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RESEARCH ARTICLE

Numerical Investigation of a Chlamydia Epidemic Model

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Abstract In this article, we analysed the numerical study of five compartmental epidemic model with Chlamydia infection. The population is divided into five classes: susceptible, exposed, infected in the asymptomatic phase, infected in the symptomatic phase and recovered (SEI_AI_SR). This model was shown to have two equilibrium points: a disease-free equilibrium and an endemic equilibrium. The local stability was defined using the computed effective reproduction number R_0, as well as the sensitivity of variables. When $R_0 < 1$, the disease-free equilibrium (ε_0) is locally asymptotically stable, and if $R_0 > 1$, the endemic equilibrium (ε_1) is locally asymptotically stable. This model is solved numerically using three numerical techniques: forward Euler, RK-4, and proposed non-standard finite difference (NSFD) techniques. The (NSFD) technique becomes a more efficient and reliable numerical technique than the forward Euler and RK-4 techniques. The NSFD technique retains all essential characteristics of a continuous (SEI_AI_SR) chlamydia epidemic model, like positivity and stability of equilibria. In contrast, well-known forward Euler and RK-4 techniques cannot sustain these characteristics. Furthermore, the NSFD technique is independent of time step size, while forward Euler and RK-4 depend on the time step size. The numerical simulations with a numerical test were represented to validate all the results.

Keywords: Chlamydia disease, mathematical model, reproductive number analysis, Euler, RK4, NSFD.

Introduction

Chlamydia trachomatis is the most commonly reported bacterial sexually transmitted infection (STI) in high-income countries and is highly prevalent globally [1,2]. While prevalence rates are relatively consistent across high-income economies [3], with pooled estimates of around 4.3% for women and 3.6% for men aged 18–26, the infection remains a significant public health concern. Many diseases are asymptomatic, regardless of the site of infection, contributing to its persistence. If left untreated, chlamydia can lead to serious health complications, including pelvic inflammatory disease (PID), chronic pelvic pain, infertility, and ectopic pregnancy in women, as well as urethritis and epididymitis in men. Despite advances in diagnostic techniques, such as highly sensitive and noninvasive nucleic acid amplification tests (NAATs), and the implementation of large-scale 'test and treat' programs over the past decades, the prevalence of chlamydia remains high [4]. These programs have had varied outcomes and, in some cases, unintended consequences, including overdiagnosis, overtreatment, relationship disruptions, and potential contributions to antimicrobial resistance. Additionally, early detection and treatment may hinder the development of immunity, increasing susceptibility to reinfection and reducing herd immunity. A comprehensive control strategy is crucial to address the persistent infection and its associated health and economic burdens [5].

Studying infectious diseases requires a multidisciplinary approach to understand their dynamics and impact fully. Chlamydia, a prevalent sexually transmitted infection, not only poses significant public health challenges but also incurs substantial economic costs due to treatment, loss of productivity, and

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long-term health complications [6]. By integrating numerical modelling with an analysis of socio-economic impacts, this study aims to provide a holistic perspective on controlling Chlamydia. This dual approach enables the identification of cost-effective intervention strategies, offering valuable insights for policymakers and healthcare professionals to mitigate the disease's epidemiological and economic burden.

Despite extensive efforts to control Chlamydia trachomatis (CT) infection, its prevalence remains high, with large-scale "test and treat" programs implemented over the past few decades yielding mixed results [7]. While these programs aim to reduce CT prevalence, they may inadvertently cause adverse effects such as relationship break-ups, overdiagnosis, overtreatment, and the potential for antimicrobial resistance. Furthermore, early detection and treatment may hinder the development of a robust immune response, increasing susceptibility to reinfection and reducing herd immunity. Studies suggest that CT infection confers partial immunity against reinfection, supported by evidence from laboratory animals and human observations indicating declining prevalence with age and reduced organism load on reinfection. Mathematical modelling estimates primary CT infection to provide over 65% protection against reinfection, fueling optimism for a partially effective CT vaccine, though vaccine development is still in the early stages [8]. Mathematical models play a crucial role in understanding CT transmission dynamics and evaluating the cost-effectiveness of novel strategies, such as routine screening for men, to prevent severe outcomes like pelvic inflammatory disease (PID) in women and epididymitis in men [9]. Preclinical vaccine research has utilized animal models, including guinea pigs, pigs, and non-human primates, offering insights despite limitations. These models provide a foundation for assessing vaccine-induced immune responses and correlates of protection, which is critical for advancing vaccine development against this persistent STI [10].

Numerical analysis provides a rigorous framework for understanding the transmission dynamics and control strategies of infectious diseases like Chlamydia. It is equally important to consider the socio-economic implications of these epidemics. The history of mathematical modelling related to Chlamydia reveals its evolution as a critical tool for studying the infection's transmission, control, and impact. Early models primarily focused on understanding the dynamic characteristics and contact structures of sexually transmitted diseases, including Chlamydia [11, 12]. Compartmental models were developed to explore Chlamydia resurgence, emphasizing the necessity of vaccines in the absence of interventions to alter sexual networks [1, 13]. Over time, more sophisticated approaches emerged, such as age-structured deterministic models that stratified populations by age, sexual behaviour, infection status, and vaccination status [14]. These models used nonlinear differential equations to simulate infection dynamics, often focusing on high-risk age groups and incorporating vaccination strategies informed by studies on other sexually transmitted infections. Further advancements included integrating co-dynamics models for Chlamydia trachomatis and gonorrhoea and exploring optimal control measures and targeted treatments. SEIRS models enhanced the understanding of Chlamydia transmission by analysing sensitivity, population turnover, and the effectiveness of screening programs [15, 16, 31], particularly highlighting the role of asymptomatic phases. Including sexual network behaviours and vaccination, effects marked a significant step in modelling approaches.

Chlamydia, a significant public health concern, has been studied using mathematical models to understand its transmission and impact. Researchers have explored its spread in human cells, co-infection with gonorrhea, and control strategies like pulse vaccination. Given its serious health and social effects, further research is essential [17,18,19]. Finding exact solutions for many physical systems is often complex or even impossible to achieve consistently. In the case of diseases, numerous mathematical models have been established [20, 21, 22], followed by the canonical approach of modelling the force of infection. Subsequent models have incorporated factors like vaccination and other diseases. Numerical methods have become essential in obtaining accurate solutions for differential equations [23]. Many dynamic systems exhibit key properties, such as boundedness and positivity, which numerical methods must preserve. Various techniques have been employed to solve dynamic systems [24]. This article presents a reliable mathematical approach to analyse nonlinear differential equation systems. The primary goal of this research is to validate the developed model through numerical simulations and evaluate its effectiveness by numerically tracking disease progression.

