_C + \phi_2 V_C - \frac{(\beta_1 I_C + \beta_2 T_C)}{N_h} S_h - P_1 S_h,$$

$$^{c}D_t^{\eta}S_h + \frac{(\beta_1 I_C + \beta_2 T_C + P_1)}{N_h} S_h = \Lambda_h + \tau_2 R_C + \phi_2 V_C,$$

But $\Lambda_h + \tau_2 R_C + \phi_2 V_C \geq 0$ then

$$^{c}D_t^{\eta}S_h + \frac{(\beta_1 I_C + \beta_2 T_C + P_1)}{N_h} S_h \geq 0.$$

Applying the Laplace transform we obtained:

$$L\left[{}^{c}D_t^{\eta}S_h\right] + L\left[\frac{(\beta_1 I_C + \beta_2 T_C + P_1)}{N_h} S_h\right] \geq 0.$$

$$S_h^{\eta} S_h(s) - S_h^{\eta-1} S_h(0) + \frac{(\beta_1 I_C + \beta_2 T_C + P_1)}{N_h} S_h(s) \geq 0,$$

$$S_h(s) \geq \frac{S_h^{\eta-1}}{S_h^{\eta} + \frac{(\beta_1 I_C + \beta_2 T_C + P_1)}{N_h} S_h} S_h(0). \quad (8)$$

By taking the inverse Laplace transforms, we obtained;

$$S_h(t) \geq E_{t\eta,1}\left(\frac{(\beta_1 I_C + \beta_2 T_C + P_1)}{N_h} S_h t^{\eta}\right) S_{h0}. \quad (9)$$

Now since the term on the right-hand side of Eq. (9) is positive, we conclude that $S_h \geq 0$ for $t \geq 0$. In the same way, we also have that $(E_C \geq 0, I_C \geq 0, T_C \geq 0, V_C \geq 0, R_C \geq 0)$ are positives, therefore, the solution will remain in R_+^6 for all $t \geq 0$ with positive initial conditions.

Boundedness of Fractional Model Solution

The total human population from our model is given by;

$$N_h(t) = S_h(t) + E_C(t) + I_C(t) + T_C(t) + V_C(t) + R_C(t)$$

So from our fractional model (6), we now obtain

$$^{c}D_t^{\eta}N_h(t) = {}^{c}D_t^{\eta}S_h(t) + {}^{c}D_t^{\eta}E_C(t) + {}^{c}D_t^{\eta}I_C(t) + {}^{c}D_t^{\eta}T_C(t) + {}^{c}D_t^{\eta}V_C(t) + {}^{c}D_t^{\eta}R_C(t). \quad (10)$$

Taking the Laplace transformation of (10) we obtained;

$$L\left[cD_t^{\eta}N_h(t)\right] = L[\Lambda_h - \mu_h N_h(t)]$$

$$S_h^{\eta} N_h(s) - S_h^{\eta-1} N_h(0) + \mu_h N_h(s) \leq \frac{\Lambda_h}{\mu_h},$$

$$N_h(s) \leq \frac{S_h^{\eta-1}}{(S^{\eta} + \mu_h)} N_h(0) + \frac{\Lambda_h}{S_h(S_h^{\eta} + \mu_h)} \quad (11)$$

Taking the inverse Laplace transform of Eq. (11) we have;

$$N_h(t) \leq E_{h\eta,1}(\mu_h t^{\eta}) N_h(0) + \Lambda_h E_{h\eta,\eta+1}(\mu_h t^{\eta}) \quad (12)$$

At $t \to \infty$, the limit of Eq. (12) becomes;

$$\underset{t\to\infty}{limSup} N_h(t) = \frac{\Lambda_h}{\mu_h}.$$

This means that, if $N_{h0} \leq \frac{\Lambda_h}{\mu_h}$

then $N_h(t) \leq \frac{\Lambda_h}{\mu_h}$ which implies that, $N_h(t)$ is bounded.

We now conclude that, this region $\Omega = \Omega_h$, is well posed and equally feasible epidemiologically.

Existence and Uniqueness of our Model Solution

Let the real non-negative be W we consider $P = [0, W]$.

