

Numerical Modelling of SEIR on Two-Dose Vaccination Against the Rubella Virus

Shah Zeb^a, Siti Ainor Mohd Yatim^{a*}, Awais Ahmad^b, Ayesha Kamran^b,
Muhammad Rafiq^c

^aSchool of Distance Education Universiti Sains Malaysia Minden USM 11800 Penang Malaysia; ^bDepartment of Mathematics University of management and technology CII, Johar Town Lahore 54770 Punjab Pakistan; ^cDepartment of Mathematics, Namal University 30km Talagang Road, Mianwali, 42250, Pakistan

Abstract In this article, we determine non-linear terms under the modulation of dynamic transmission in childhood diseases analyzed to explore the effect of Rubella virus, along with double-dose vaccination strategy which was suggested by WHO. Firstly, basic properties of model were calculated such as positiveness, boundedness, disease free and endemic points. Model stability was proved at the disease-free and endemic equilibrium points. The Reproductive number was calculated using the next generation matrix method. Furthermore, sensitivity analysis is used to ascertain how parameter changes impact the system's dynamic behavior. We used Euler, RK-4 and NSFD method. The purpose of the numerical simulations is to demonstrate the importance of the theoretical findings using numerical methods and the viability of the numerical schemes. Convergence and consistency analysis of NSFD scheme were proven. Additionally, we proved that NSFD is more reliable than Euler and RK4 through graphical interpretation. Incorporating this method enhanced the model's accuracy, stability, and predictions for rubella dynamics.

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

Introduction

Rubella is an infectious disease caused by a virus. Additionally, it is referred to as "German measles," and is caused by a virus distinct from measles. In 2004, the United States declared the elimination of the rubella virus. The lack of ongoing disease transmission in a particular geographic area for a minimum of 12 months is known as rubella eradication [1]. German measles has been a serious illness and a global public health issue ever since the epidemic of congenital cataracts brought on by maternal infection in 1941. The most dreadful outcome of a rubella infection in an early-phase fetus is congenital rubella syndrome [2, 3]. Congenital rubella syndrome (CRS), a condition in babies caused by maternal rubella virus infection during pregnancy, and it is the most dangerous consequence of rubella virus infection. Before rubella was originally identified as a distinct illness in German medical literature in 1814, it was thought to be a variation of measles or scarlet fever. Since then, several initiatives have been made to stop the illness from spreading [4, 5]. The World Health Organization (WHO) has strongly advised the use of Measles, Mumps, Rubella (MMR) vaccinations in countries pursuing widespread immunization programs to eradicate the mumps, measles, and rubella viruses [6]. This recommendation resulted from in-depth research on the outcomes of several clinical trials. According to the research, using multivalent immunizations, such as the MMR vaccine, was advised to stop the spread of the rubella virus and eradicate both CRS and rubella. A comprehensive analysis was carried out by Vynnický *et al.* [7] on vaccination coverage and seroprevalence to calculate the worldwide CRS burden.

The research indicated that, outside of the Americas, Europe, and the Eastern Mediterranean, the projected global prevalence of CRS remains high even after vaccination. Following the Global Vaccine Action Plan [8] which did not explicitly mention the aim of eradicating measles or rubella worldwide, the World Health Organization released the Global Measles and Rubella Strategic Plan 2012-2020 [9]. This strategy calls for nations to attain and preserve a high-level of vaccination coverage (at least 80%) with two dose MCV-RCV or combination vaccines (MR or MMR) as one of the measures to eliminate measles,

*For correspondence:

ainor@usm.my

Received: 14 May 2024

Accepted: 10 Jan. 2025

©Copyright Zeb. This article is distributed under the terms of the [Creative Commons Attribution License](#), which permits unrestricted use and redistribution provided that the original author and source are credited.

rubella, and CRS. It is advised to employ a 2-dose vaccination approach since the majority of people's levels of rubella antibodies following a single dose of the vaccine may eventually decline. It is evident that after 12 to 15 years following the second dose, between 91 and 100 % of those who had two doses developed detectable antibodies [10]. It is standard practice to propose a 2-dose MMR immunization regimen for children who are 12 months or older. Rubella should be administered as a first dose between the ages of 12 and 15 months. If the first dose fails to elicit an immunological response, a second dose should be administered.

Second dose is usually administered between the age of 4 and 6 before a child starts school [11]. Numerous authors have carried out extensive research on the spread of the rubella virus, including different immunization approaches. There is research on the attempts to eradicate rubella that might be discussed by a few WHO regions. For example, in 2013 Gao *et al.* [12] reported that in Australia, vaccination rates were successfully increased by 99% in 2010 compared to the prevention era (1960–70). Similarly, Lambert *et al.* [3] said that although while rubella vaccinations have been successfully administered throughout the Americas since 2009, incomplete immunization programs have allowed the virus to continue spreading, as seen in recent outbreaks in Japan and other countries.

In 2016, Wu *et al.* [13] presented research on the age structured rubella transmission model in East Java, Indonesia, using seven different vaccination approaches. Their findings suggested that, if vaccination coverage is kept at least the same as it was for the prior two dose vaccination, substituting the current two dose measles vaccination with MMR vaccines would be more successful in achieving a 99% yearly decrease in rubella transmission after 20 years. By the LeBaron research [31], which assessed the immunogenicity of MMR2 short and long-term, reinforced the significance of the second dose of the rubella immunization (MMR2). From blood specimens obtained over a 12-year period, they assessed the two groups' rubella antibody levels in children who obtained MMR2 at ages 4–6 and 10–12. According to their findings, rubella antibodies responded well to MMR2, but after 12 years, the antibodies' levels tended to decline. The model [1] reflects the decline in immunity from the first dose, noting that rubella antibodies may decrease around 12 years after MMR1, emphasizing the importance of the second dose (MMR2). The second dose's efficacy is represented by a parameter θ_2 , which extends immunity and lowers the number of susceptible individuals in the older age group. Most researchers use Kermack-McKendrick's standard model when discussing studies on the model of viral transmission [14, 18, 19, 20] and changes, contingent upon the inclusion of new variables in the systematic model. Zhou *et al.* [15] discussed a SEIR rubella transmission framework with age factor. They inquired about the global stability of endemic equilibria of SEIR epidemic models with the rubella virus in susceptible, exposed and infected populations. Furthermore, they introduced a vaccination framework with four age groups to investigate different vaccination strategies for rubella virus. Sun and Hsieh [16] studied a SEIR model in which they considered to varying population size and vaccination strategy. The system's fundamental reproduction ratios were thoroughly analyzed, and the results were utilized to assess the stability of the endemic and disease-free equilibria.

It is more critical to investigate the impact of vaccine induced immunity reduction on the transmission dynamics of childhood illnesses. Most semi-analytic and conventional numerical methods fail to explain the true behavior of an infectious disease in a community. These methods have some drawbacks and are incompatible with the continuous model's biological nature [21, 22]. Sometimes, it is very difficult to find the exact and reliable solution of many systems, so researchers use numerical techniques for finding the result. Because this model is following the WHO criteria, so we have done it using numerical techniques for reliable results. We applied the Euler, RK4, and NSFD methods to a model [1] in order to assess and compare their effectiveness in simulating the model's dynamics. The assumptions made by Hethcote [17] in 1976 are the foundation for this model. By testing multiple numerical techniques on an established model, we were able to evaluate their reliability and determine which method most accurately preserves the model's biological features. According to our study, this model is being studied first time using NSFD. This comparison provides valuable insights into the model's practical applicability and can inform future research on selecting suitable numerical methods for similar systems [23, 24, 25, 30, 32]. A reliable numerical analysis that preserves all the fundamental attributes is required to examine the precise behaviors of such a model. NSFD show convergence at very small step size, and this method is more reliable for other models with vaccination such as Rubella, Covid [23, 24, 25, 26]. Using methods like Euler, RK4, and especially NSFD with this model improves its accuracy and stability, making long-term predictions about rubella more reliable. These methods help keep population numbers realistic, which is important for planning effective vaccination strategies.

This paper is divided into five sections. Section 1 is about literature review and some background; Section 2 is presenting the model formulation. In section 3, the equilibrium states are presented. Basic reproductive number, sensitivity and stability analysis of the model are presented in section 4, while in section 5 we discussed numerical simulation and discussion of the results. Section 6 presents the

stability analysis of NSFD and in section 7 we discussed the conclusion of the paper.

Model Formulation

The model primarily targets females because rubella increases the risk of early pregnancies, which in turn causes CRS in the newborns where the mother was infected during pregnancy. This sex differential is attributed to the danger of rubella in females of childbearing age rather than a differential susceptibility of females and males to the virus [1]. The Vaccination strategy is not gender-specific and there is no need to target males; however, targeting the females is an important requirement in eradicating CRS due to rubella which falls under public health priority for rubella elimination. The mathematical model [1] continued to focus on women. Still due to the deployment of two dose vaccine method, the population was split between girls aged 4 and under, girls aged 12 to 15 months and the larger population.

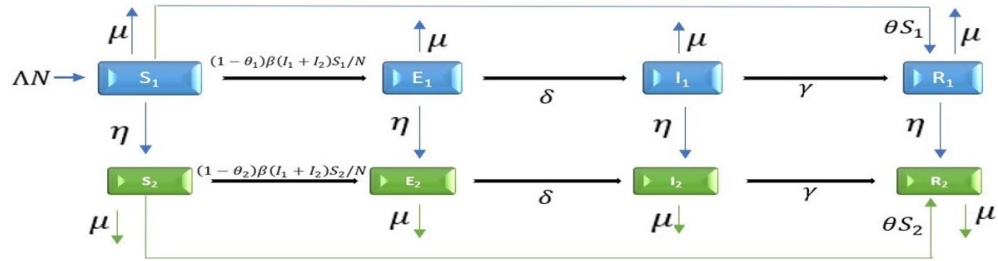


Figure 1. SEIR two dose vaccination flow chart

Protein Preparation

The entire human population in figure 1 is represented by the letter N and is subdivided into eight epidemiological sub-populations: susceptible populations S1 and S2 infected populations I1 and I2, Exposed populations E1 and E2 and recovered populations R1 and R2 representing the recovered individual population.

$$S_1 + S_2 + I_1 + I_2 + E_1 + E_2 + R_1 + R_2 = N$$

Variable of compartmental model S: Susceptible, I: Infected, E: Exposed, R: Recovered.

$$\frac{dS_1}{dt} = \Delta N - \frac{(1-\theta_1)\beta S_1(I_1+I_2)}{N} - (\mu + \eta + \theta_1)S_1, \tag{1a}$$

$$\frac{dE_1}{dt} = \frac{(1-\theta_1)\beta S_1(I_1+I_2)}{N} - (\mu + \eta + \delta)E_1, \tag{1b}$$

$$\frac{dI_1}{dt} = \delta E_1 - (\mu + \eta + \gamma)I_1, \tag{1c}$$

$$\frac{dR_1}{dt} = \gamma I_1 - (\mu + \eta)R_1 + \theta_1 S_1. \tag{1d}$$

$$\frac{dS_2}{dt} = \eta S_1 - \frac{(1-\theta_2)\beta S_2(I_1+I_2)}{N} - (\mu + \theta_2)S_2, \tag{1e}$$

$$\frac{dE_2}{dt} = \eta E_1 + \frac{(1-\theta_2)\beta S_2(I_1+I_2)}{N} - (\mu + \delta)E_2, \tag{1f}$$

$$\frac{dI_2}{dt} = \eta I_1 + \delta E_2 - (\mu + \gamma)I_2, \tag{1g}$$

$$\frac{dR_2}{dt} = \eta R_1 + \gamma I_2 - \mu R_2 + \theta_2 S_2. \tag{1h}$$

We assumed all parameters of the system are non-negative.