This paper is organized into seven sections. Section 1 provides a comprehensive literature review and background. Section 2 focuses on the formulation of the mathematical model, while Section 3 examines the equilibrium points of the system. Section 4 delves into the derivation of the basic reproduction number alongside sensitivity and stability analyses. Section 5 presents the results of numerical simulations, and Section 6 addresses the convergence analysis of the non-standard finite difference (NSFD) method. Finally, Section 7 concludes the study, summarizing its key findings and implications.

Model Formulation

This section examines a dynamic model [1] for the transmission of Chlamydia, a disease caused by the bacterium *Chlamydia trachomatis*. The model is based on the SE$I_A I_S$R framework, which categorizes the human population into five compartments: susceptible (S), exposed (E), infective in the asymptomatic phase (I_A), infective in the symptomatic phase (I_S), and recovered (R) [1]. The total population (N) remains constant over time and is given by N=S+E+I_A+I_S+R. The susceptible population increases through the recruitment rate (Λ), which includes newly sexually active individuals, migration, and previously recovered individuals who regain susceptibility. Susceptible individuals leave this compartment due to natural death, at a rate of μ, or recovery from infection. Infected individuals may initially exhibit no symptoms but remain capable of transmitting the disease, transitioning to the asymptomatic compartment at a rate of $\eta\rho$, where 0 < ρ <1. Alternatively, infected individuals may develop symptoms and move to the symptomatic compartment at a rate of $\eta(1-\rho)$. Recovered individuals can revert to the susceptible class at a rate of α.

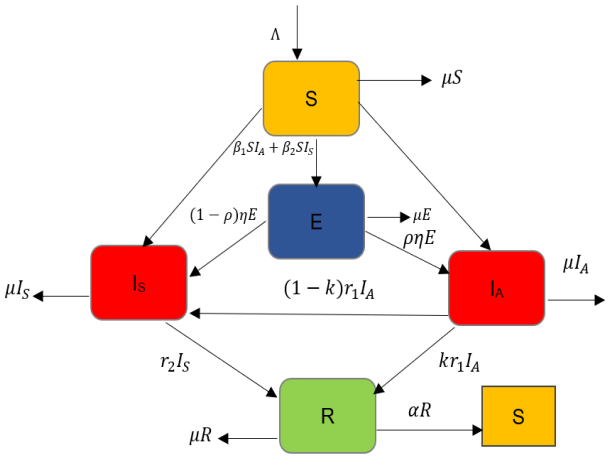

Figure 1. Flow chart

The total human population in **Figure 1** is represented by the letter N and is subdivided into five compartments

S +E +I_A +I_S +R=N
here,

S: Susceptible humans
E: Exposed
I_A: Infective individuals in the asymptomatic phase
I_S: Infective humans in the symptomatic phase
R: Recovered

$$\begin{aligned} dS/dt &= \Lambda - \beta_1 S(t) I_A(t) - \beta_2 S(t) I_S(t) - \mu S(t) + \alpha R(t) \\ dE/dt &= \beta_1 S(t) I_A(t) + \beta_2 S(t) I_S(t) - \rho\eta E(t) - \mu E(t) \\ dI_A/dt &= \rho\eta E(t) - (r_1 + \mu) I_A(t) \\ dI_S/dt &= (1-\rho)\eta E(t) + (1-k) r_1 I_A(t) - (r_2 + \mu) I_S(t) \\ dR/dt &= k r_1 I_A(t) + r_2 I_S(t) - \mu R(t) - \alpha R(t) \end{aligned} \quad (1)$$

Where all the parameters are positive β_1, β_2, α, η, ρ, r_1, r_2 , k and μ is > 0.

Definition of Parameters

System Explained by with the help of some parameters their details are below

Λ: Recruitment rate.
β_1: Infection transmission coefficient from asymptomatic phase to susceptible.
β_2: Infection transmission coefficient from symptomatic phase to susceptible.
μ: Natural death rate coefficient.
η (1 - ρ): Ratio of newly infected humans after showing infection symptoms move to class I_S.
ηρ: A fraction of newly infected individuals not showing infection symptoms move to class I_A.
(1 - κ) r_1: Asymptomatically infectious individuals eventually show disease symptoms and move to class I_S at a rate.
κr_1: Asymptomatically infectious humans that describe disease symptoms and recovery rate.
r_2: Infectious individuals in the symptomatic situation are apparent in infection and move to class R at rate.
α: Recovery rate

Theorem 1: For a given time (t), the system ensures the preservation of solution positivity within the framework of the system of equations.
Proof:

$$\left.\frac{dS}{dt}\right|_{S=0} = \Lambda \geq 0\ , \qquad \left.\frac{dE}{dt}\right|_{E=0} = \beta_1 S\, I_A + \beta_2 S\, I_S \geq 0, \qquad \left.\frac{dI_A}{dt}\right|_{I_A=0} = \rho\eta E \geq 0,$$

$$\left.\frac{dI_S}{dt}\right|_{I_S=0} = (1-\rho)\eta E + (1-\kappa) r_1 I_A \geq 0, \quad \left.\frac{dR}{dt}\right|_{R=0} = \kappa r_1 I_A + r_2 I_S \geq 0.$$

Which indicates that the solution is positive in the system.

Theorem 2: The system's solution is bounded in a feasible region.

Proof:

The total population is defined as

$S+E+I_A+I_S+R=N$.

Differentiate with respect to time on both sides

$$\frac{dN}{dt} = \frac{dS}{dt} + \frac{dE}{dt} + \frac{dI_A}{dt} + \frac{dI_S}{dt} + \frac{dR}{dt}\ ,$$

using the system's differential equations for each compartment, substitute each term on the right-hand side:

$$\frac{dN}{dt} = \Lambda N - \mu(S + E + I_A + I_S + R).$$

Since N(t) = S + E + I_A + I_S + R so, according to given condition

$$\frac{dN}{dt} = \Lambda - \mu N(t).$$

This is a first-order linear differential equation with the solution:

$$N(t) = \frac{\Lambda}{\mu} + \left(N(0) - \frac{\Lambda}{\mu}\right) e^{-\mu\tau},$$

As t→ ∞, the exponential term $e^{-\mu\tau} \to 0$. Therefore, N(t) approaches the steady state value

$$N(t) \leq \frac{\Lambda}{\mu}$$

So, the feasible region is

$$\Omega = \{(S, E, I_A, I_S, R)\ \mathcal{E}\ \mathbb{R}_+^5 \geq 0 \mid S+E+I_A+I_S+R \leq \frac{\Lambda}{\mu}\}. \qquad (2)$$