All continuous function that exists on P belongs to $N_{he}^0(W)$ with norm as;

$$\|K\| = Sup\{|K(t)|, t \in W\}.$$

The modeled system (6) along with specified initial (8) enables solving for a system of differential equations presented in (13).

$$^{c}D_t^{\rho}K(t) = Z(t, K(t)), 0 < t < W < \infty, \quad (13)$$

$$K(0) = K_0.$$

Where $K(t) = (S_h, E_C, I_C, T_C, V_C, R_C)$ represents the classes and Z be a continuous function defined as follows;

$$Z(t, K(t)) = \begin{pmatrix} Z_1(t, S_h(t)) \\ Z_2(t, E_C(t)) \\ Z_3(t, I_C(t)) \\ Z_4(t, T_C(t)) \\ Z_5(t, V_C(t)) \\ Z_6(t, R_C(t)) \end{pmatrix} = \begin{pmatrix} \Lambda_h + \tau_2 R_C + \phi_2 V_C - \frac{(\beta_1 I_C + \beta_2 T_C)}{N_h} S_h - (\phi_1 + \mu_h) S_h \\ \frac{(\beta_1 I_C + \beta_2 T_C)}{N_h} S_h - (\psi_2 + \mu_h) E_C \\ \psi_2 E_C - (\alpha_2 + \delta_1 + \mu_h) I_C \\ \alpha_2 I_C - (\sigma_1 + \delta_5 + \mu_h)_4 T_C \\ \phi_1 S_h - (\phi_2 + \mu_h) V_C \\ \sigma_1 T_C - (\tau_2 + \mu_h) R_C \end{pmatrix}, \quad (14)$$

Using proposition (2.1), we have that,

$$S_h(t) = S_{h0} + I_t^{\eta}\left[\Lambda_h + \tau_2 R_C + \phi_2 V_C - \frac{(\beta_1 I_C + \beta_2 T_C)}{N_h} S_h - (\phi_1 + \mu_h) S_h\right],$$
$$E_C(t) = E_{C0} + I_t^{\eta}\left[\frac{(\beta_1 I_C + \beta_2 T_C)}{N_h} S_h - (\psi_2 + \mu_h) E_C\right] \quad (15)$$
$$I_C(t) = I_{C_0} + I_t^{\eta}[\psi_2 E_C - (\alpha_2 + \delta_1 + \mu_h) I_C],$$
$$V_C(t) = V_{C_0} + I_t^{\eta}[\phi_1 S_h - (\sigma_1 + \delta_5 + \mu_h) V_C],$$
$$T_C(t) = T_{C_0} + I_t^{\eta}[\alpha_2 I_C - (\phi_2 + \mu_h) T_C],$$
$$R_C(t) = R_{C0} + I_t^{\eta}[\sigma_1 T_C - (\tau_2 + \mu_h) R_C].$$

We obtain the Picard iteration of (15) as follows;

$$S_h(t) = S_{h0} + \frac{1}{\Gamma(\eta)}\int_0^t (t - \lambda_C)^{\eta-1} Z_1\left(\lambda, S_{h(n-1)}(\lambda_C)\right) d\,\lambda_C,$$
$$E_C(t) = E_{C0} + \frac{1}{\Gamma(\eta)}\int_0^t (t - \lambda_C)^{\eta-1} Z_2\left(\lambda_C, E_{C(n-1)}(\lambda_C)\right) d\lambda_C, \quad (16)$$
$$I_C(t) = I_{C0} + \frac{1}{\Gamma(\eta)}\int_0^t (t - \lambda_C)^{\eta-1} Z_3\left(\lambda_C, I_{C(n-1)}(\lambda_C)\right) d\lambda_C,$$
$$T_C(t) = T_{C0} + \frac{1}{\Gamma(\eta)}\int_0^t (t - \lambda_C)^{\eta-1} Z_4\left(\lambda_C, T_{C(n-1)}(\lambda_C)\right) d\lambda_C,$$
$$V_C(t) = V_{C0} + \frac{1}{\Gamma(\eta)}\int_0^t (t - \lambda_C)^{\eta-1} Z_5\left(\lambda_C, V_{C(n-1)}(\lambda_C)\right) d\lambda_C,$$
$$R_C(t) = R_{C0} + \frac{1}{\Gamma(\eta)}\int_0^t (t - \lambda_C)^{\eta-1} Z_6\left(\lambda_C, R_{C(n-1)}(\lambda_C)\right) d\lambda_C.$$