Using the following re-scaling for simplification

$$\begin{aligned} \hat{S}_1 &= \frac{S_1}{N}, & \hat{E}_1 &= \frac{E_1}{N}, & \hat{I}_1 &= \frac{I_1}{N}, & \hat{R}_1 &= \frac{R_1}{N} \\ \hat{S}_2 &= \frac{S_2}{N}, & \hat{E}_2 &= \frac{E_2}{N}, & \hat{I}_2 &= \frac{I_2}{N}, & \hat{R}_2 &= \frac{R_2}{N} \end{aligned}$$

After using these values and dropping the caps, we got new system in a simple structure,

$$\left. \begin{aligned}
 \frac{dS_1}{dt} &= \Lambda - (1 - \theta_1)\beta S_1(I_1 + I_2) - (\mu + \eta + \theta_1)S_1, \\
 \frac{dE_1}{dt} &= (1 - \theta_1)\beta S_1(I_1 + I_2) - (\mu + \eta + \delta)E_1, \\
 \frac{dI_1}{dt} &= \delta E_1 - (\mu + \eta + \gamma)I_1, \\
 \frac{dR_1}{dt} &= \gamma I_1 - (\mu + \eta)R_1 + \theta_1 S_1, \\
 \frac{dS_2}{dt} &= \eta S_1 - (1 - \theta_2)\beta S_2(I_1 + I_2) - (\mu + \theta_2)S_2, \\
 \frac{dE_2}{dt} &= \eta E_1 + (1 - \theta_2)\beta S_2(I_1 + I_2) - (\mu + \delta)E_2, \\
 \frac{dI_2}{dt} &= \eta I_1 + \delta E_2 - (\mu + \gamma)I_2, \\
 \frac{dR_2}{dt} &= \eta R_1 + \gamma I_2 - \mu R_2 + \theta_2 S_2.
 \end{aligned} \right\} \tag{2}$$

Theorem 1: For given time (t) the system holds the positivity of the solution at the system of Equations (2).

Proof:

$$\begin{aligned}
 \left. \frac{dS_1}{dt} \right|_{S_1=0} &= \Lambda \geq 0, & \left. \frac{dE_1}{dt} \right|_{E_1=0} &= (1 - \theta_1)\beta S_1(I_1 + I_2) \geq 0, & \left. \frac{dI_1}{dt} \right|_{I_1=0} &= \delta E_1 \geq 0, \\
 \left. \frac{dR_1}{dt} \right|_{R_1=0} &= \gamma I_1 + \theta_1 S_1 \geq 0, \\
 \left. \frac{dS_2}{dt} \right|_{S_2=0} &= \eta S_1 \geq 0, & \left. \frac{dE_2}{dt} \right|_{E_2=0} &= \eta E_1 + (1 - \theta_2)\beta S_2(I_1 + I_2) \geq 0, & \left. \frac{dI_2}{dt} \right|_{I_2=0} &= \eta I_1 + \delta E_2 \geq 0, \\
 \left. \frac{dR_2}{dt} \right|_{R_2=0} &= \eta R_1 + \gamma I_2 + \theta_2 S_2 \geq 0.
 \end{aligned}$$

Which clearly indicate that positivity exists in the system.

Theorem 3: System's solution is bounded in feasible region.

Proof:

The total population defined as

$$S_1 + S_2 + I_1 + I_2 + E_1 + E_2 + R_1 + R_2 = N.$$

Differentiate with respect to time on both sides

$$\frac{dN}{dt} = \frac{dS_1}{dt} + \frac{dS_2}{dt} + \frac{dI_1}{dt} + \frac{dI_2}{dt} + \frac{dE_1}{dt} + \frac{dE_2}{dt} + \frac{dR_1}{dt} + \frac{dR_2}{dt},$$

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

$$\frac{dN}{dt} = \Lambda N - \mu(S_1 + S_2 + I_1 + I_2 + E_1 + E_2 + R_1 + R_2).$$

According to given condition

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

Solve the differential equation for N by variable separation and integrate,

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

integrate both sides and we got

$$\ln(n) = (\Lambda - \mu)t + C, \quad \text{where } C \text{ is the constant of integration exponentiating both sides}$$

letting $e^C = N_0$ the initial population at $t = 0$, we find

$$N = N_0 e^{(\Lambda - \mu)t}.$$

If $\Lambda = \mu$, then $\frac{dN}{dt} = 0$, which implies $N = N_0$ meaning the population N remains constant over time. Thus, the total population conserved.

So, the feasible region Ω is,

$$\Omega = \{(S_1, S_2, I_1, I_2, E_1, E_2, R_1, R_2) \in \mathbb{R}_+^8 \mid S_1 + S_2 + I_1 + I_2 + E_1 + E_2 + R_1 + R_2 = N\}. \quad (3)$$

Equilibrium Points

Disease free and endemic equilibrium points discussed in this section.

From above equation we get

$$S_1^0 = \frac{\Lambda - (1 - \theta_1)\beta S_1(I_1 + I_2)}{(\mu + \eta + \theta_1)}, \quad E_1^0 = \frac{(1 - \theta_1)\beta S_1(I_1 + I_2)}{(\mu + \eta + \delta)}, \quad I_1^0 = 0, \quad S_2^0 = \frac{\eta S_1}{(1 - \theta_2)\beta(I_1 + I_2)(\mu + \theta_2)},$$

$$E_2^0 = \frac{\eta E_1 + (1 - \theta_2)\beta S_2(I_1 + I_2)}{(\mu + \delta)}, \quad I_2^0 = 0.$$

So, the disease-free equilibrium points are

$$\varepsilon_0 = (S_1^0, E_1^0, I_1^0, S_2^0, E_2^0, I_2^0) = \left(\frac{\Lambda}{(\mu + \eta + \theta_1)}, \frac{(1 - \theta_1)\beta S_1(I_1 + I_2)}{(\mu + \eta + \delta)}, 0, \frac{\eta \Lambda}{(\eta + \mu + \theta_1)(\mu + \theta_2)}, \frac{\eta E_1 + (1 - \theta_2)\beta S_2(I_1 + I_2)}{(\mu + \delta)}, 0 \right). \quad (4)$$

And from system equation's after simplification endemic equilibrium points obtained which are

$$S_1^* = \frac{\Lambda}{(1 - \theta_1)\beta(I_1 + I_2) + (\mu + \eta + \theta_1)}, \quad E_1^* = \frac{(1 - \theta_1)\beta S_1(I_1 + I_2)}{(\mu + \eta + \delta)}, \quad I_1^* = \frac{\delta E_1}{(\mu + \eta + \gamma)}, \quad S_2^* = \frac{\eta S_1}{(1 - \theta_2)\beta(I_1 + I_2) + (\mu + \theta_2)},$$

$$E_2^* = \frac{\eta E_1 + (1 - \theta_2)\beta S_2(I_1 + I_2)}{(\mu + \delta)}, \quad I_2^* = \frac{\eta I_1 + \delta E_2}{(\mu + \gamma)}.$$

Hence, endemic points are

$$\varepsilon_1 = (S_1^*, E_1^*, I_1^*, S_2^*, E_2^*, I_2^*) = \left(\frac{\Lambda}{(1 - \theta_1)\beta(I_1 + I_2) + (\mu + \eta + \theta_1)}, \frac{(1 - \theta_1)\beta S_1(I_1 + I_2)}{(\mu + \eta + \delta)}, \frac{\delta E_1}{(\mu + \eta + \gamma)}, \frac{\eta S_1}{(1 - \theta_2)\beta(I_1 + I_2) + (\mu + \theta_2)}, \frac{\eta E_1 + (1 - \theta_2)\beta S_2(I_1 + I_2)}{(\mu + \delta)}, \frac{\eta I_1 + \delta E_2}{(\mu + \gamma)} \right). \quad (5)$$

Basic Reproductive Number

In this section, we use the next generation matrix technique to calculate the generation number by calculating the transfer and transmission matrix.

$$E_1 = (1 - \theta_1)\beta S_1(I_1 + I_2) - (\mu + \eta + \delta)E_1,$$

$$I_1 = \delta E_1 - (\mu + \eta + \gamma)I_1,$$

$$E_2 = \eta E_1 + (1 - \theta_2)\beta S_2(I_1 + I_2) - (\mu + \delta)E_2,$$

$$I_2 = \eta I_1 + \delta E_2 - (\mu + \gamma)I_2.$$

As a threshold, this number is used to analyse whether a disease will spread or decrease from the population. It may be determined with the approach of the next generation matrix. In this disease system, the infected population is indicated by E and I , whereas Without infection compartments are marked by S and R . The new rate of infection formation and the rate of stage changeover are represented by the two matrices, T and V , respectively.

By next generation matrix method

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

$$T = \begin{bmatrix} (1 - \theta_1)\beta S_1 I_1 + (1 - \theta_1)\beta S_1 I_2 & 0 \\ (1 - \theta_2)\beta S_2 I_1 + (1 - \theta_2)\beta S_2 I_2 & 0 \end{bmatrix}$$

$$V = \begin{bmatrix} -(\mu + \eta + \delta)E_1 & & & \\ \delta E_1 - (\mu + \eta + \gamma)I_1 & & & \\ \eta E_1 - (\mu + \delta)E_2 & & & \\ \eta I_1 + \delta E_2 - (\mu + \gamma)I_2 & & & \end{bmatrix}$$

Thus, at the disease-free equilibrium (DFE), the transmissions matrix T and V becomes

$$\bar{T} = \begin{bmatrix} 0 & (1 - \theta_1)\beta S_1 & 0 & (1 - \theta_1)\beta S_1 \\ 0 & 0 & 0 & 0 \\ 0 & (1 - \theta_1)\beta S_1 & 0 & (1 - \theta_1)\beta S_1 \\ 0 & 0 & 0 & 0 \end{bmatrix}$$

$$\bar{V} = \begin{bmatrix} -(\mu + \eta + \delta) & 0 & 0 & 0 \\ \delta & -(\mu + \eta + \gamma) & 0 & 0 \\ \eta & 0 & -(\mu + \delta) & 0 \\ 0 & \eta & \delta & -(\mu + \gamma) \end{bmatrix}$$

\bar{T} represents the changes of new infections and \bar{V} transitions approaching equilibrium. Here we calculate the \bar{V} inverse, which indicate the time spent in each compartment and the total number of new infections produced during the duration of the infection. So, the fundamental reproductive number has the highest eigenvalue. Largest eigenvalue of

$$\bar{T} \bar{V}^{-1} = \begin{bmatrix} \frac{(1 - \theta_1)\beta\delta\Lambda}{(\mu + \eta + \gamma)(\mu + \eta + \delta)(\eta + \mu + \theta_1)} + \frac{(1 - \theta_1)\beta\delta\Lambda\eta(\gamma + \delta + \eta + 2\mu)}{(\mu + \eta + \delta)(\eta + \gamma + \mu)(\mu + \delta)(\gamma + \mu)(\eta + \mu + \theta_1)} & \frac{(1 - \theta_1)\beta\delta\Lambda}{(\mu + \delta)(\gamma + \mu)(\eta + \mu + \theta_1)} \\ \frac{(1 - \theta_2)\beta\delta\Lambda\eta}{(\mu + \eta + \gamma)(\mu + \eta + \delta)(\eta + \mu + \theta_1)(\mu + \theta_2)} + \frac{(1 - \theta_2)\beta\delta\Lambda\eta^2(\gamma + \delta + \eta + 2\mu)}{(\mu + \eta + \gamma)(\mu + \eta + \delta)(\mu + \delta)(\gamma + \mu)(\eta + \mu + \theta_1)(\mu + \theta_2)} & \frac{(1 - \theta_2)\beta\delta\Lambda\eta}{(\mu + \delta)(\gamma + \mu)(\eta + \mu + \theta_1)(\mu + \theta_2)} \end{bmatrix}$$

It's worth noting that $\bar{T} \bar{V}^{-1}$ spectral radius is referred to as the reproduction number and is Denoted as R_0 . It is critical for disease control since it switches the illness from one equilibrium point to the next. So,

$$R_0 = \frac{(1 - \theta_1)\beta\delta\Lambda}{(\mu + \eta + \gamma)(\mu + \eta + \delta)(\eta + \mu + \theta_1)} + \frac{(1 - \theta_1)\beta\delta\Lambda\eta(\gamma + \delta + \eta + 2\mu)}{(\mu + \eta + \delta)(\eta + \gamma + \mu)(\mu + \delta)(\gamma + \mu)(\eta + \mu + \theta_1)} \tag{6}$$

Sensitivity Analysis of Parameters

By calculated 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 represented in **figure 2**.