Equilibrium Points

Disease-free and endemic equilibrium points are discussed in this section. From the above system equations, we get disease-free equilibrium points are

$$\varepsilon_0 = (S_0,\ E_0,\ I_{A_0},\ I_{S_0},\ R_0) = (\tfrac{\Lambda}{\mu},0,0,0,0)\ . \tag{3}$$

From the system of equations after simplification, endemic equilibrium points are obtained, which are

$$\varepsilon_1 = \left(S_1^*, E_1^*, I_{A_1}^*, I_{S_1}^*, R_1^*\right)$$

$$\left.\begin{aligned}
S_1^* &= \left(\frac{\Lambda}{\mu R_0}\right),\\
E_1^* &= \frac{(r_1+\mu)I_{A_1}^*}{\rho\eta},\\
I_{A_1}^* &= \frac{\Lambda\rho\eta(\mu+\alpha)(r_2+\mu)\left(1-\frac{1}{R_0}\right)}{(\rho\eta+\mu)(\mu+\alpha)(r_1r_2+\mu(r_1+r_2)+\mu^2)-\alpha\eta(\kappa r_1\,\rho r_2+\kappa r_1\rho\mu+r_2r_1+r_2\mu-r_2\rho r_1-r_2\rho r_1-r_2\rho\mu+r_1\rho-\kappa r_1\rho)},\\
I_{S_1}^* &= \frac{[(1-\rho)(r_1+\mu)+\rho(1-\kappa)r_1]I_{A_1}^*}{(r_2+\mu)\rho},\\
R_1^* &= \frac{[\,\kappa r_1(r_2+\mu)\rho+r_2((1-\rho)(r_1+\mu)+(1-\kappa)\rho r_1)]I_{A_1}^*}{(\mu+\alpha)(r_2+\mu)\rho}\ .
\end{aligned}\right\} \tag{4}$$

Basic Reproductive Number

Let the infection-free equilibrium reign supreme. The structure is now referred to as the cutting-edge grid. The basic propagation value of the model's largest eigenvalue or another worldly sweep. The cutting-edge grid is a procedure used in the research of disease transformation to deduce the necessary multiplication number for a compartmental model of the spread of unstoppable diseases [20]. The estimate of the Jacobian networks F(x) and V (x)are prerequisites for the free balance ε_0. Following it, there is a constant state structure.

$$\frac{\mathrm{dF_i}}{\mathrm{dX_j}}(\varepsilon_0) = \begin{pmatrix} \mathrm{F} & 0 \\ 0 & 0 \end{pmatrix},$$

$$\frac{\mathrm{dV_i}}{\mathrm{dX_j}}(\varepsilon_0) = \begin{pmatrix} \mathrm{F} & 0 \\ \mathrm{J_3} & \mathrm{J_4} \end{pmatrix},$$

here, F and V are m × m matrix.

We apply the next-generation matrix technique to the system in this part to calculate the basic reproduction number by computing the transition and transmission matrices as follows by the next-generation matrix method. We have to take only the disease class

$$\frac{\mathrm{dE}}{\mathrm{dt}} = \beta_1 \mathrm{S\,I_A} + \beta_2 \mathrm{SI_S} - \mathrm{E}(\rho\eta + \mu).$$

$$\frac{\mathrm{dI_A}}{\mathrm{dt}} = \rho\eta\mathrm{E} - (\mathrm{r_1} + \mu)\mathrm{I_A}.$$

$$\frac{\mathrm{dI_S}}{\mathrm{dt}} = (1-\rho)\eta\mathrm{E} + (1-\kappa)\mathrm{r_1 I_A} - (\mathrm{r_2} + \mu)\mathrm{I_S}.$$

$\frac{\mathrm{dx}}{\mathrm{dt}} = F(x,y) - V(x,y)$.

$$F(x) = \begin{bmatrix} \beta_1 SI_A + \beta_2 SI_S \\ 0 \\ 0 \end{bmatrix},$$

$$\mathrm{V(x)} = \begin{bmatrix} \mathrm{E}(\rho\eta + \mu) \\ -\rho\eta\mathrm{E} + \mathrm{I_A}(\mathrm{r_1} + \mu) \\ -(1-\rho)\eta\mathrm{E} - \mathrm{I_A r_1}(1-\kappa) + \mathrm{I_S}(\mathrm{r_2} + \mu) \end{bmatrix}.$$

Thus, at the DFE, the transmission matrices F and V are,

$$\bar{F} = \begin{bmatrix} 0 & \beta_1 S & \beta_2 S \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix},$$

$$\bar{V} = \begin{bmatrix} \rho\eta + \mu & 0 & 0 \\ -\rho\eta & r_1 + \mu & 0 \\ -\eta(1-\rho) & -r_1(1-\kappa) & r_2 + \mu \end{bmatrix}.$$

The term $\bar{F}$ characterizes the progression of new infections, whereas $\bar{V}$ represents the transitions toward equilibrium. By computing the inverse of $\bar{V}$.

We obtain a measure of the average time spent within each compartment and the total number of secondary infections produced over the infectious period. Thus, this system's dominant eigenvalue determines the basic reproduction number.

$$\bar{F}\bar{V}^{-1} = \frac{\begin{bmatrix} \beta_1 \frac{\lambda}{\mu}\rho\eta(r_2+\mu) + \beta_2 \frac{\lambda}{\mu}(r_1\rho\eta(1-\kappa) + \eta(1-\rho)(r_1+\mu)) & \beta_1 \frac{\lambda}{\mu}(\rho\eta+\mu)(r_2+\mu) + \beta_2 \frac{\lambda}{\mu}(r_1(\rho\eta+\mu)(1-\kappa)) & \beta_2 \frac{\lambda}{\mu}(\rho\eta+\mu)(r_1+\mu) \\ -\rho\eta & r_1+\mu & 0 \\ -\eta(1-\rho) & -r_1(1-\kappa) & r_2+\mu \end{bmatrix}}{(\rho\eta + \mu)(r_1 + \mu)(r_2 + \mu)}.$$

The most significant value in the matrix is known as Spectral radii; therefore, we take the greatest value as R_0, which is
So,

$$R_0 = \frac{(\Lambda/\mu)\,[\beta 1\rho\eta(r_2+\mu)+\beta_2\eta(\rho(1-\kappa)r_1+(r_1+\mu)(1-\rho))]}{(\rho\eta+\mu)(r_1+\mu)(r_2+\mu)} \quad (5)$$

Sensitivity Analysis of Parameters

By calculating the partial derivative of **R_0** with respect to each parameter to determine if the change in R_0 is positive or negative for each parameter.

$$\text{Sensitivity Analysis} = \frac{\text{Parameter}}{R_0} \times \frac{\text{partial derivative } R_0}{\text{partial derivative Parameter}}$$

According to the analysis, certain factors have negative sensitivity indices, while others show positive sensitivity. This suggests that while certain parameters have an inverse relationship with the reproduction number, others have a direct relationship with R_0, as one of them is represented in Figure **2**.