Transforming equation eq. (13) to get

$$X(t) = X(0) + \frac{1}{\Gamma(\eta)}\int_0^t (t - \lambda_C)^{\eta-1} Z\left(\lambda_C, X(\lambda_C)\right) d\,\lambda_C. \quad (17)$$

Lemma 1, The equation (14) gives us the definition of the Lipchitz condition which vector satisfies; $Z(t, K(t))$ on a set $[0, W[]_+^6]$ with the Lipchitz constant given as;

$$\omega = max\left(\left(\beta_1^* + \beta_2^* + \phi_1 + \mu_h\right), (\psi_2 + \mu_h), (\alpha_2 + \delta_1 + \mu_h), (\sigma_1 + \delta_5 + \mu_h), (\phi_2 + \mu_h), (\tau_2 + \mu_h)\right).$$

Proof:

$$\|Z_1(t, S_h) - Z_1(t, S_{h1})\|,$$
$$= \|\Lambda_h + \tau_2 R_C + \phi_2 V_C - \lambda_C S_h - (\phi_1 + \mu_h) S_h, -\Lambda_h + \tau_2 R_C + \phi_2 V_C - \lambda_C S_h - (\phi_1 + \mu_h) S_{h1}\|,$$
$$= \left\|-\Lambda_h + \tau_2 R_C + \phi_2 V_C - \lambda_C S_h - (\phi_1 + \mu_h)\left(S_h - S_{h_1}\right) + \mu_h\left(S_h - S_{h_1}\right)\right\| \leq \left(\beta_1^* + \beta_1^* + \phi_1 + \mu_h\right)\|S_h - S_{h1}\| + \mu_h\|S_h - S_{h1}\|, \quad \therefore \|Z_1(t, S_h) - Z_1(t, S_{h1})\| \leq \left(\beta_1^* + \beta_2^* + \phi_1 + \mu_h\right)\|S_h - S_{h1}\|.$$

Similarly, we obtained the following:

$$\left\|Z_2\left(t, \mathrm{E}_C\right) - Z_2\left(t, \mathrm{E}_{C1}\right)\right\| \leq \left(\psi_2 + \mu_h\right)\left\|\mathrm{E}_C - \mathrm{E}_{C1}\right\|,$$
$$\left\|Z_3\left(t, \mathrm{I}_C\right) - Z_3\left(t, \mathrm{I}_{C1}\right)\right\| \leq \left(\alpha_2 + \delta_1 + \mu_h\right)\left\|I_C - I_{C1}\right\|,$$
$$\left\|Z_4\left(t, \mathrm{T}_C\right) - Z_4\left(t, \mathrm{T}_{41}\right)\right\| \leq \left(\sigma_1 + \delta_5 + \mu_h\right)\left\|T_C - T_{C1}\right\|, \quad (18)$$
$$\left\|Z_5\left(t, \mathrm{V}_C\right) - Z_5\left(t, \mathrm{V}_{C1}\right)\right\| \leq \left(\phi_2 + \mu_h\right)\left\|V_C - V_{C1}\right\|,$$
$$\|Z_6(t, R_C\quad) - Z_6(t, R_{C1}\quad)\| \leq (\tau_2 + \mu_h)\|R_C - R_{C1}\|.$$

Where we obtained:

$$\|Z(t, K_1(t)) - Z(t, K_2(t))\| \leq \omega\|K_1 - K_2\|,$$
$$\omega = max\left(\left(\beta_1^* + \beta_2^* + \phi_1 + \mu_h\right), (\psi_2 + \mu_h), (\alpha_2 + \delta_1 + \mu_h), (\sigma_1 + \delta_5 + \mu_h), (\phi_2 + \mu_h), (\tau_2 + \mu_h)\right). \quad (19)$$

Lemma 2: The initial value problem (6), (7) in Eq. (19) exists and will have a unique solution $K(t) \in D_c^0(E)$.