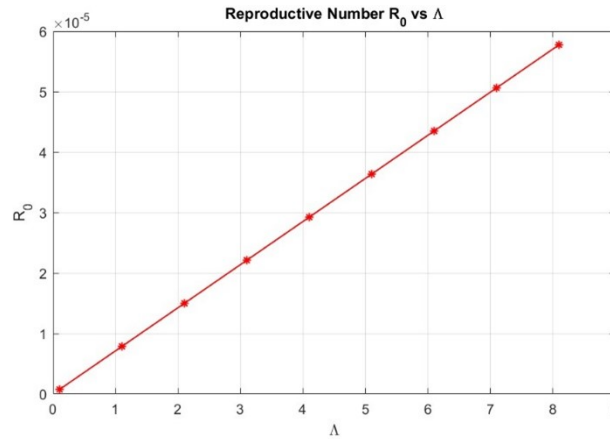


Figure 2. Behavior of R₀ due to Λ

Local Stability of Disease-Free and Endemic Equilibrium Points

Theorem 2: The disease-free equilibrium of system point is locally asymptotically stable whenever $R_0 < 1$ and unstable otherwise.

Proof: System of differential equation took from (1) as written below

$$\frac{dS_1}{dt} = \Lambda - (1 - \theta_1)\beta S_1(I_1 + I_2) - (\mu + \eta + \theta_1)S_1,$$

$$\frac{dE_1}{dt} = (1 - \theta_1)\beta S_1(I_1 + I_2) - (\mu + \eta + \delta)E_1,$$

$$\frac{dI_1}{dt} = \delta E_1 - (\mu + \eta + \gamma)I_1,$$

$$\frac{dS_2}{dt} = \eta S_1 - (1 - \theta_2)\beta S_2(I_1 + I_2) - (\mu + \theta_2)S_2,$$

$$\frac{dE_2}{dt} = \eta E_1 + (1 - \theta_2)\beta S_2(I_1 + I_2) - (\mu + \delta)E_2,$$

$$\frac{dI_2}{dt} = \eta I_1 + \delta E_2 - (\mu + \gamma)I_2,$$

to analyze the local stability of disease-free equilibrium point, Jacobian matrix of the system of equation disease free equation point

$$F = \Lambda - (1 - \theta_1)\beta S_1(I_1 + I_2) - (\mu + \eta + \theta_1)S_1 \tag{7}$$

$$G = (1 - \theta_1)\beta S_1(I_1 + I_2) - (\mu + \eta + \delta)E_1 \tag{8}$$

$$H = \delta E_1 - (\mu + \eta + \gamma)I_1 \tag{9}$$

$$L = \eta S_1 - (1 - \theta_2)\beta S_2(I_1 + I_2) - (\mu + \theta_2)S_2 \tag{10}$$

$$M = \eta E_1 + (1 - \theta_2)\beta S_2(I_1 + I_2) - (\mu + \delta)E_2 \tag{11}$$

$$N = \eta I_1 + \delta E_2 - (\mu + \gamma)I_2 \tag{12}$$

Differentiating F, G, H, L, M and N with respect to compartmental model perimeters S, E, I and R. Now J will be

$$J = \begin{bmatrix} F_{S_1} & F_{E_1} & F_{I_1} & F_{S_2} & F_{E_2} & F_{I_2} \\ G_{S_1} & G_{E_1} & G_{I_1} & G_{S_2} & G_{E_2} & G_{I_2} \\ H_{S_1} & H_{E_1} & H_{I_1} & H_{S_2} & H_{E_2} & H_{I_2} \\ L_{S_1} & L_{E_1} & L_{I_1} & L_{S_2} & L_{E_2} & L_{I_2} \\ M_{S_1} & M_{E_1} & M_{I_1} & M_{S_2} & M_{E_2} & M_{I_2} \\ N_{S_1} & N_{E_1} & N_{I_1} & N_{S_2} & N_{E_2} & N_{I_2} \end{bmatrix},$$

$$J = \begin{bmatrix} -(\mu + \eta + \theta_1) & 0 & \frac{-(1-\theta_1)\beta\Lambda}{(\mu+\eta+\theta_1)} & 0 & 0 & \frac{-(1-\theta_1)\beta\Lambda}{(\mu+\eta+\theta_1)} \\ 0 & -(\mu + \eta + \delta) & \frac{(1-\theta_1)\beta\Lambda}{(\mu+\eta+\theta_1)} & 0 & 0 & \frac{(1-\theta_1)\beta\Lambda}{(\mu+\eta+\theta_1)} \\ 0 & \delta & -(\mu + \eta + \gamma) & 0 & 0 & 0 \\ \eta & 0 & \frac{-(1-\theta_2)\beta\Lambda\eta}{(\mu+\eta+\theta_1)(\mu+\theta_2)} & -(\mu + \theta_2) & 0 & \frac{-(1-\theta_2)\beta\Lambda\eta}{(\mu+\eta+\theta_1)(\mu+\theta_2)} \\ 0 & \eta & \frac{-(1-\theta_2)\beta\Lambda\eta}{(\mu+\eta+\theta_1)(\mu+\theta_2)} & 0 & -(\mu + \delta) & \frac{(1-\theta_2)\beta\Lambda\eta}{(\mu+\eta+\theta_1)(\mu+\theta_2)} \\ 0 & 0 & \eta & 0 & \delta & -(\mu + \gamma) \end{bmatrix}.$$

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

$$|J - \lambda I| = \begin{vmatrix} -(\mu + \eta + \theta_1) - \lambda & 0 & \frac{-(1-\theta_1)\beta\Lambda}{(\mu+\eta+\theta_1)} & 0 & 0 & \frac{-(1-\theta_1)\beta\Lambda}{(\mu+\eta+\theta_1)} \\ 0 & -(\mu + \eta + \delta) - \lambda & \frac{(1-\theta_1)\beta\Lambda}{(\mu+\eta+\theta_1)} & 0 & 0 & \frac{(1-\theta_1)\beta\Lambda}{(\mu+\eta+\theta_1)} \\ 0 & \delta & -(\mu + \eta + \gamma) - \lambda & 0 & 0 & 0 \\ \eta & 0 & \frac{-(1-\theta_2)\beta\Lambda\eta}{(\mu+\eta+\theta_1)(\mu+\theta_2)} & -(\mu + \theta_2) - \lambda & 0 & \frac{-(1-\theta_2)\beta\Lambda\eta}{(\mu+\eta+\theta_1)(\mu+\theta_2)} \\ 0 & \eta & \frac{-(1-\theta_2)\beta\Lambda\eta}{(\mu+\eta+\theta_1)(\mu+\theta_2)} & 0 & -(\mu + \delta) - \lambda & \frac{(1-\theta_2)\beta\Lambda\eta}{(\mu+\eta+\theta_1)(\mu+\theta_2)} \\ 0 & 0 & \eta & 0 & \delta & -(\mu + \gamma) - \lambda \end{vmatrix}.$$

which give us the following eigen values by solving this matrix

$$\begin{aligned} \lambda_1 &= -\delta - \eta - \theta_1, \\ \lambda_2 &= -\eta - \gamma - \mu, \\ \lambda_3 &= -\eta - \mu - \theta_1, \\ \lambda_4 &= -\mu - \theta_2, \end{aligned}$$

the rest of two were complex so we found by python and checked they are also negative but for more clarification we used MATLAB and found the numeric values as given below

$$\lambda = -0.9503, -0.8541, -0.0284, -0.0244, -0.3041, -0.3005.$$

From which we conclude that $R_0 < 1$.

Theorem 3: The endemic equilibrium points is locally asymptotically stable if $R_0 > 1$.

Proof:

System obtained by linearizing with endemic equilibrium points which gives the following matrix

$$J = \begin{bmatrix} -\beta(1 - \theta_1)(I_1^* - I_2^*) & 0 & -\beta S_1^*(1 - \theta_1) & 0 & 0 & -\beta S_1^*(1 - \theta_1) \\ \beta I_1^*(1 - \theta_1) + \beta I_2^*(1 - \theta_1) & -(\mu + \eta + \delta) & \beta S_1^*(1 - \theta_1) & 0 & 0 & \beta S_1^*(1 - \theta_1) \\ 0 & \delta & -(\mu + \eta + \gamma) & 0 & 0 & 0 \\ 0 & 0 & -\beta S_2^*(1 - \theta_2) & -\beta I_1^*(1 - \theta_2) - \beta I_2^*(1 - \theta_2) & -(\mu + \delta) & -\beta S_2^*(1 - \theta_2) \\ 0 & 0 & \beta S_2^*(1 - \theta_2) & \beta I_1^*(1 - \theta_2) & -(\mu + \delta) & \beta S_2^*(1 - \theta_2) \\ 0 & 0 & \eta & 0 & \delta & -(\mu + \gamma) \end{bmatrix}.$$

So, we calculated $\det(J - \lambda I)$,

$$\det(J - \lambda I) =$$

$$\begin{vmatrix} -\beta(1-\theta_1)(I_1^* - I_2^*) - \lambda & 0 & -\beta S_1^*(1-\theta_1) & 0 & 0 & -\beta S_1^*(1-\theta_1) \\ \beta I_1^*(1-\theta_1) + \beta I_2^*(1-\theta_1) & -(\mu + \eta + \delta) - \lambda & \beta S_1^*(1-\theta_1) & 0 & 0 & \beta S_1^*(1-\theta_1) \\ 0 & \delta & -(\mu + \eta + \gamma) - \lambda & 0 & 0 & 0 \\ 0 & 0 & -\beta S_2^*(1-\theta_2) & -\beta I_1^*(1-\theta_2) - \beta I_2^*(1-\theta_2) - \lambda & -(\mu + \delta) & -\beta S_2^*(1-\theta_2) \\ 0 & 0 & \beta S_2^*(1-\theta_2) & \beta I_1^*(1-\theta_2) & -(\mu + \delta) - \lambda & \beta S_2^*(1-\theta_2) \\ 0 & 0 & \eta & 0 & \delta & -(\mu + \gamma) - \lambda \end{vmatrix}$$

Now,

$$|J - \lambda I| = 0.$$

One of the roots is $-(\mu + \eta + \gamma)$ rest are so complex so by Mathematica calculations and by MATLAB numeric values we checked all eigen values are with negative real parts so, endemic points are locally asymptotically stable if $R_0 > 1$.