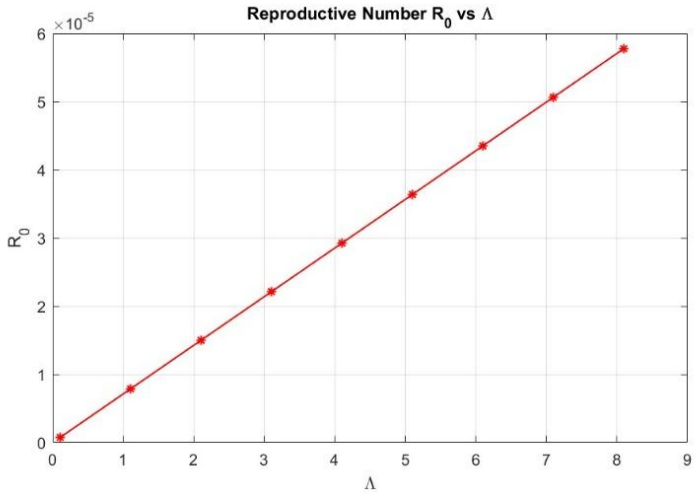

Figure 2. Performance of R_0 due to Λ

Local Stability of Disease-Free and Endemic Equilibrium Points

With the help of stability analysis at disease-free and endemic points, we can easily justify the Reproductive number effect with these points.

Theorem 3: The disease-free equilibrium of system point (ε_0) is locally asymptotically stable if $R_0 < 1$.

Proof: System of differential equations

$\frac{dS}{dt} = \Lambda - \beta_1 S I_A - \beta_2 S I_S - \mu S + \alpha R$,

$\frac{dE}{dt} = \beta_1 S I_A + \beta_2 S I_S - \rho\eta E - \mu E$,

$dI_A/dt = \rho\eta E - (r_1 + \mu) I_A$,

$\frac{dI_S}{dt} = (1-\rho)\eta E + (1-\kappa) r_1 I_A - (r_2 + \mu) I_S$,

$dR/dt = \kappa r_1 I_A + r_2 I_S - \mu R - \alpha$.

To create the Jacobian matrix of the system at DFE, we assumed,

F= $\Lambda - \beta_1 S I_A - \beta_2 S I_S - \mu S + \alpha R$,

$G = \beta_1 S I_A + \beta_2 S I_S - \rho\eta E - \mu E$,

$H = \rho\eta E - (r_1 + \mu) I_A$,

$M = (1-\rho)\eta E + (1-\kappa) r_1 I_A - (r_2 + \mu) I_S$,

$N = \kappa r_1 I_A + r_2 I_S - \mu R - \alpha$.

Now, differentiate F, G, H, M, and N with respect to each compartmental and then put the disease-free points. Now, J will be

$$J = \begin{bmatrix} F_S & F_E & F_{I_A} & F_{I_S} & F_R \\ G_S & G_E & G_{I_A} & G_{I_S} & G_R \\ H_S & H_E & H_{I_A} & H_{I_S} & H_R \\ M_S & M_E & M_{I_A} & M_{I_S} & M_R \\ N_S & N_E & N_{I_A} & N_{I_S} & N_R \end{bmatrix},$$

J at disease-free points is

$$J = \begin{bmatrix} -\mu & 0 & \frac{-\beta_1 \Lambda}{\mu} & \frac{-\beta_2 \Lambda}{\mu} & \alpha \\ 0 & -\rho\eta - & \frac{\beta_1 \Lambda}{\mu} & \frac{\beta_2 \Lambda}{\mu} & 0 \\ 0 & \rho\eta & -(r_1+\mu) & 0 & 0 \\ 0 & \eta(1-\rho) & (1-\kappa) r_1 & -r_2 - \mu & 0 \\ 0 & 0 & \kappa r_1 & r_2 & -\mu + \alpha \end{bmatrix}.$$

We calculated $det(J - \lambda I)$,

$$|J - \lambda I| = \begin{vmatrix} -\mu - \lambda & 0 & \frac{-\beta_1 \Lambda}{\mu} & \frac{-\beta_2 \Lambda}{\mu} & \alpha \\ 0 & -\rho\eta - \mu - \lambda & \frac{\beta_1 \Lambda}{\mu} & \frac{\beta_2 \Lambda}{\mu} & 0 \\ 0 & \rho\eta & -(r_1+\mu) - \lambda & 0 & 0 \\ 0 & \eta(1-\rho) & (1-\kappa) r_1 & -r_2 - \mu - \lambda & 0 \\ 0 & 0 & \kappa r_1 & r_2 & -\mu + \alpha - \lambda \end{vmatrix}$$

$|J - \lambda I| = (-\mu - \lambda)(\mu + \alpha + \lambda)|A|$.

Here

$$A = \begin{vmatrix} -\rho\eta - \mu - \lambda & \beta_1 \frac{\Lambda}{\mu} & \beta_2 \frac{\Lambda}{\mu} \\ \rho\eta & -(r_1 + \mu) - \lambda & 0 \\ \eta(1-\rho) & (1-\kappa)r_1 & -r_2 - \mu - \lambda \end{vmatrix},$$

Now, find the remaining three eigenvalues using Routh-Hurwitz criteria.

By using characteristic equation

$P(\lambda) = \lambda^3 - A_1\lambda^2 + A_2\lambda - A_3 = 0$

Here,
−A1=Sum of diagonal,
A2=Sum of Co-diagonal,
and
−A3= Determinant of A
by Routh Hurwitz criterion, it analyses that P(λ) has negative real roots if $R_0 < 1$; hence, the system of differential equations shows local stability at DFE when $R_0 < 1$. This completes the proof.

Preposition: The endemic equilibrium points are locally asymptotically stable if $R_0 > 1$.

Numerical Analysis of the SEI_AI_SR System

We will employ numerical modelling techniques to address the problem. Initially, the Forward Euler scheme will be utilized, followed by the application of the Fourth-Order Runge-Kutta scheme and, ultimately, the Non-standard Finite Difference (NSFD) scheme.