Applying PicardLindelöfand fixed-point conjecture, we consider the solution of

$$K(t) = S_h\big(K(t)\big),$$

where S is defined as the Picard operator expressed as:

$$S_h: D_c^0(E, R_+^6) \to D_c^0(E, R_+^6).$$

Therefore,

$$S_h\big(K(t)\big) = K(0) + \frac{1}{\Gamma(\eta)}\int_0^t (t - \lambda_C)^{\eta-1} Z\,(\lambda_C, K(\lambda_C))\,d\,\lambda_C.$$

which becomes,

$$\left\|S_h\big(K_1(t)\big) - S_h\big(K_2(t)\big)\right\|$$
$$= \left\|\frac{1}{\Gamma(\eta)}\left[\int_0^t (t - \lambda_C)^{\eta-1} Z(\lambda_C, K_1(\lambda_C)) - Z(\lambda_C, K_2(\lambda_C))\,d\,\lambda_C\right]\right\|$$

$\leq \frac{1}{\Gamma(\eta)}\int_0^t (t-\lambda_C)^{\eta-1}\,\|Z(\lambda_C, K_1(\lambda_C)) - Z(\lambda_C, K_2(\lambda_C))\,d\,\lambda_C\|.$
$\leq \frac{\omega}{\Gamma(\eta)}\int_0^t (t-\lambda_C)^{\eta-1}\,\|K_1 - K_2\| d\lambda_C.$
$\left\|S_h\big(K_1(t)\big) - S_h\big(K_2(t)\big)\right\| \leq \frac{\omega}{\Gamma(\eta+1)S_h}.$
When $\frac{\omega}{\Gamma(\eta+1)}S_h \leq 1,$
then the Picard operator gives a contradiction, so Eq. (6), (7) solution is unique.
Lemma 2: The initial value problem (6), (7) in Eq. (19) exists and will have a unique solution.
$X(t) \in A_c^0(f).$
Using Picard-Lindelöf and fixed- point theory, we consider the solution of
$X(t) = S_h(X(t)),$
where S is defined as the Picard operator expressed as;
$S_h: A_c^0(f, R_+^6) \to A_c^0(f, R_+^6).$
Therefore,
$S_h\big(X(t)\big) = X(0) + \frac{1}{\Gamma(\eta)}\int_0^t (t-\lambda_C)^{\eta-1} Z\,(\lambda_C, X(\lambda_C))\,d\,\lambda_C.$
This becomes,
$\|S_h(X_1(t)) - S_h(X_2(t))\|$
$= \left\|\frac{1}{\Gamma(\eta)}\left[\int_0^t (t-\lambda_C)^{\eta-1} Z(\lambda_C, X_1(\lambda_C)) - Z(\lambda_C, X_2(\lambda_C))\,d\,\lambda_C\right]\right\|,$
$\leq \frac{1}{\Gamma(\eta)}\int_0^t (t-\lambda_C)^{\eta-1}\,\|Z(\lambda_C, X_1(\lambda_C)) - Z(\lambda_C, X_2(\lambda_C))\,d\,\lambda_C\|.$
$\leq \frac{\psi}{\Gamma(\eta)}\int_0^t (t-\lambda_C)^{\eta-1}\,\|X_1 - X_2 \quad\| d\lambda_C.$
$\|S_h(X_1(t)) - S_h(X_2(t))\| \leq \frac{\psi}{\Gamma(\eta+1)S_h}.$
When $\frac{\psi}{\Gamma(\eta+1)}S_h \leq 1,$ then the Picard operator gives a contradiction,
So Eq. (6), (7) solution is unique.

Fractional Order Model Numerical Results

The generalized fractional Adams-Bashforth-Moulton step-by-step technique by Bonyah. et al. (2020) was used to numerically solve the same fractional-order Chlamydia model. Table 1 indicates the parameters values of the model and Table 2 indicates different fractional values of the order used and simulated by the model.