Numerical Analysis of SEIR Model

In this section for numerical investigation, we used EULER, RK4 and NSFD method, NSFD method is positive preserving numerical scheme. Further justification proved in this section by graphical proving. The recovered sub-populations R_1 and R_2 were not considered in the analysis, because they do not support other subpopulations. Therefore, the research focuses on the following simple systems. Parametric values used in numerical analysis written in **Table 1** obtained from [1].

Table 1. Parametric values

Parameters	Properties	DFE	EE	UNIT	Reference
Λ	Influx of susceptible	1000	1000	1000 Per week	[1]
β	Transmission rate	0.000001	0.0007		DFE(Fitted), EE [1]
δ	Aging ratio	0.3	0.3		[1]
γ	Incubation period	0.0243	0.0243	Per week	[1]
η	Recovery ratio	0.00385	0.00385	Per week	[1]
μ	Natural death rate	0.3	0.3	Per week	[1]
θ_1	MMR1 impact	0.85	0.85		[1]
θ_2	MMR2 impact	0.95	0.95		[1]

From SEIR model, now we will use the numerical modelling to solve the problem. First, we'll use the Forward Euler scheme, followed by the Fourth Order Runge-Kutta scheme, and finally the proposed approach.

Euler Scheme

By the model's equations Euler scheme represented as

$$S_1^{n+1} = S_1^n + h(\Lambda - (1 - \theta_1)\beta S_1^n(I_1^n + I_2^n) - (\mu + \eta + \theta_1)S_1^n), \tag{13.1}$$

$$E_1^{n+1} = E_1^n + h((1 - \theta_1)\beta S_1^n(I_1^n + I_2^n) - (\mu + \eta + \delta)E_1^n), \tag{13.2}$$

$$I_1^{n+1} = I_1^n + h(\delta E_1^n - (\mu + \eta + \gamma)I_1^n), \tag{13.3}$$

$$S_2^{n+1} = S_2^n + h(\eta S_1 - (1 - \theta_2)\beta S_2^n(I_1^n + I_2^n) - (\mu + \theta_2)S_2^n), \tag{13.4}$$

$$E_2^{n+1} = E_2^n + h(\eta E_1^n + (1 - \theta_2)\beta S_2^n(I_1^n + I_2^n) - (\mu + \delta)E_2^n), \tag{13.5}$$

$$I_2^{n+1} = I_2^n + h(\eta I_1^n + \delta E_2^n - (\mu + \gamma)I_2^n). \tag{13.6}$$

Runge-Kutta Fourth Order Method

We use the SEIR system to develop an explicit RK4-method.

$$K_1 = h(\Lambda - (1 - \theta_1)\beta S_1^n(I_1^n + I_2^n) - (\mu + \eta + \theta_1)S_1^n), \tag{13.7}$$

$$m_1 = h((1 - \theta_1)\beta S_1^n(I_1^n + I_2^n) - (\mu + \eta + \delta)E_1^n), \tag{13.8}$$

$$n_1 = h(\delta E_1^n - (\mu + \eta + \gamma)I_1^n), \tag{13.9}$$

$$p_1 = h(\eta S_1^n - (1 - \theta_2)\beta S_2^n(I_1^n + I_2^n) - (\mu + \theta_2)S_2^n), \tag{13.10}$$

$$t_1 = h(\eta E_1^n + (1 - \theta_2)\beta S_2^n(I_1^n + I_2^n) - (\mu + \delta)E_2^n), \tag{13.11}$$

$$u_1 = h(\eta I_1^n + \delta E_2^n - (\mu + \gamma)I_2^n). \tag{13.12}$$

$$k_2 = h(\Lambda - (1 - \theta_1)\beta(S_1^n + \frac{k_1}{2})(I_1^n + \frac{n_1}{2}) + (I_2^n + \frac{u_1}{2}) - (\mu + \eta + \theta_1)(S_1^n + \frac{k_1}{2})), \tag{13.13}$$

$$m_2 = h((1 - \theta_1)\beta(S_1^n + \frac{k_1}{2})(I_1^n + \frac{n_1}{2}) + (I_2^n + \frac{u_1}{2}) - (\mu + \eta + \delta)(E_1^n + \frac{m_1}{2})), \tag{13.14}$$

$$n_2 = h(\delta(E_1^n + \frac{m_1}{2}) - (\mu + \eta + \gamma)(I_1^n + \frac{n_1}{2})), \tag{13.15}$$

$$p_2 = h(\eta(S_1^n + \frac{k_1}{2}) - (1 - \theta_2)\beta(S_2^n + \frac{p_1}{2})(I_1^n + \frac{n_1}{2}) + (I_2^n + \frac{u_1}{2})) - (\mu + \theta_2)(S_2^n + \frac{p_1}{2})), \tag{13.16}$$

$$t_2 = h(\eta(E_1^n + \frac{m_1}{2}) + (1 - \theta_2)\beta(S_2^n + \frac{p_1}{2})(I_1^n + \frac{n_1}{2}) + (I_2^n + \frac{u_1}{2}) - (\mu + \delta)(E_2^n + \frac{t_1}{2})), \tag{13.17}$$

$$u_2 = h(\eta(I_1^n + \frac{n_1}{2}) + \delta(E_2^n + \frac{t_1}{2}) - (\mu + \gamma)(I_2^n + \frac{u_1}{2})). \tag{13.18}$$

$$k_3 = h(\Lambda - (1 - \theta_1)\beta(S_1^n + \frac{k_2}{2})(I_1^n + \frac{n_2}{2}) + (I_2^n + \frac{u_2}{2}) - (\mu + \eta + \theta_1)(S_1^n + \frac{k_2}{2})), \tag{13.19}$$

$$m_3 = h((1 - \theta_1)\beta(S_1^n + \frac{k_2}{2})(I_1^n + \frac{n_2}{2}) + (I_2^n + \frac{u_2}{2}) - (\mu + \eta + \delta)(E_1^n + \frac{m_2}{2})), \tag{13.20}$$

$$n_3 = h(\delta(E_1^n + \frac{m_2}{2}) - (\mu + \eta + \gamma)(I_1^n + \frac{n_2}{2})), \tag{13.21}$$

$$p_3 = h(\eta(S_1^n + \frac{k_2}{2}) - (1 - \theta_2)\beta(S_2^n + \frac{p_2}{2})(I_1^n + \frac{n_2}{2}) + (I_2^n + \frac{u_2}{2}) - (\mu + \theta_2)(S_2^n + \frac{p_2}{2})), \tag{13.22}$$

$$t_3 = h(\eta(E_1^n + \frac{m_2}{2}) + (1 - \theta_2)\beta(S_2^n + \frac{p_2}{2})(I_1^n + \frac{n_2}{2}) + (I_2^n + \frac{u_2}{2}) - (\mu + \delta)(E_2^n + \frac{t_2}{2})), \tag{13.23}$$

$$u_3 = h(\eta(I_1^n + \frac{n_2}{2}) + \delta(E_2^n + \frac{t_2}{2}) - (\mu + \gamma)(I_2^n + \frac{u_2}{2})). \tag{13.24}$$

$$k_4 = h(\Lambda - (1 - \theta_1)\beta(S_1^n + k_3)(I_1^n + n_3) + (I_2^n + u_3) - (\mu + \eta + \theta_1)(S_1^n + k_3)), \tag{13.25}$$

$$m_4 = h((1 - \theta_1)\beta(S_1^n + k_3)(I_1^n + n_3) + (I_2^n + u_3) - (\mu + \eta + \delta)(E_1^n + m_3)), \tag{13.26}$$

$$n_4 = h(\delta(E_1^n + m_3) - (\mu + \eta + \gamma)(I_1^n + n_3)), \tag{13.27}$$

$$p_4 = h(\eta(S_1^n + k_3) - (1 - \theta_2)\beta(S_2^n + p_3)(I_1^n + n_3) + (I_2^n + u_3) - (\mu + \theta_2)(S_2^n + p_3)), \tag{13.28}$$

$$t_4 = h(\eta(E_1^n + m_3) + (1 - \theta_2)\beta(S_2^n + p_3)(I_1^n + n_3) + (I_2^n + u_3) - (\mu + \delta)(E_2^n + t_3)), \tag{13.29}$$

$$u_4 = h(\eta(I_1^n + n_3) + \delta(E_2^n + t_3) - (\mu + \gamma)(I_2^n + u_3)). \tag{13.30}$$

$$S_1^{n+1} = S_1^n + \frac{1}{6}(k_1 + 2k_2 + 2k_3 + k_4). \tag{14}$$

$$E_1^{n+1} = E_1^n + \frac{1}{6}(m_1 + 2m_2 + 2m_3 + m_4). \tag{15}$$

$$I_1^{n+1} = I_1^n + \frac{1}{6}(n_1 + 2n_2 + 2n_3 + n_4). \tag{16}$$

$$S_2^{n+1} = S_2^n + \frac{1}{6}(p_1 + 2p_2 + 2p_3 + p_4). \tag{17}$$

$$E_2^{n+1} = E_2^n + \frac{1}{6}(t_1 + 2t_2 + 2t_3 + t_4). \tag{18}$$

$$I_2^{n+1} = I_2^n + \frac{1}{6}(u_1 + 2u_2 + 2u_3 + u_4). \tag{19}$$

Non-Standard Finite Difference Scheme

In this part, we'll look at the stability of the NSFDF schemes of the SEIR model's disease-free equilibrium point (DFE).

$$S_1^{n+1} = \frac{S_1^n + h\Lambda}{1 + h(1 - \theta_1)\beta(I_1^n + I_2^n) + h(\mu + \eta + \theta_1)} \tag{20.1}$$

$$E_1^{n+1} = \frac{E_1^n + h(1 - \theta_1)\beta S_1^n (I_1^n + I_2^n)}{1 + h(\mu + \eta + \delta)} \tag{20.2}$$

$$I_1^{n+1} = \frac{I_1^n + h\delta E_1^n}{1 + h(\mu + \eta + \gamma)} \tag{20.3}$$

$$S_2^{n+1} = \frac{S_2^n + h\eta S_1^n}{1 + h(1 - \theta_2)\beta(I_1^n + I_2^n) + h(\mu + \theta_2)} \tag{20.4}$$

$$E_2^{n+1} = \frac{E_2^n + h\eta E_1^n + h(1 - \theta_2)\beta S_2^n (I_1^n + I_2^n)}{1 + h(\mu + \delta)} \tag{20.5}$$

$$I_2^{n+1} = \frac{I_2^n + h\eta I_1^n + h\delta E_2^n}{1 + h(\mu + \gamma)} \tag{20.6}$$

Stability Analysis of NSFDF Scheme

After taking the partial derivative of NSFDF equations and putting the disease-free points, we got the matrix *J*.

$$F = \frac{S_1^n + h\Lambda}{1 + h(1 - \theta_1)\beta(I_1^n + I_2^n) + h(\mu + \eta + \theta_1)} \tag{21.1}$$

$$G = \frac{E_1^n + h(1 - \theta_1)\beta S_1^n (I_1^n + I_2^n)}{1 + h(\mu + \eta + \delta)} \tag{21.2}$$

$$H = \frac{I_1^n + h\delta E_1^n}{1 + h(\mu + \eta + \gamma)} \tag{21.3}$$

$$L = \frac{S_2^n + h\eta S_1^n}{1 + h(1 - \theta_2)\beta(I_1^n + I_2^n) + h(\mu + \theta_2)} \tag{21.4}$$

$$M = \frac{E_2^n + h\eta E_1^n + h(1 - \theta_2)\beta S_2^n (I_1^n + I_2^n)}{1 + h(\mu + \delta)} \tag{21.5}$$

$$N = \frac{I_2^n + h\eta I_1^n + h\delta E_2^n}{1 + h(\mu + \gamma)} \tag{21.6}$$

Differentiating these F, G, H, L, M and N with respect to compartmental model perimeters S, E, I and R. Now *J* will be

$$J = \begin{bmatrix} F_{S_1} & F_{E_1} & F_{I_1} & F_{S_2} & F_{E_2} & F_{I_2} \\ G_{S_1} & G_{E_1} & G_{I_1} & G_{S_2} & G_{E_2} & G_{I_2} \\ H_{S_1} & H_{E_1} & H_{I_1} & H_{S_2} & H_{E_2} & H_{I_2} \\ L_{S_1} & L_{E_1} & L_{I_1} & L_{S_2} & L_{E_2} & L_{I_2} \\ M_{S_1} & M_{E_1} & M_{I_1} & M_{S_2} & M_{E_2} & M_{I_2} \\ N_{S_1} & N_{E_1} & N_{I_1} & N_{S_2} & N_{E_2} & N_{I_2} \end{bmatrix},$$

From the above Jacobian matrix, we obtained eigenvalues. So, by the determinant of the Jacobian matrix we analyzed that our scheme is stable.