Table 1. Parametric values

Parameters	Values	Reference
Λ	1.12	[1]
β_1	0.1	[1]
β_2	0.15	[1]
μ	0.15	[1]
α	0.5	[1]
η	0.5	[1]
ρ	0.7	[1]
r_1	0.7	[1]
r_2	0.8	[1]
κ	0.7	[1]

Euler Scheme

By the model's equations, the Euler scheme is represented as

$$S^{n+1} = S^n + h(\Lambda - \beta_1 S^n I_A^n - \beta_2 S^n I_S^n - \mu S^n + \alpha R^n), \quad (6.1)$$

$$E^{n+1} = E^n + h(\beta_1 S^n I_1^n + \beta_2 S^n I_S^n - \rho\eta E^n - \mu E^n), \quad (6.2)$$

$$I_A^{n+1} = I_A^n + h(\rho\eta E^n - (r_1 + \mu) I_A^n), \quad (6.3)$$

$$I_S^{n+1} = I_S^n + h\big((1-\rho)\eta E^n + (1-\kappa) r_1 I_A^n - (r_2 + \mu) I_S^n\big), \quad (6.4)$$

$$R^{n+1} = R^n + h(\kappa r_1 I_A^n + r_2 I_S^n - \mu R^n - \alpha R^n). \quad (6.5)$$

Runge-Kutta Fourth Order Method

We use the system to develop an explicit RK4 method.

$$K_1 = \mathrm{h}(\Lambda - \beta_1 S^n I_A^n - \beta_2 S^n I_S^n - \mu S^n + \alpha R^n), \tag{6.6}$$

$$l_1 = \mathrm{h}(\beta_1 S^n I_A^n + \beta_2 S^n I_S^n - \rho\eta E^n - \mu E^n), \tag{6.7}$$

$$m_1 = \mathrm{h}(\rho\eta E^n - (r_1 + \mu) I_A^n), \tag{6.8}$$

$$n_1 = \mathrm{h}\big((1-\rho)\eta E^n + (1-\kappa) r_1 I_A^n - (r_2 + \mu) I_S^n\big), \tag{6.9}$$

$$o_1 = \mathrm{h}(\kappa r_1 I_A^n + r_2 I_S^n - \mu R^n - \alpha R^n). \tag{6.10}$$

$$k_2 = \mathrm{h}\left(\Lambda - \beta_1 \left(S^n + \frac{k_1}{2}\right)\left(I_A^n + \frac{m_1}{2}\right) - \beta_2 \left(S^n + \frac{k_1}{2}\right)\left(I_S^n + \frac{n_1}{2}\right) - \mu\left(S^n + \frac{k_1}{2}\right) + \alpha\left(R^n + \frac{O_1}{2}\right)\right), \tag{6.11}$$

$$l_2 = \mathrm{h}\left(\beta_1 \left(S^n + \frac{k_1}{2}\right)\left(I_A^n + \frac{m_1}{2}\right) + \beta_2 \left(S^n + \frac{k_1}{2}\right)\left(I_S^n + \frac{n_1}{2}\right) - \rho\eta\left(E^n + \frac{l_1}{2}\right) - \mu\left(E^n + \frac{l_1}{2}\right)\right), \tag{6.12}$$

$$m_2 = \mathrm{h}\left(\rho\eta\left(E^n + \frac{l_1}{2}\right) - (r_1 + \mu)\left(I_A^n + \frac{m_1}{2}\right)\right), \tag{6.13}$$

$$n_2 = \mathrm{h}\left((1-\rho)\eta\left(E^n + \frac{l_1}{2}\right) + (1-\kappa) r_1 \left(I_A^n + \frac{m_1}{2}\right) - (r_2 + \mu)\left(I_S^n + \frac{n_1}{2}\right)\right), \tag{6.14}$$

$$o_2 = \mathrm{h}\left(\kappa r_1 \left(I_A^n + \frac{m_1}{2}\right) + r_2\left(I_S^n + \frac{n_1}{2}\right) - \mu\left(R^n + \frac{o_1}{2}\right) - \alpha\left(R^n + \frac{o_1}{2}\right)\right). \tag{6.15}$$

$$k_3 = \mathrm{h}\left(\Lambda - \beta_1 \left(S^n + \frac{k_2}{2}\right)\left(I_A^n + \frac{m_2}{2}\right) - \beta_2 \left(S^n + \frac{k_2}{2}\right)\left(I_S^n + \frac{n_2}{2}\right) - \mu\left(S^n + \frac{k_2}{2}\right) + \alpha\left(R^n + \frac{o_2}{2}\right)\right), \tag{6.16}$$

$$l_3 = \mathrm{h}\left(\beta_1 \left(S^n + \frac{k_2}{2}\right)\left(I_A^n + \frac{m_2}{2}\right) + \beta_2 \left(S^n + \frac{k_2}{2}\right)\left(I_S^n + \frac{n_2}{2}\right) - \rho\eta\left(E^n + \frac{l_2}{2}\right) - \mu\left(E^n + \frac{l_2}{2}\right)\right), \tag{6.17}$$

$$m_3 = \mathrm{h}(\rho\eta\left(\left(E^n + \frac{l_2}{2}\right) - (r_1 + \mu)\left(I_A^n + \frac{m_2}{2}\right)\right), \tag{6.18}$$

$$n_3 = \mathrm{h}\left((1-\rho)\eta\left(E^n + \frac{l_2}{2}\right) - (1-\kappa) r_1 \left(I_A^n + \frac{m_2}{2}\right) - (r_2 + \mu)\left(I_S^n + \frac{n_2}{2}\right)\right), \tag{6.19}$$

$$o_3 = \mathrm{h}\left(\kappa r_1 \left(I_A^n + \frac{m_2}{2}\right) + r_2\left(I_S^n + \frac{n_2}{2}\right) - \mu\left(R^n + \frac{o_2}{2}\right) - \alpha\left(R^n + \frac{o_2}{2}\right)\right). \tag{6.20}$$

$$k_4 = \mathrm{h}(\Lambda - \beta_1 (S^n + k_3)(I_A^n + m_3) - \beta_2 (S^n + K_3)(I_S^n + n_3) - \mu(S^n + k_3) + \alpha(R^n + O_3)\,), \tag{6.21}$$

$$l_4 = \mathrm{h}\big(\beta_1 (S^n + k_3)(I_A^n + m_3) + \beta_2 (S^n + k_3)(I_S^n + n_3) - \rho\eta(E^n + l_3) - \mu(E^n + l_3)\big), \tag{6.22}$$

$$m_4 = \mathrm{h}\big(\rho\eta(E^n + l_3) - (r_1 + \mu)(I_A^n + m_3)\big), \tag{6.23}$$