Implementation of Fractional Adams–Bashforth–Moulton Method

The method described by Baskonus. and Bulut (2015) has been employed by the current study. Approximate solution of fractional Chlamydia model (6) obtained by the fractional Adams-Bashforth-Moulton method is as follows. The fractional heterogeneous form (6) is, then, obtained.

$${}^{c}D_t^{\eta} H(t) = Q(t, q(t)), 0 < t < \omega, \tag{20}$$
$H^{(n)}(0) = H_0^{(n)}, n = 1,0,\ldots, q, q = [\alpha].$
The $H = \big(S_h^*, E_C^*, I_C^*, T_C, V_C^*, R_C^* \quad\big) \in R_+^6$ and $V(t, q(t))$
the above (open-ended delivery order (27) may thus be written in terms of the concept of fractional integral as;
$$H(t) = \sum_{n=0}^{m-1} H_0^{(n)} \frac{t^n}{n!} + \frac{1}{\Gamma(\eta)}\int_0^t (t-y)^{\eta-1} R\big(k, m\,(k)\big)dk \tag{21}$$
Using the method described in Amos et al.(2024), we let the step size $g = \frac{\omega}{N}, N \in \mathrm{N}$ with a grid that is uniform on $[0, \omega]$. Where $t_c = cr, c = 0,1,1,\ldots N$. Thus, and fractional order model of Chlamydia model could be well approximated as (6) creates:

$$S_{h(k+1)}(t) = S_{h0} + \frac{g^{\eta}}{\Gamma(\eta+2)}\left\{\Lambda_h + \tau_2 R_C^n + \phi_2 V_C^n - \frac{(\beta_1 I_C + \beta_2 T_C)}{N_h^n} S_C^n - (\phi_1 + \mu_h) S_C^n\right\} + \frac{g^{\eta}}{\Gamma(\eta+2)}\sum_{y=0}^{k} dy, k+1\left\{\Lambda_h + \tau_2 R_{Cy} + \phi_2 V_{Cy} - \frac{(\beta_1 I_C + \beta_2 T_C)}{N_{hy}} S_{hy} - (\phi_1 + \mu_h) S_{hy}\right\},$$
$$E_{C(k+1)}(t) = E_{C0} + \frac{g^{\eta}}{\Gamma(\eta+2)}\left\{\frac{(\beta_1 I_C^n + \beta_2 T_C^n)}{N_h^n} S_C^n - (\psi_2 + \mu_h) E_C^n\right\} + \frac{g^{\eta}}{\Gamma(\eta+2)}\sum_{y=0}^{k} dy, k+1\left\{\frac{(\beta_1 I_{Cy} + \beta_2 T_{Cy})}{N_{hy}} S_{hy} - (\psi_2 + \mu_h) E_{Cy}\right\}, \tag{22}$$
$$I_{C(k+1)}(t) = I_0 + \frac{g^{\eta}}{\Gamma(\eta+2)}\{\psi_2 E_C^n - (\alpha_2 + \delta_1 + \mu_h) I_C^n\} + \frac{g^{\eta}}{\Gamma(\eta+2)}\sum_{y=0}^{k} dy, k+1\{\psi_2 E_{Cy} - (\alpha_2 + \delta_1 + \mu_h) I_{Cy}\},$$
$$T_{C(k+1)}(t) = T_{C0} + \frac{g^{\eta}}{\Gamma(\eta+2)}\{\alpha_2 I_C^n - (\sigma_1 + \delta_5 + \mu_h) T_C^n\} + \frac{g^{\eta}}{\Gamma(\eta+2)}\sum_{y=0}^{k} dy, k+1\{\alpha_2 I_{Cy} - (\sigma_1 + \delta_5 + \mu_h) T_{Cy}\},$$
$$V_{C(k+1)}(t) = V_{C0} + \frac{g^{\eta}}{\Gamma(\eta+2)}\{\phi_1 S_h^n - (\phi_2 + \mu_h) V_C^n\} + \frac{g^{\eta}}{\Gamma(\eta+2)}\sum_{y=0}^{k} dy, k+1\{\phi_1 S_{hy} - (\phi_2 + \mu_h) V_{Cy}\},$$

$R_{C(k+1)}(t) = R_{C0} + \frac{g^{\eta}}{\Gamma(\sigma+2)}\{\sigma_1 T_C^n - (\tau_2 + \mu_h)R_C^n\} + \frac{g^{\eta}}{\Gamma(\eta+2)}\sum_{y=0}^{k} dy, k+1\{\sigma_1 T_{Cy} - (\tau_2 + \mu_h)R_{Cy}\}.$