Consistency Analysis

Consistency analysis of NSFD scheme is performed by using Taylor's series expansion [29,30]. First, we took (20.1) then applied Taylor series

$$S_1^{n+1} = S_1^n + \frac{h dS_1^n}{dt} + \frac{h^2 d^2 S_1^n}{2! dt^2} + \frac{h^3 d^3 S_1^n}{3! dt^3} + \dots \tag{22}$$

In the following expression

$$S_1^{n+1} = \frac{S_1^n + h\Lambda}{1 + h(1 - \theta_1)\beta(I_1^n + I_2^n) + h(\mu + \eta + \theta_1)}$$

$$S_1^{n+1} (1 + h(1 - \theta_1)\beta(I_1^n + I_2^n) + h(\mu + \eta + \theta_1)) = S_1^n + h\Lambda$$

$$(S_1^n + \frac{h dS_1^n}{dt} + \frac{h^2 d^2 S_1^n}{2! dt^2} + \frac{h^3 d^3 S_1^n}{3! dt^3} + \dots) (1 + h(1 - \theta_1)\beta(I_1^n + I_2^n) + h(\mu + \eta + \theta_1)) = S_1^n + h\Lambda$$

$$S_1^n + S_1^n h(1 - \theta_1)\beta(I_1^n + I_2^n) + S_1^n h(\mu + \eta + \theta_1) + \frac{h dS_1^n}{dt} + \frac{h^2 d^2 S_1^n}{2! dt^2} \beta(1 - \theta_1)(I_1^n + I_2^n) + \frac{h^2 d^2 S_1^n}{2! dt^2} (\mu + \eta + \theta_1) + (\frac{h^3 d^3 S_1^n}{3! dt^3} + \dots) (1 + h(1 - \theta_1)\beta(I_1^n + I_2^n) + h(\mu + \eta + \theta_1)) = S_1^n + h\Lambda$$

$$S_1^n h(1 - \theta_1)\beta(I_1^n + I_2^n) + S_1^n h(\mu + \eta + \theta_1) + \frac{h dS_1^n}{dt} + \frac{h^2 d^2 S_1^n}{2! dt^2} \beta(1 - \theta_1)(I_1^n + I_2^n) + \frac{h^2 d^2 S_1^n}{2! dt^2} (\mu + \eta + \theta_1) + (\frac{h^3 d^3 S_1^n}{3! dt^3} + \dots) (1 + h(1 - \theta_1)\beta(I_1^n + I_2^n) + h(\mu + \eta + \theta_1)) = h\Lambda$$

By taking h common and then apply h→0

$$S_1^n (1 - \theta_1)\beta(I_1^n + I_2^n) + S_1^n (\mu + \eta + \theta_1) + \frac{dS_1^n}{dt} = \Lambda$$

$$\frac{dS_1^n}{dt} = \Lambda - S_1^n (1 - \theta_1)\beta(I_1^n + I_2^n) - S_1^n (\mu + \eta + \theta_1) \tag{23}$$

Similarly taking equation (20.2) and then apply Taylor expansion for E₁ⁿ⁺¹

$$E_1^{n+1} = E_1^n + \frac{h dE_1^n}{dt} + \frac{h^2 d^2 E_1^n}{2! dt^2} + \frac{h^3 d^3 E_1^n}{3! dt^3} + \dots \tag{24}$$

$$E_1^{n+1} = \frac{E_1^n + h(1 - \theta_1)\beta S_1^n (I_1^n + I_2^n)}{1 + h(\mu + \eta + \delta)}$$

$$E_1^{n+1} (1 + h(\mu + \eta + \delta)) = E_1^n + h(1 - \theta_1)\beta S_1^n (I_1^n + I_2^n)$$

$$(E_1^n + \frac{h dE_1^n}{dt} + \frac{h^2 d^2 E_1^n}{2! dt^2} + \frac{h^3 d^3 E_1^n}{3! dt^3} + \dots) (1 + h(\mu + \eta + \delta)) = E_1^n + h(1 - \theta_1)\beta S_1^n (I_1^n + I_2^n)$$

from the following expression we obtained,

$$E_1^n + E_1^n h(\mu + \eta + \delta) + \frac{h dE_1^n}{dt} + \frac{h^2 d^2 E_1^n}{2! dt^2} (\mu + \eta + \delta) + (\frac{h^3 d^3 E_1^n}{3! dt^3} + \dots) (1 + h(\mu + \eta + \delta)) = E_1^n + h(1 - \theta_1)\beta S_1^n (I_1^n + I_2^n)$$

after simplification and then apply h→0 we got

$$\frac{dE_1^n}{dt} = (1 - \theta_1)\beta S_1^n (I_1^n + I_2^n) - E_1^n (\mu + \eta + \delta) \tag{25}$$

Now by taking equation (20.3)

$$I_1^{n+1} = I_1^n + \frac{h dI_1^n}{dt} + \frac{h^2 d^2 I_1^n}{2! dt^2} + \frac{h^3 d^3 I_1^n}{3! dt^3} + \dots \tag{26}$$

$$I_1^{n+1} = \frac{I_1^n + h\delta E_1^n}{1 + h(\mu + \eta + \gamma)}$$

$$I_1^{n+1} (1 + h(\mu + \eta + \gamma)) = I_1^n + h\delta E_1^n$$

$$(I_1^n + \frac{h dI_1^n}{dt} + \frac{h^2 d^2 I_1^n}{2! dt^2} + \frac{h^3 d^3 I_1^n}{3! dt^3} + \dots)(1 + h(\mu + \eta + \gamma)) = I_1^n + h\delta E_1^n,$$

$$I_1^n + I_1^n h(\mu + \eta + \gamma) + \frac{h dI_1^n}{dt} + \frac{h^2 d^2 I_1^n}{2! dt^2} (\mu + \eta + \gamma) + (\frac{h^2 d^2 I_1^n}{2! dt^2} + \frac{h^3 d^3 I_1^n}{3! dt^3} \dots)(1 + h(\mu + \eta + \gamma)) = I_1^n + h\delta E_1^n,$$

By simplification and then after applying $h \rightarrow 0$ we got

$$\frac{dI_1^n}{dt} = \delta E_1^n - I_1^n(\mu + \eta + \gamma) \tag{27}$$

Now if we take equation (20.4)

$$S_2^{n+1} = S_2^n + \frac{h dS_2^n}{dt} + \frac{h^2 d^2 S_2^n}{2! dt^2} + \frac{h^3 d^3 S_2^n}{3! dt^3} + \dots \tag{28}$$

$$S_2^{n+1} = \frac{S_2^n + h \eta S_1^n}{1 + h(1 - \theta_2)\beta(I_1^n + I_2^n) + h(\mu + \theta_2)},$$

$$S_2^{n+1}(1 + h(1 - \theta_2)\beta(I_1^n + I_2^n) + h(\mu + \theta_2)) = S_2^n + h \eta S_1^n,$$

$$(S_2^n + \frac{h dS_2^n}{dt} + \frac{h^2 d^2 S_2^n}{2! dt^2} + \frac{h^3 d^3 S_2^n}{3! dt^3} + \dots)(1 + h(1 - \theta_2)\beta(I_1^n + I_2^n) + h(\mu + \theta_2)) = S_2^n + h \eta S_1^n,$$

by applying simplification and then $h \rightarrow 0$ we got

$$\frac{h dS_2^n}{dt} = \eta S_1^n - (1 - \theta_2)\beta S_2^n (I_1^n + I_2^n) - S_2^n(\mu + \theta_2) \tag{29}$$

By equation (20.5)

$$E_2^{n+1} = E_2^n + \frac{h dE_2^n}{dt} + \frac{h^2 d^2 E_2^n}{2! dt^2} + \frac{h^3 d^3 E_2^n}{3! dt^3} + \dots \tag{30}$$

$$E_2^{n+1} = \frac{E_2^n + h\eta E_1^n + h(1 - \theta_2)\beta S_2^n (I_1^n + I_2^n)}{1 + h(\mu + \delta)},$$

$$E_2^{n+1}(1 + h(\mu + \delta)) = E_2^n + h\eta E_1^n + h(1 - \theta_2)\beta S_2^n (I_1^n + I_2^n),$$

$$(E_2^n + \frac{h dE_2^n}{dt} + \frac{h^2 d^2 E_2^n}{2! dt^2} + \frac{h^3 d^3 E_2^n}{3! dt^3} + \dots)(1 + h(\mu + \delta)) = E_2^n + h\eta E_1^n + h(1 - \theta_2)\beta S_2^n (I_1^n + I_2^n),$$

by simplification and with same step as above we got

$$\frac{dE_2^n}{dt} = \eta E_1^n + h(1 - \theta_2)\beta S_2^n (I_1^n + I_2^n) - E_2^n(\mu + \delta) \tag{31}$$

with same method, by applying expansion and then $h \rightarrow 0$ we can get our system from equation (20.6)

$$\frac{dI_2^n}{dt} = \eta I_1^n + \delta E_2^n - I_2^n(\mu + \gamma) \tag{32}$$

Hence NSFD numerical scheme is consistent with the system.