$$n_4 = \mathrm{h}\big((1-\rho)\eta(E^n + l_3) - (1-\kappa) r_1 (I_A^n + m_3) - (\mu + r_2)(I_S^n + n_3)\big), \tag{6.24}$$

$$o_4 = \mathrm{h}\big(\kappa r_1 (I_A^n + m_3) + r_2 (I_S^n + n_3) - \mu(R^n + o_3) - \alpha(R^n + o_3)\big). \tag{6.25}$$

$$S^{n+1} = S^n + \frac{1}{6}(k_1 + 2k_2 + 2k_3 + k_4). \tag{7}$$

$$E^{n+1} = E^n + \frac{1}{6}(l_1 + 2l_2 + 2l_3 + l_4). \tag{8}$$

$$I_A^{n+1} = I_A^n + \frac{1}{6}(m_1 + 2m_2 + 2m_3 + m_4). \tag{9}$$

$$I_S^{n+1} = I_S^n + \frac{1}{6}(n_1 + 2n_2 + 2n_3 + n_4). \tag{10}$$

$$R^{n+1} = R^n + \frac{1}{6}(o_1 + 2o_2 + 2o_3 + o_4)\,. \tag{11}$$

Non-Standard Finite Difference Scheme

$$S^{n+1} = \frac{S^n + \mathrm{h}\Lambda + \mathrm{h}\alpha R^n}{1 + \mathrm{h}\beta_1 I_A^n + h\beta_2 I_S^n + h\mu}, \tag{12.1}$$

$$E^{n+1} = \frac{E^n + \mathrm{h}\,\beta_1 S^n I_A^n + \mathrm{h}\,\beta_2 S^n I_S^n}{1 + \mathrm{h}\rho\eta + \mathrm{h}\mu}, \tag{12.2}$$

$$I_A^{n+1} = \frac{I_A^n + \mathrm{h}\rho\eta E^n}{1 + \mathrm{h}(r_1 + \mu)}, \tag{12.3}$$

$$I_S^{n+1} = \frac{I_S^n + \mathrm{h}\,(1-\rho)\eta E^n + h(1-\kappa) r_1 I_A^n}{1 + \mathrm{h}(r_2 + \mu)}\,, \tag{12.4}$$

$$R^{n+1} = \frac{R^n + \mathrm{h}\kappa r_1 I_A^n + \mathrm{h} r_2 I_S^n}{1 + \mathrm{h}\mu + \mathrm{h}\alpha}\,. \tag{12.5}$$

Stability Analysis of NSFD Scheme

The Jacobian matrix J was obtained by calculating the partial derivatives of the NSFD equations and substituting the disease-free equilibrium points.

$$C = S^{n+1} = \frac{S^n + \mathrm{h}\Lambda + \mathrm{h}\alpha R^n}{1 + \mathrm{h}\beta_1 I_A^n + h\beta_2 I_S^n + h\mu} \tag{12.6}$$

$$D = E^{n+1} = \frac{E^n + \mathrm{h}\,\beta_1 S^n I_A^n + \mathrm{h}\,\beta_2 S^n I_S^n}{1 + \mathrm{h}\rho\eta + \mathrm{h}\mu} \tag{12.7}$$

$$F = I_A^{n+1} = \frac{I_A^n + \mathrm{h}\rho\eta E^n}{1 + \mathrm{h}(r_1 + \mu)} \tag{12.8}$$

$$G = I_S^{n+1} = \frac{I_S^n + \mathrm{h}\,(1-\rho)\eta E^n + h(1-\kappa) r_1 I_A^n}{1 + \mathrm{h}(r_2 + \mu)} \tag{12.9}$$

$$H = R^{n+1} = \frac{R^n + \mathrm{h}\kappa r_1 I_A^n + \mathrm{h} r_2 I_S^n}{1 + \mathrm{h}\mu + \mathrm{h}\alpha} \tag{12.10}$$

Differentiating these C, D, F, G and H with respect to compartmental perimeters S, E, I_A, I_S and R. Now J will be

$$J = \begin{bmatrix} C_S & C_E & C_{I_A} & C_{I_S} & C_R \\ D_S & D_E & D_{I_A} & D_{I_S} & D_R \\ F_S & F_E & F_{I_A} & F_{I_S} & F_R \\ G_S & G_E & G_{I_A} & G_{I_S} & G_R \\ H_S & H_E & H_{I_A} & H_{I_S} & H_R \end{bmatrix},$$

$$J = \begin{bmatrix} \frac{1}{1+h\mu} & 0 & \frac{-h\beta_1(\frac{\Lambda}{\mu}+h\Lambda)}{(1+h\mu)^2} & \frac{-h\beta_2(\frac{\Lambda}{\mu}+h\Lambda)}{(1+h\mu)^2} & \frac{h\alpha}{1+h\mu} \\ 0 & \frac{1}{1+h\rho\eta+h\mu} & \frac{h\beta_1\frac{\Lambda}{\mu}}{1+h\rho\eta+h\mu} & \frac{h\beta_2\frac{\Lambda}{\mu}}{1+h\rho\eta+h\mu} & 0 \\ 0 & \frac{h\rho\eta}{1+h(r_1+\mu)} & \frac{1}{1+h(r_1+\mu)} & 0 & 0 \\ 0 & \frac{h\eta(1-\rho)}{1+h(r_2+\mu)} & \frac{hr_1(1-\kappa)}{1+h(r_2+\mu)} & \frac{1}{1+h(r_2+\mu)} & 0 \\ 0 & 0 & \frac{h\kappa r_1}{1+h\mu+h\alpha} & \frac{hr_2}{1+h\mu+h\alpha} & \frac{1}{1+h\mu+h\alpha} \end{bmatrix},$$

Schur-Cohn Stability Criteria For the characteristics of polynomial

$$P(\lambda)=\lambda^3+A_1\,\lambda^2+A_2\,\lambda+A_3$$

carrying solutions λ=1,2,3 of equation p(λ)=0 satisfy

- $p(1) = 1 + A_1 + A_2 + A_3 > 0$.
- $(-1)^3 p(-1) = 1 - A_1 + A_2 - A_3 > 0$.
- $1 - (A_3)^2 > |A_2 - A_1 A_3|$.

As the above three conditions are satisfied, the Schur-Cohn Stability Criterium states that all Eigenvalues lie in the unit circle, and this theorem confirms the local asymptotic stability of the developed NSFD scheme.