Where

$$S_{h(k+1)}^n(t) = S_{h0} + \frac{1}{\Gamma(\eta)}\sum_{y=0}^{k} f_{y,k+1}\left\{\Lambda_h + \tau_2 R_{Cy} + \phi_2 V_{Cy} - \frac{(\beta_1 I_C + \beta_2 T_C)}{N_{hy}} S_{hy} - (\phi_1 + \mu_h)S_{hy}\right\},$$

$$E_{C(k+1)}^n(t) = E_{C0} + \frac{1}{\Gamma(\eta)}\sum_{y=0}^{k} f_{y,k+1}\left\{\frac{(\beta_1 I_{Cy} + \beta_2 T_{Cy})}{N_{hy}} S_{hy} - (\psi_2 + \mu_h)E_{Cy}\right\}, \quad (23)$$

$$I_{C(k+1)}^n(t) = I_{C0} + \frac{1}{\Gamma(\eta)}\sum_{y=0}^{k} f_{y,k+1}\{\psi_2 E_{Cy} - (\alpha_2 + \delta_1 + \mu_h)I_{Cy}\},$$

$$T_{C(k+1)}^n(t) = T_{C0} + \frac{1}{\Gamma(\eta)}\sum_{y=0}^{k} f_{y,k+1}\{\alpha_2 I_{Cy} - (\sigma_1 + \delta_5 + \mu_h)T_{Cy}\},$$

$$V_{C(k+1)}^n(t) = V_{C0} + \frac{1}{\Gamma(\eta)}\sum_{y=0}^{k} f_{y,k+1}\{\phi_1 S_{hy} - (\phi_2 + \mu_h)V_{Cy}\},$$

$$R_{C(k+1)}^n(t) = R_{C0} + \frac{1}{\Gamma(\eta)}\sum_{y=0}^{k} f_{y,k+1}\{\sigma_1 T_{Cy} - (\tau_2 + \mu_h)R_{Cy}\}.$$

From (29) and (30) obtained;

$dy_{,K+1} = K^{\eta+1} - (k-\eta)(k+\eta)^{\eta}, y = 0.$

$(k-y+2)^{\eta+1} + (k-\eta)^{\eta+1} - 2(k-y+1)^{\eta+1}, 1 \le y \le k$

and $f_{y,k+1} = \frac{g^{\eta}}{\eta}[(k-y+1)^{\eta}(k-y)^{\eta}], 0 \le y \le k.$

Importance of using the Fractional Adam-Bashforth Moulton Method in Obtaining the Numerical Solutions of the Model

i. The fractional Adams-Bashforth-Moulton scheme merely requires an additional evaluation of a single function per step and is of high order of accuracy.
ii. An advantage of this approach is built-in error control, and it can frequently be used to implement ODE solvers to perform integration.

Table 2: Parameter Values and Sources

Parameter	Value	Source
Λ	0.007	Joseph et al. (2025)
β_1	0.3425	WHO (2022)
β_2		Joseph et al. (2025)
ψ_2	0.21	Estimated
α_2	0.1	Joseph et al. (2025)
σ_1	0.05	Joseph et al. (2025)
μ_h	0.012	WHO (2021)
τ_2	0.5	Estimated
ϕ_1	0.4	WHO (2021)
ϕ_2	0.67	WHO (2021)
δ_1	0.0054	Estimated
δ_2	0.0023	Estimated