Graphs at Different Points

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

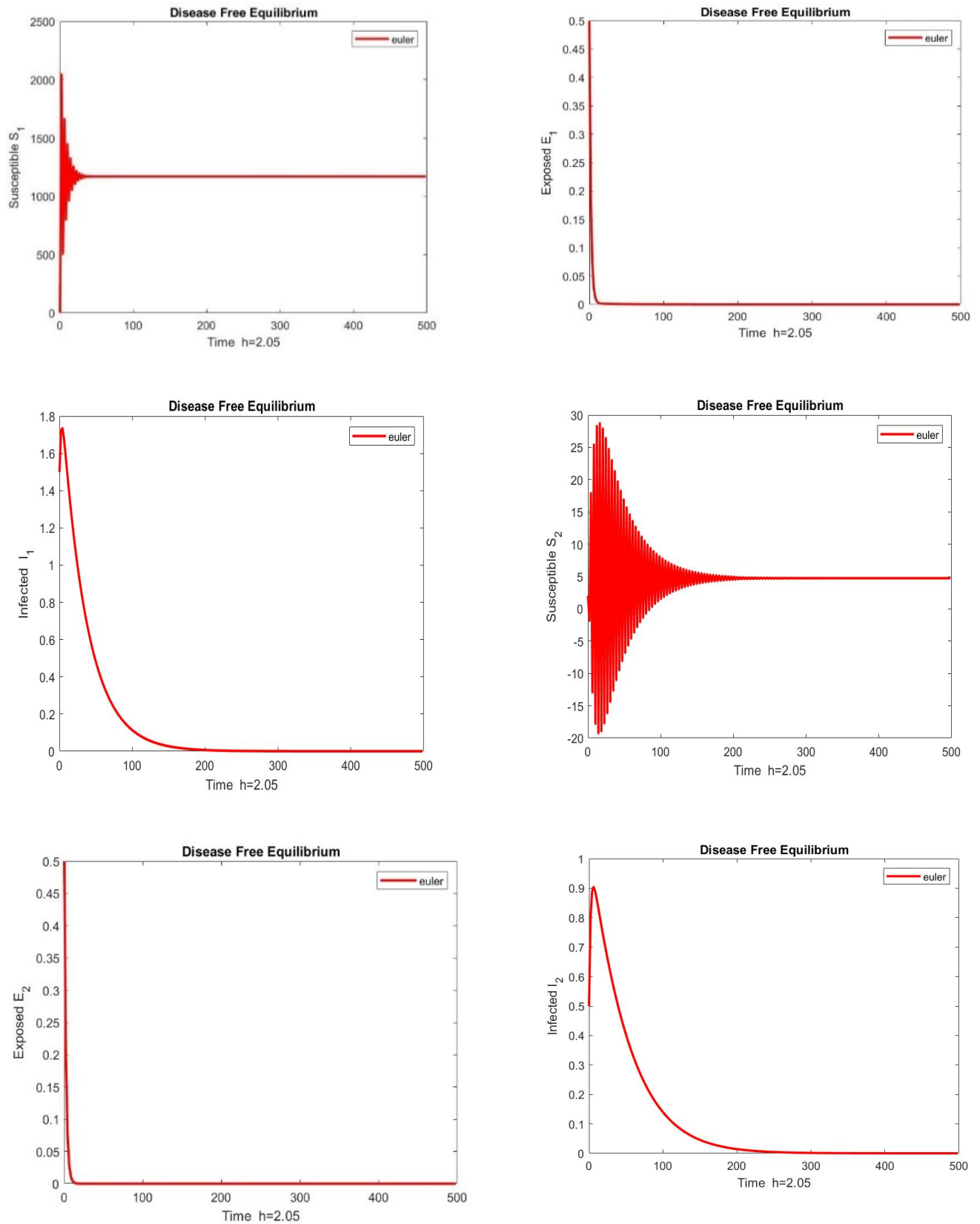


Figure 3. Euler behavior on SEIR first and second dose when $h = 2.05$

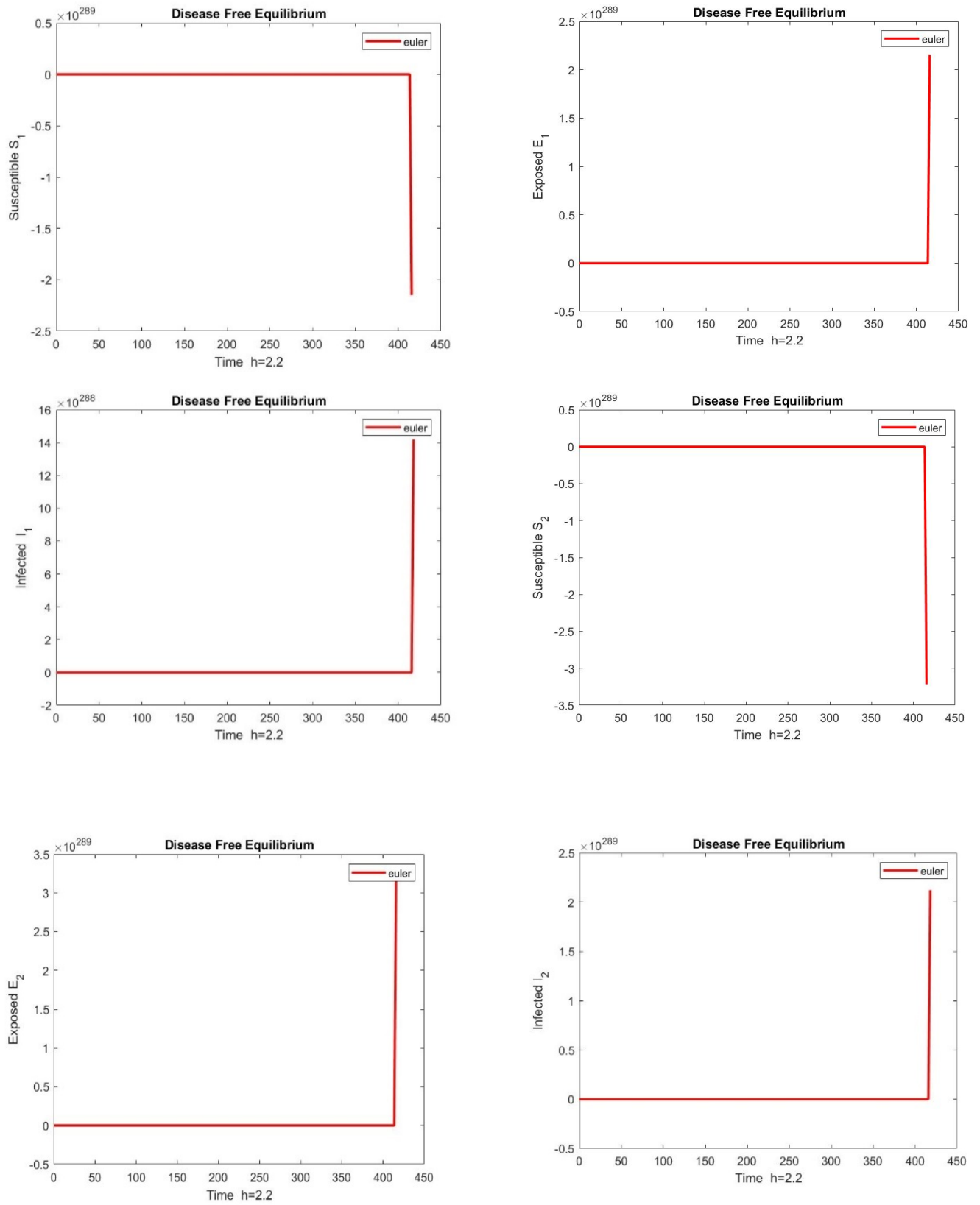


Figure 4. Euler behavior on SEIR first and second dose when $h = 2.2$

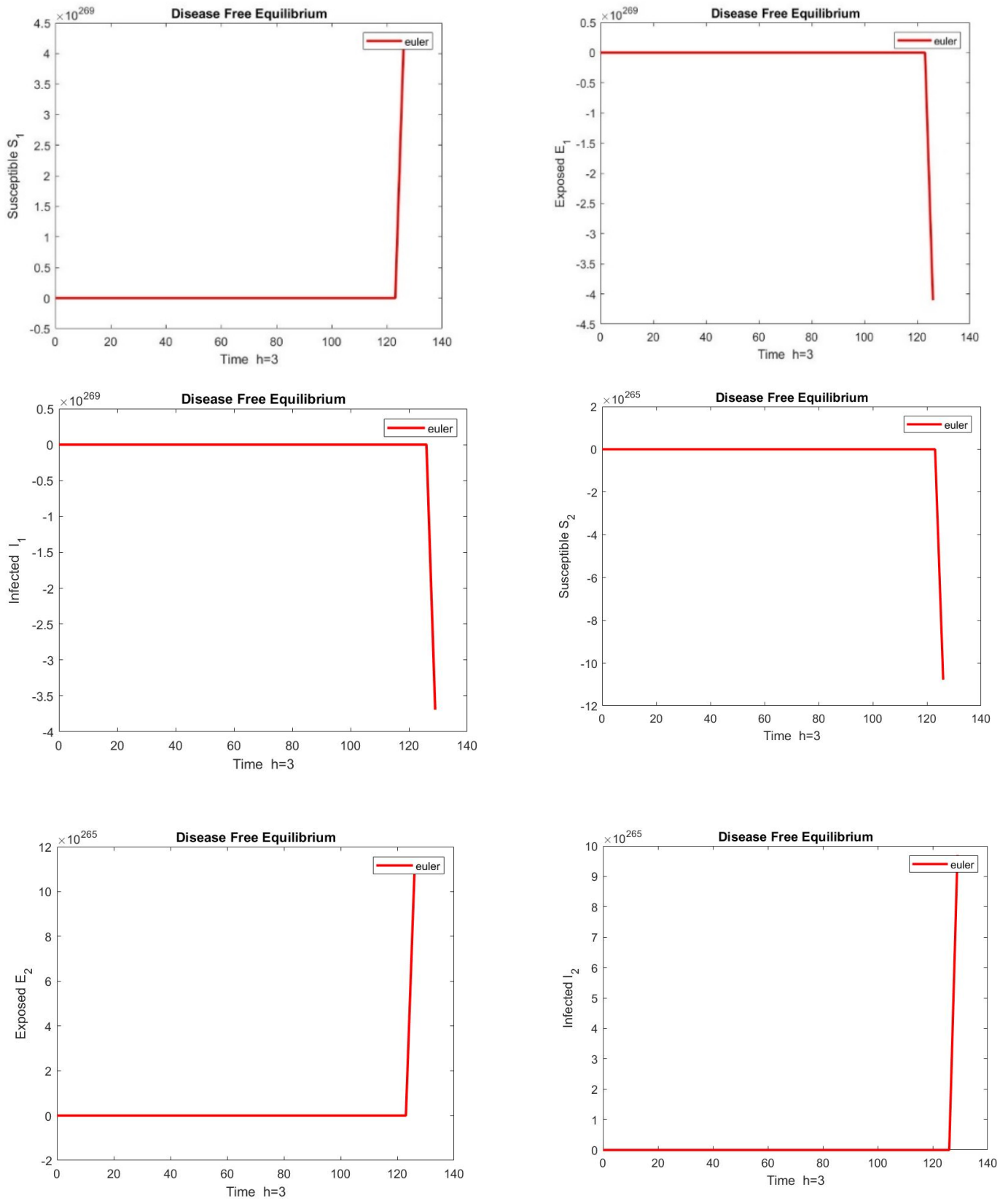


Figure 5. Euler behavior on SEIR first and second dose when $h = 3.0$

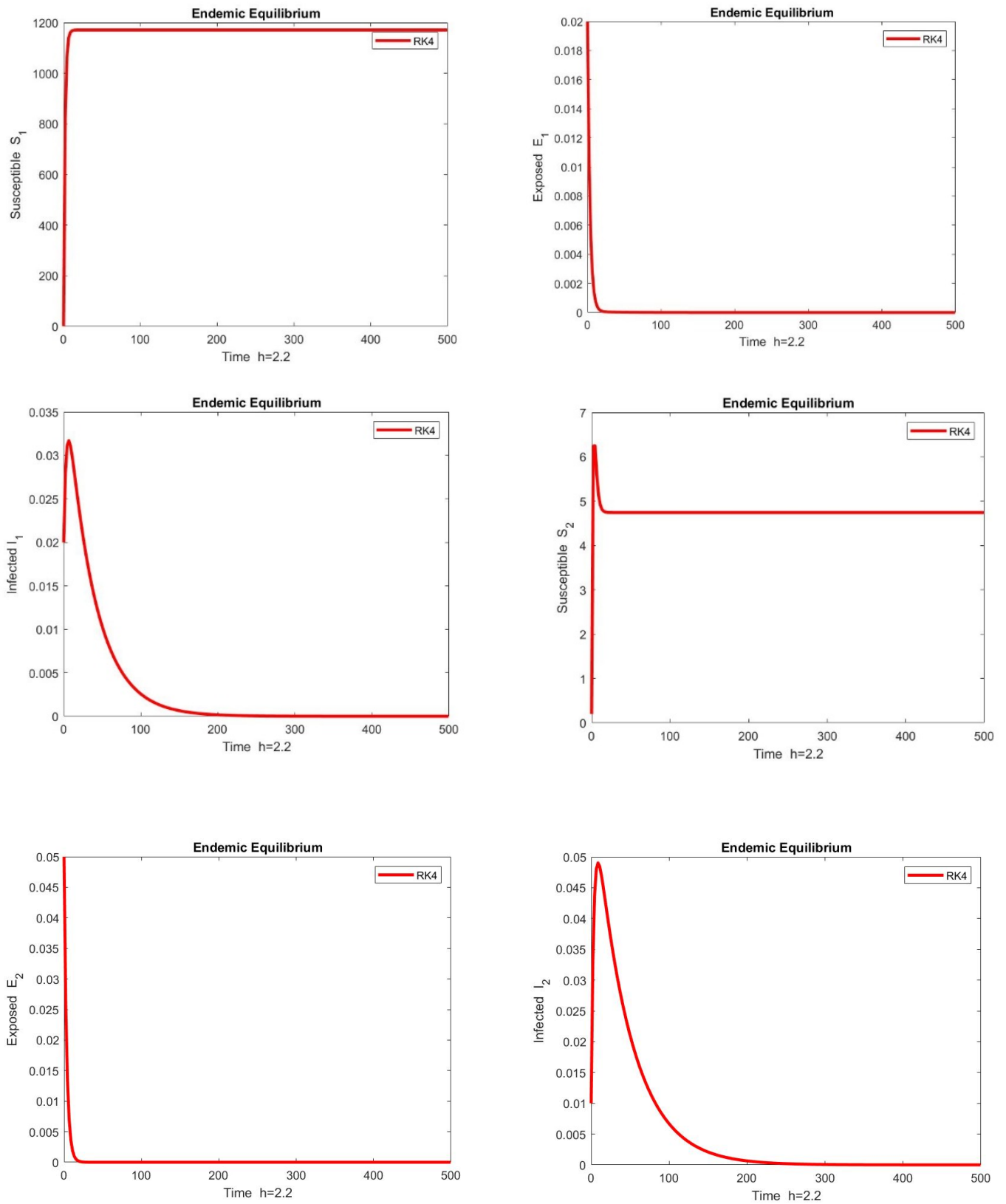


Figure 6. RK4 behavior on SEIR first and second dose when $h = 2.2$

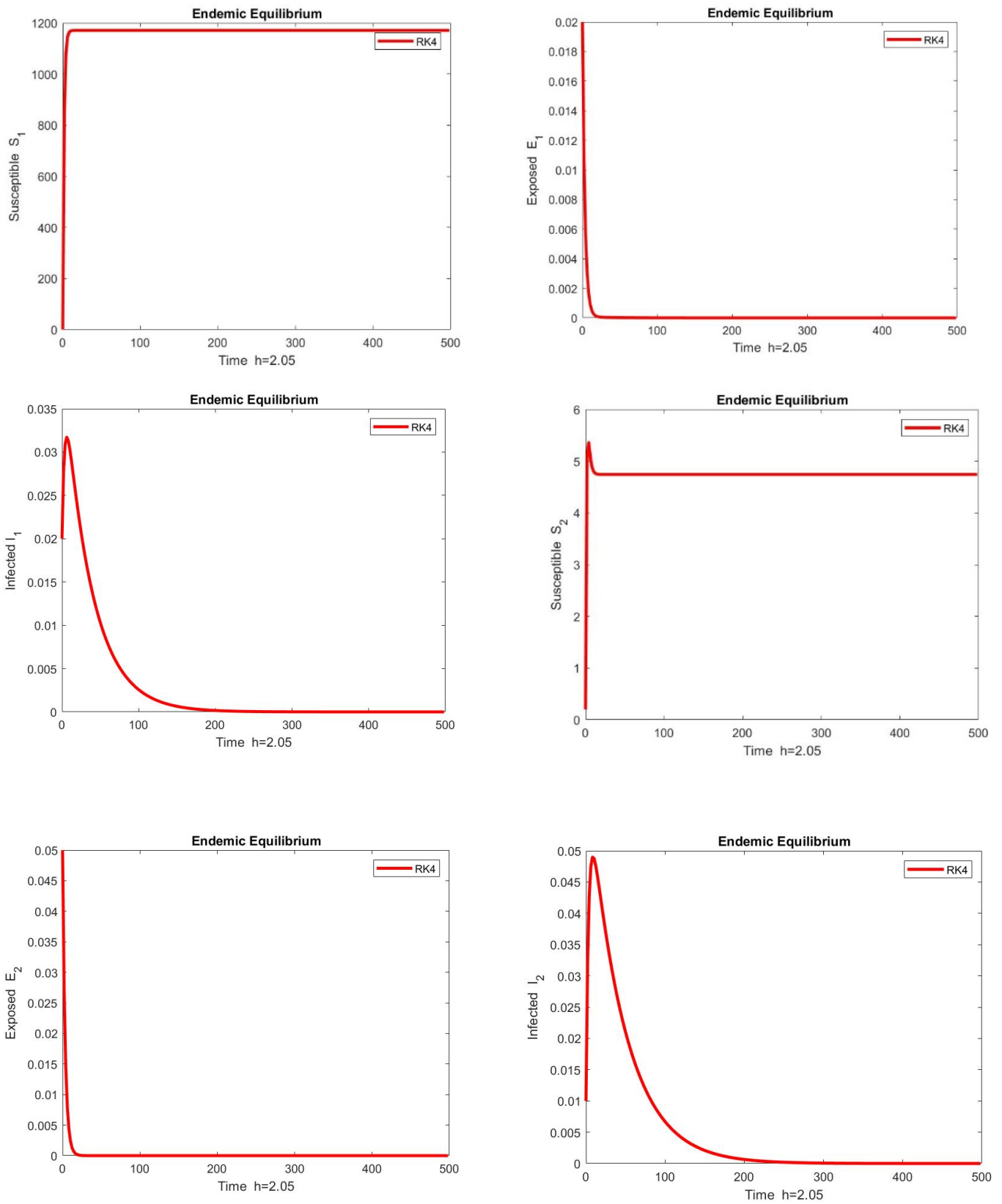


Figure 7. RK4 behavior on SEIR first and second dose when $h = 2.05$