Consistency of the NSFD Scheme

The expressions were obtained by solving the NSFD equations with the Taylor series. By taking equation 12.1 and then applying the Taylor series

$$S^{n+1}[1 + h\beta_1 I_A^n + h\beta_2 I_S^n + h\mu] = S^n + h\Lambda + h\alpha R^n$$

Here if we expand the Taylor series for S^{n+1} then, we will get

$$S^{n+1} = S^n + h\frac{dS}{dt} + \frac{h^2}{2!}\frac{d^2S}{dt^2} + \frac{h^3}{3!}\frac{d^3S}{dt^3} + \cdots$$

$$\left(S^n + h\frac{dS}{dt} + \frac{h^2}{2!}\frac{d^2S}{dt^2} + \frac{h^3}{3!}\frac{d^3S}{dt^3} + \cdots\right)(1 + h\beta_1 I_A^n + h\beta_2 I_S^n + h\mu) = S^n + h\Lambda + h\alpha R^n$$

$$S^n + h\beta_1 I_A^n S^n + h\beta_2 I_S^n S^n + h\mu S^n + h\frac{dS}{dt} + h^2\frac{dS}{dt}\beta_1 I_A^n + h^2\frac{dS}{dt}\beta_2 I_S^n + h^2\mu\frac{dS}{dt} + \left(\frac{h^2}{2!}\frac{d^2S}{dt^2} + \frac{h^3}{3!}\frac{d^3S}{dt^3} + \cdots\right)(1 + h\beta_1 I_A^n + h\beta_2 I_S^n + h\mu) = S^n + h\Lambda + h\alpha R^n$$

$$h\left[\left(\beta_1 I_A^n S^n + \beta_2 I_S^n S^n + \mu S^n + \frac{dS}{dt} + h\frac{dS}{dt}\beta_1 I_A^n + h\frac{dS}{dt}\beta_2 I_S^n + h\mu\frac{dS}{dt}\right) + \left(\frac{h}{2!}\frac{d^2S}{dt^2} + \frac{h^2}{3!}\frac{d^3S}{dt^3} + \cdots\right)(1 + h\beta_1 I_A^n + h\beta_2 I_S^n + h\mu)\right] = h(\Lambda + \alpha R^n)$$

Applying limit h approaches to zero, so we got

$$\frac{dS}{dt} = \Lambda - \beta_1 S^n I_A^n - \beta_2 S^n I_S^n - \mu S^n + \alpha R^n$$

Now take equation 12.2 and then apply the Taylor expansion

$$E^{n+1}[1 + h\rho\eta + h\mu] = E^n + h\beta_1 S^n I_A^n + h\beta_2 S^n I_S^n$$

$$h\left[E^n\rho\eta + E^n\mu + \frac{dE}{dt} + h\rho\eta\frac{dE}{dt} + \left(\frac{h}{2!}\frac{d^2E}{dt^2} + \frac{h^2}{3!}\frac{d^3E}{dt^3}\right)(1 + h\rho\eta + h\mu)\right] = h[\beta_1 S^n I_A^n + \beta_2 S^n I_S^n]$$

Applying limit h→0

$$\frac{dE}{dt} = \beta_1 S^n I_A^n + \beta_2 S^n I_S^n - \rho\eta E^n - \mu E^n$$

Now with 12.3

$$I_A^{n+1}(1 + h(r_1 + \mu)) = I_A^n + h\rho\eta E^n$$

Applying the Taylor series and then h →0 we got

$$(r_1 + \mu)I_A^n + \frac{dI_A}{dt} = \rho\eta E^n$$

So, we got

$$\frac{dI_A}{dt} = \rho\eta E^n - (r_1 + \mu)I_A^n$$

By equation 12.4

$$I_S^{n+1}\big(1 + h(r_2 + \mu)\big) = I_S^n + h(1-\rho)\eta E^n + h(1-\kappa)r_1 I_A^n$$

With the same procedure, applying series and then h →0

$$\frac{dI_S}{dt} = (1-\rho)\eta E^n + (1-\kappa)r_1 I_A^n - (r_2 + \mu)I_S^n$$

At last, by equation 12.5

$$R^{n+1}(1 + h\mu + h\alpha) = R^n + h\kappa r_1 I_A^n + h r_2 I_S^n$$

By Taylor series expansion, we got

$$h\left[\mu R^n + \alpha R^n + \frac{dR}{dt} + h\mu\frac{dR}{dt} + h\alpha\frac{dR}{dt} + \left(\frac{h^2}{2!}\frac{d^2R}{dt^2} + \frac{h^3}{3!}\frac{d^3R}{dt^3}\right)(1 + h\mu + h\alpha)\right] = R^n + h\kappa r_1 I_A^n + h r_2 I_S^n$$

Now, by h →0 we got

$$\frac{dR}{dt} = \kappa r_1 I_A^n + r_2 I_S^n - \mu R^n - \alpha R^n$$

Hence, NSFD is consistent with the system.

Graphs at Different Points

Using Euler, Runge-Kutta 4 (RK4) and Non-Standard Finite Difference (NSFD) methods for this system provides accurate and stable approximate solutions, especially when applied to dynamic and long-term model base predictions [23, 24, 25]. Solutions available in Euler and RK4 are more straightforward and have increased accuracy, and RK-4 is especially suitable for differential system problems. NSFD, though, is especially useful since it retains all-important model properties. Thus, the system results do reflect realities of biological relevance even when the system is analyzed for longer periods. Such stability level makes NSFD especially useful for epidemiological models where sustainable, precise predictions are needed most.

a b

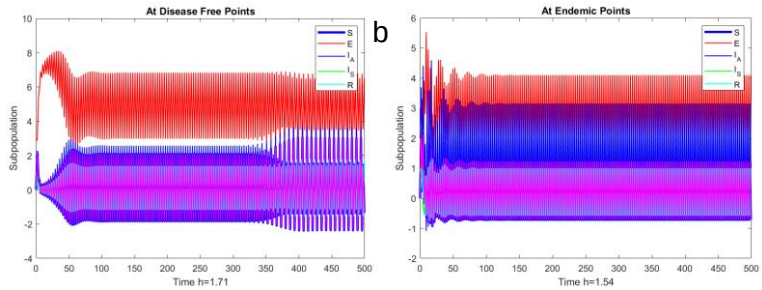

Figure 3. Euler behavior with (a) disease free points at h=1.71 and (b) with endemic points at h=1.54