Numerical Simulation

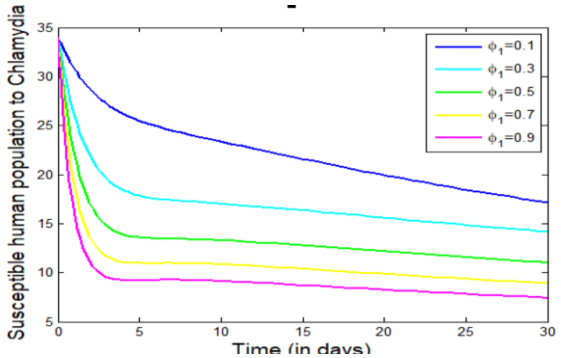

Figure 2a: Simulation of Susceptible humans to Chlamydia

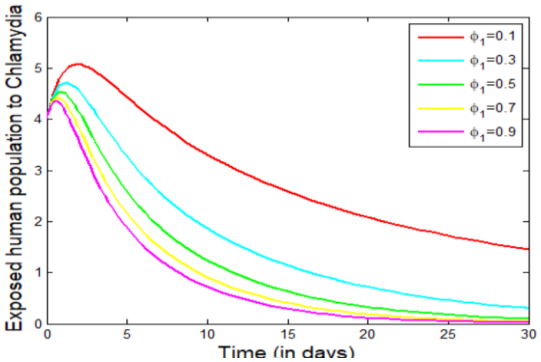

Figure 2b: Simulation of Exposed humans to Chlamydia

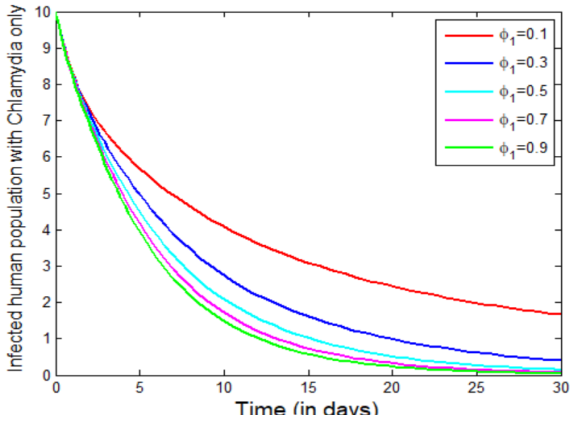

Figure 2c: Simulation of Infected humans population with Chlamydia

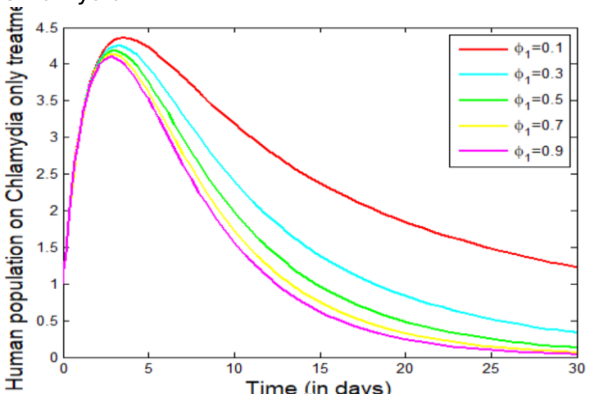

Figure 2d: Simulation of humans on Chlamydia treatment

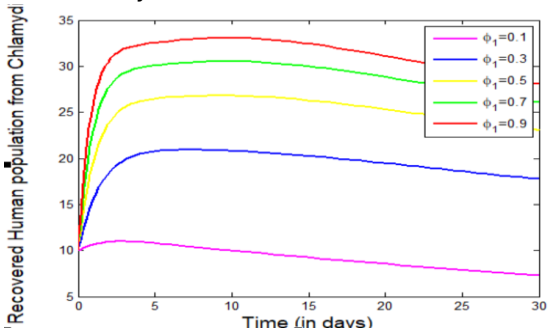

Figure 2e: Simulation of Recovered humans from Chlamydia

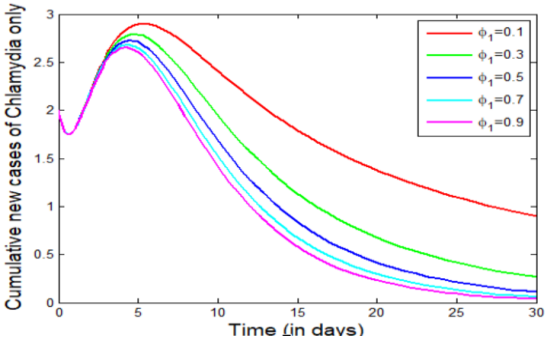

Figure 2f: Simulation of cumulative new cases of Chlamydia

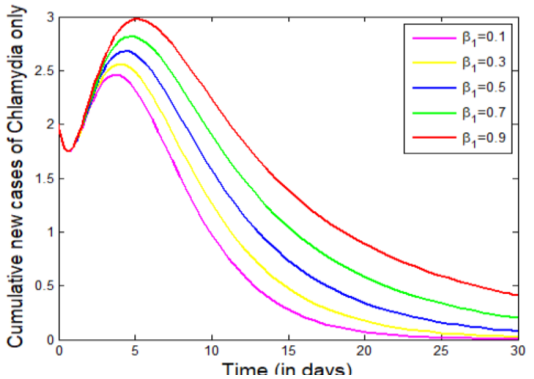

Figure 2g: Simulation of cumulative new cases of Chlamydia

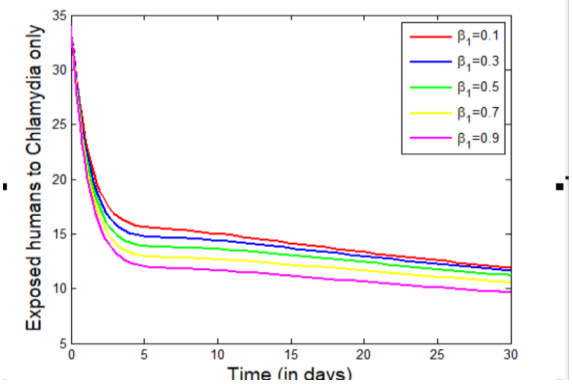

Figure 2h: Simulation of Exposed human population to Chlamydia

(2a) demonstrates computational modeling of the effects of vaccination rate (ϕ_1)among susceptible individuals on the susceptible human population. As can be seen, increased rate (ϕ_1) of vaccination contributes to lessening of the susceptible human to Chlamydia.
(2b) illustrates the modeling of how a vaccination rate (ϕ_1) of susceptible human population to Chlamydia affects exposed people. It is possible to note that the further increase in the rate (ϕ_1) of vaccination will result in decreasing the number of humans exposed to Chlamydia.
(2c) presents the simulation of effects of the rate (ϕ_1)of vaccination of susceptible human population to Chlamydia on infected human population with Chlamydia. This illustrates that the increment in the rate (ϕ_1)of vaccination lead to the declining of the level of infected human by the Chlamydia.
(2d) illustrates the result of the simulation of meaningfulness of the rate of vaccination (ϕ_1) of susceptible human population against the Chlamydia to human population on Chlamydia treatment. It has been noted that the higher the rate of vaccination (ϕ_1)the fewer the human beings who are under treatment in terms of Chlamydia disease.
(2e) exhibits the simulation of the influence of the rate of vaccination (ϕ_1) on people, who were susceptible to Chlamydia, on the population of humans who have already recovered, but are not immune to Chlamydia. This implies that the more the human population is vaccination of human population (ϕ_1)the lower it is recovered with Chlamydia disease.