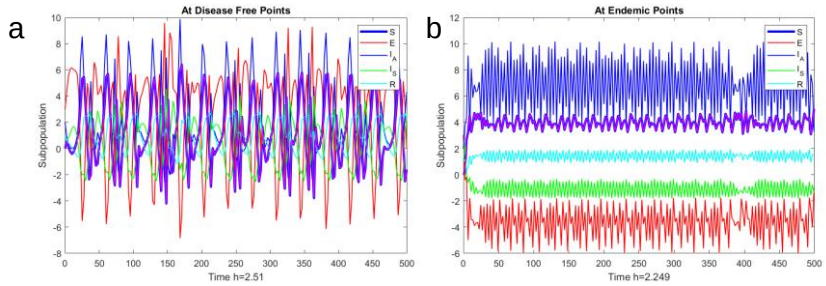

Figure 4. RK4 behavior with (a) disease free at h=2.51 and (b) with endemic points at h=2.249

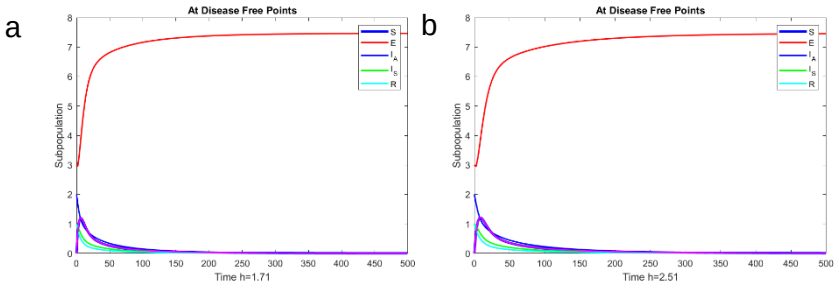

Figure 5. NSFD behavior with (a) disease free points at h =1.71 and (b) h=2.51

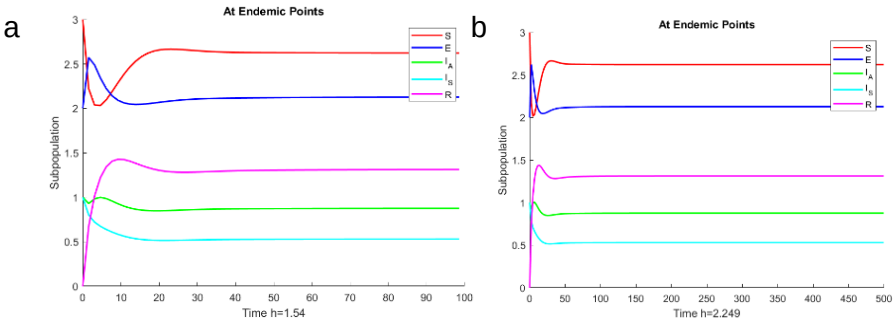

Figure 6. NSFD behavior with (a) endemic points at h =1.54 and (b) h=2.249

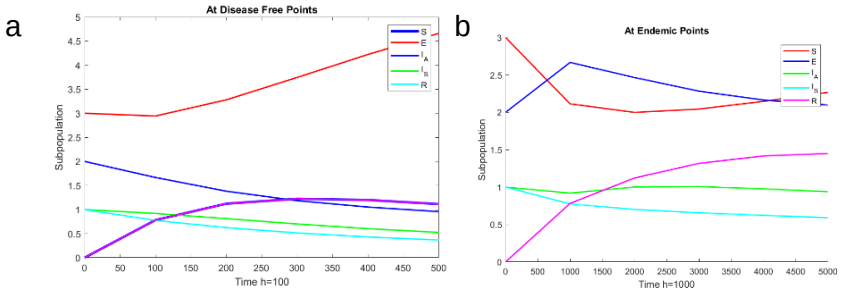

Figure 7. NSFD behavior with (a) disease-free points at h =100 and (b) at h=1000

The Euler and RK4 techniques can provide negative solutions or diverge at tiny step sizes and different behaviour at large step sizes, as shown in **Figures 3, 4, 5, 6 and 7**. By way of comparison, the Non-Standard Finite Difference (NSFD) approach produces convergent solutions even with the exact and tiny step sizes while maintaining the system's qualitative characteristics, as shown in **Figures 3, 4, 5, 6 and**

7. Because of this, NSFD ensures numerical stability and is more suited for managing intricate epidemiological models.

Hence, the graphic analysis proved that NSFD is more reliable than Euler and Rk4 [26, 27, 28]. NSFD methods offer greater accuracy and stability compared to Euler and RK4 by better preserving the qualitative dynamics of disease models. Euler, which may introduce errors over time, and RK4, which can be less efficient for stiff systems, NSFD ensures more reliable long-term predictions [28, 29, 30]. This makes NSFD particularly useful for guiding public health strategies. The NSFD method helps preserve realistic properties and ensure that solutions remain biologically feasible across longer periods, which strengthens the model's practical applicability for public health planning.

Conclusion

The mathematical modelling of epidemiological disorders serves as a crucial tool for analysing disease dynamics and devising strategies to mitigate the impact of infectious diseases globally. This study highlights the significant role of model equilibrium points and the basic reproduction number (R_0) in understanding disease spread. An increase in R_0 correlates with a greater likelihood of disease propagation within a community, whereas a reduction in R_0 signifies disease containment. Sensitivity analysis has proven instrumental in identifying the influence of model parameters on R_0. Employing the nonstandard finite difference (NSFD) method, this study provides an accurate and robust numerical solution for a chlamydia epidemic model. The NSFD method preserves the fundamental properties of the epidemic model, demonstrating superior efficiency and reliability compared to classical methods such as forward Euler and Runge-Kutta 4th order (RK-4). Simulations reveal that traditional methods fail to yield precise results at petite and large step sizes. In contrast, the NSFD method ensures convergence and dynamic consistency across varying step sizes. The model identifies two equilibrium points: a disease-free equilibrium (DFE) and an endemic equilibrium (EE). Stability analysis reveals that the DFE is locally asymptotically stable when $R_0 < 1$, indicating successful disease eradication, while it becomes unstable when $R_0 > 1$, signifying disease persistence. The NSFD approach, validated through comparative analysis and graphical evidence, demonstrates its capacity to uphold the structural properties of the chlamydia mathematical model, making it a reliable and effective numerical technique. also, by finding consistency analysis, we ensure that NSFD is consistent with the system. This study contributes to a deeper understanding of chlamydia dynamics, with significant implications for public health planning. Future extensions of this model could incorporate fractional-order derivatives and stochastic components to account for variability and uncertainty in disease transmission, thereby enhancing its relevance and application to real-world public health challenges. Additionally, incorporating real-world data into the model could further enhance its predictive accuracy and applicability to epidemiological studies. Furthermore, extending the NSFD method to multi-strain or co-infection scenarios could provide deeper insights into complex disease interactions and control strategies.

Conflicts of Interest

The authors confirm that they have no conflicts of interest related to the publication of this paper.

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