(2f) illustrates the simulated effect of the rate of human vaccination (ϕ_1) against Chlamydia on the cumulatively new cases of Chlamydia. This implies that the great population coverage of vaccination has led to the reduced cases of the new instances of the Chlamydia disease.
(2g) shows the simulation of the effect of contact rate (β_1) of susceptible human population and infected human population with Chlamydia on cumulative new cases of Chlamydia. It is observed that, in effect, as the contact rate (β_1) increases the number of new cases of Chlamydia increases.
(2h) shows the simulation of the impact of contact rate (β_1) of susceptible humans and infected human population to Chlamydia on exposed human population with Chlamydia. It is noted that, the faster the rate of contact (β_1)the higher the population of exposed human being to Chlamydia.

CONCLUSION

In this article, we applied Caputo fractional derivative as a fraction operator in developing a mathematical model that could enable us explore the transmission of Chlamydia and the actions taken to thwart the same. Our study triggered a profound theorization of this fractional Chlamydia model since it should be noted that fractional modeling has been deemed essential in the management of this disease. It numerically solved the mathematical model in the fractional Adams-Bashforth-Moulton scheme. Fractional orders of the Caputo operator and parameters of the model defined the evolution of the disease incidence according to the simulation. We carried out a numerical simulation of a modulus of variation in intensity of immunization of susceptible people and the exposure rate of the infected human population. The resultant implication was that, an increase in the vaccination level will decrease the prevalence rate of Chlamydia whereas a decrease in the contact rate would drasticize the prevalence rate of Chlamydia amongst the general population. The outcome of the study is recommended to address the non-linear types of partial differentiations by using the analytical process of solving of partial differentiations as described by Amos et al. (2024).

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