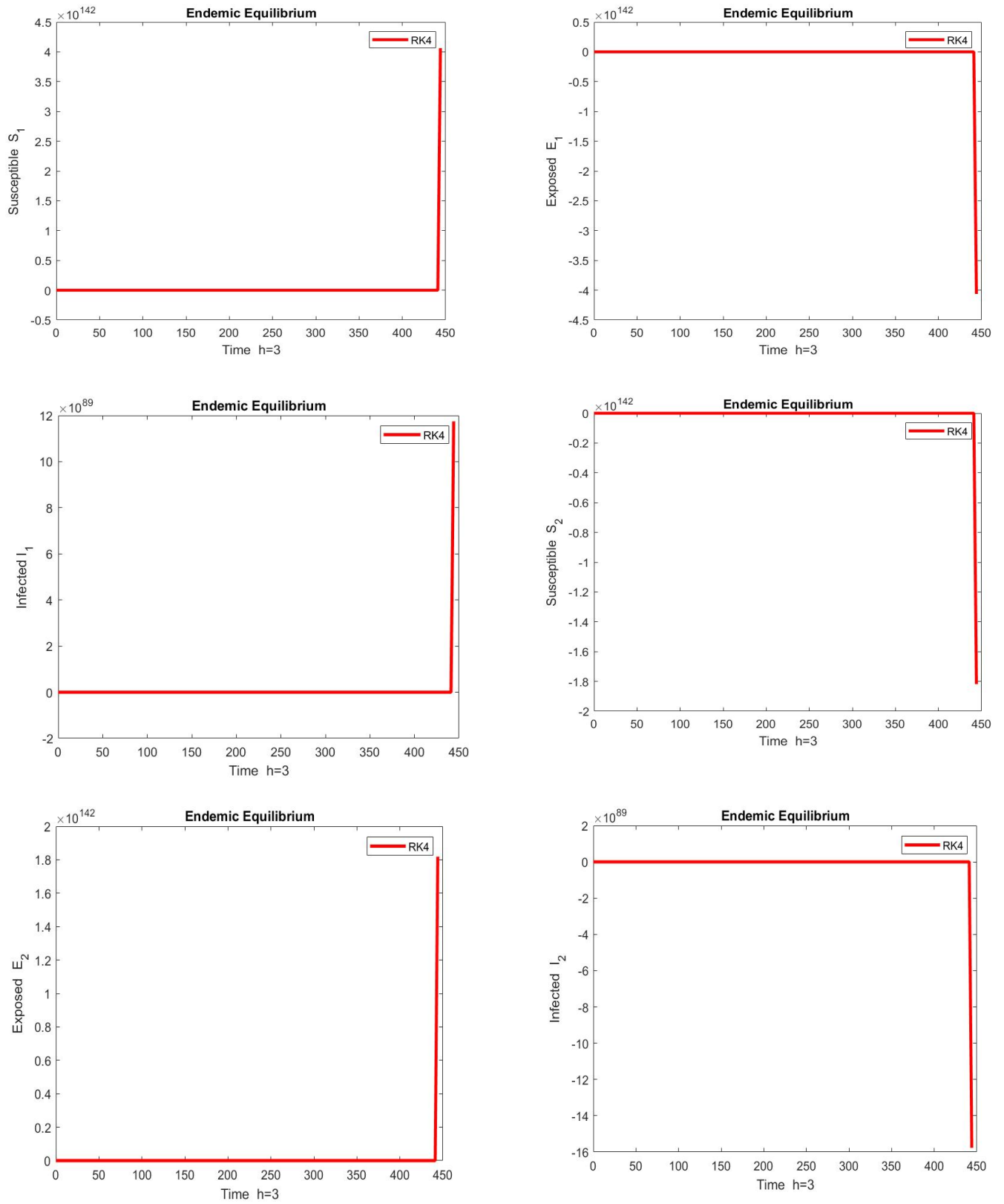


Figure 8. RK4 behavior on SEIR first and second dose when $h = 3$

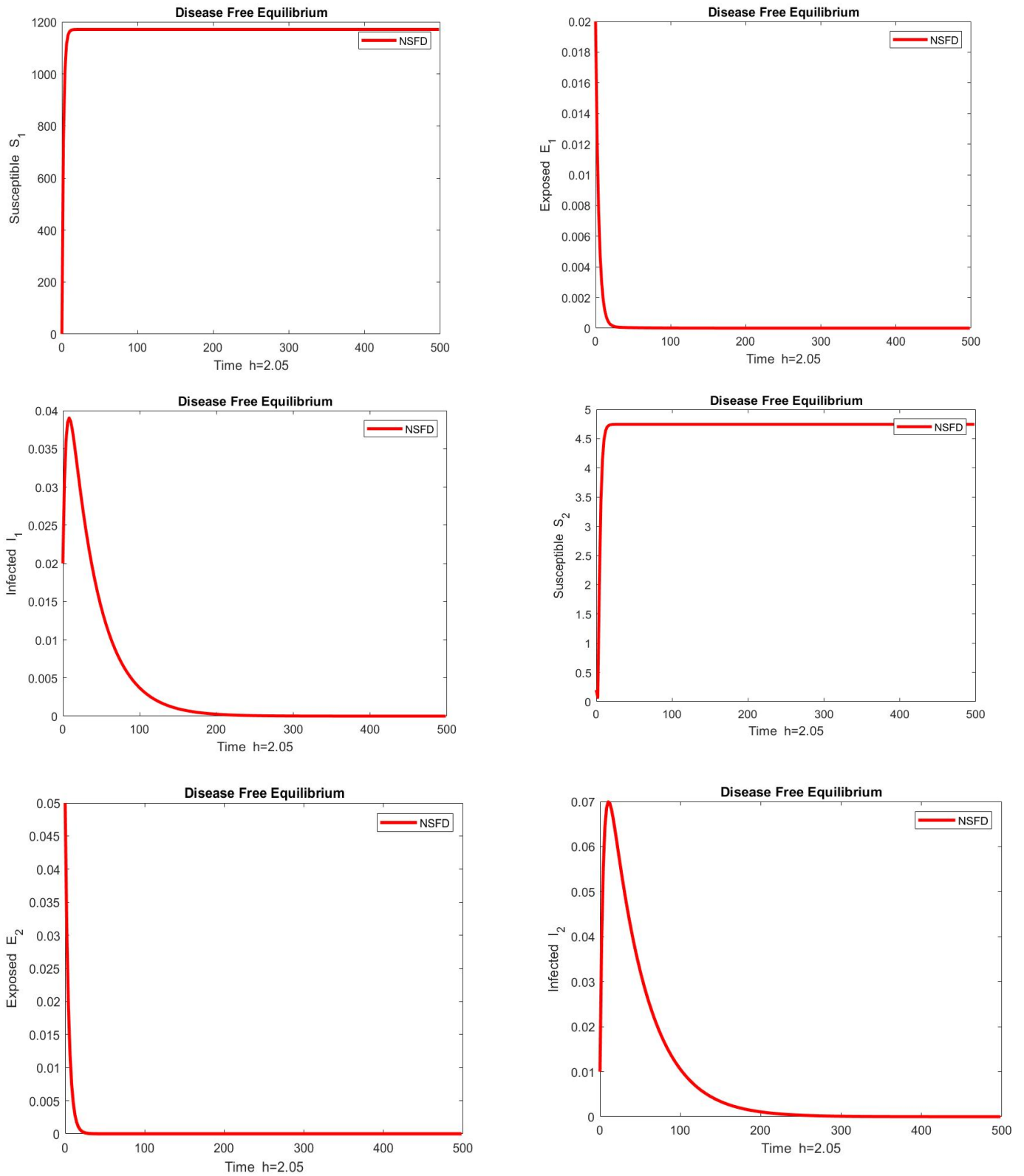


Figure 9. NSFD behavior on SEIR first and second dose when $h = 2.05$

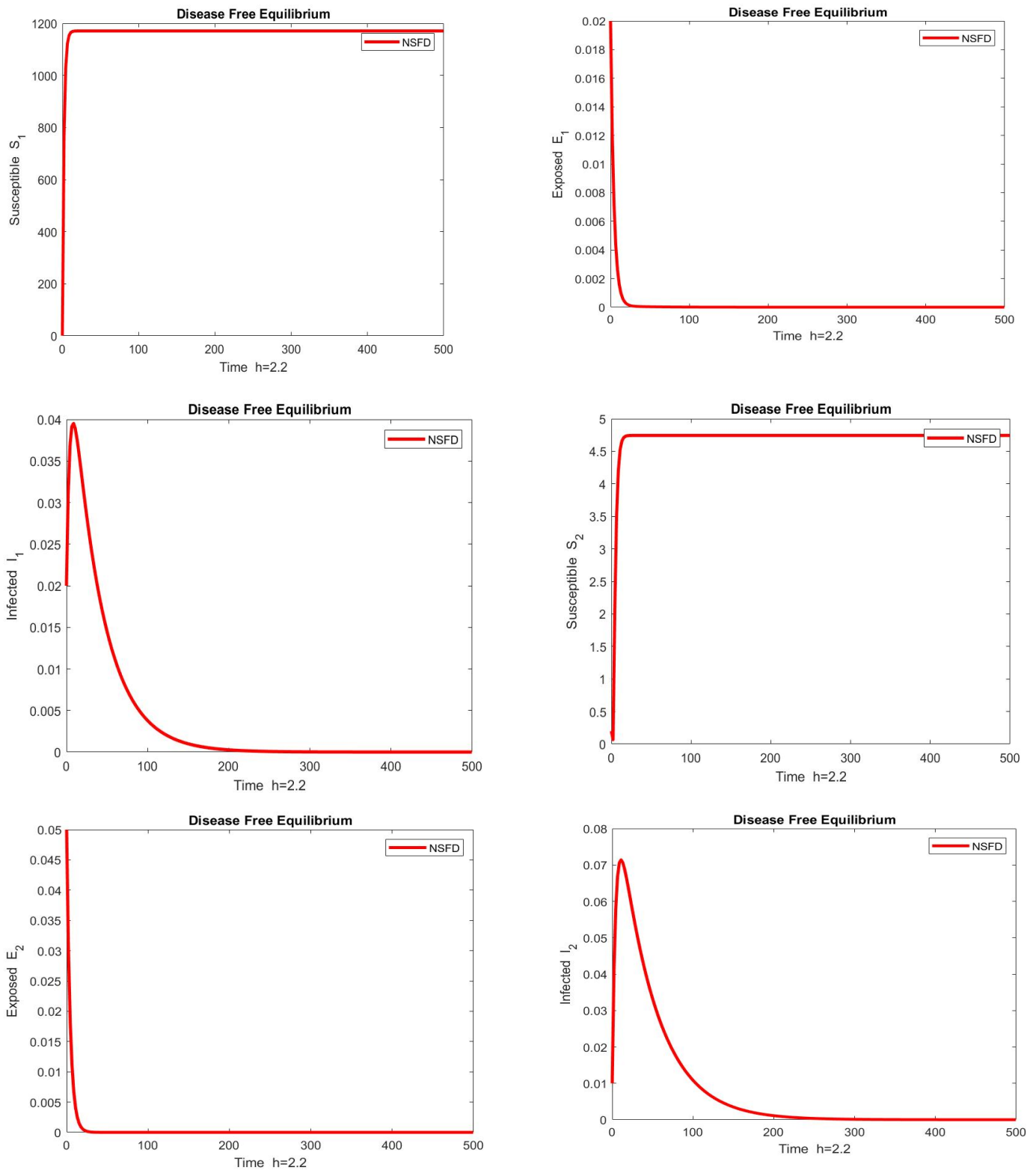


Figure 10. NSFD behavior on SEIR first and second dose when $h = 2.2$

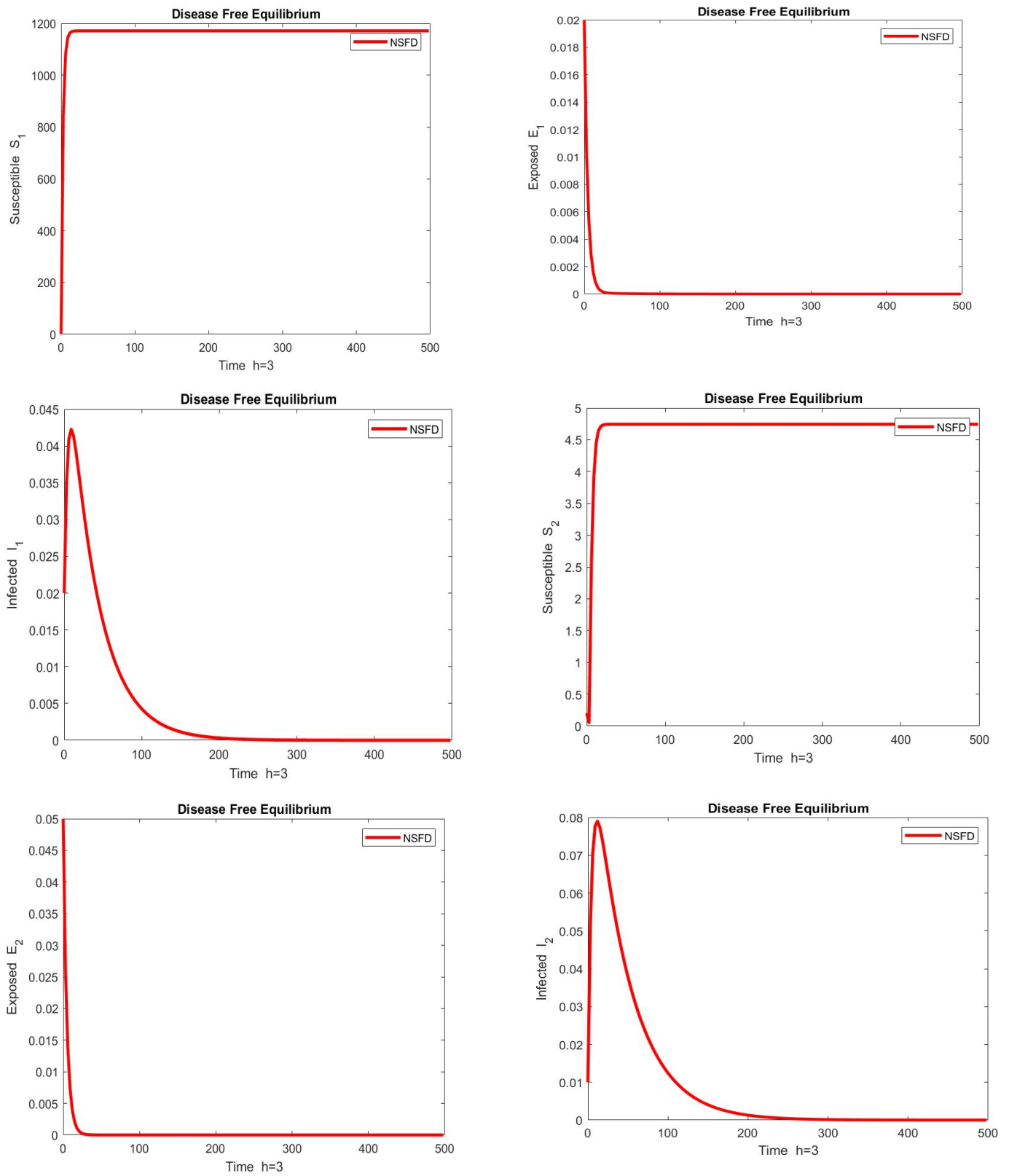


Figure 11. NSFD behavior on SEIR first and second dose when $h = 3$

The Euler and RK4 techniques can provide divergent solutions at tiny step sizes and different behaviour at large step size as shown in **figure 3, 4, 5, 6, 7 and 8**. By way of comparison, the Non-Standard Finite Difference (NSFD) approach produces convergent solutions even with same and tiny step sizes while maintaining the system's qualitative characteristics as shown in **figure 9, 10 and 11**. Because of this, NSFD ensures numerical stability and is more suited for managing intricate epidemiological models [27][28].

Hence by graphically analysis proved that NSFD is more reliable than Euler and Rk4. 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. This makes NSFD particularly useful for guiding public health strategies like the WHO's two-dose vaccination plan. The NSFD method, helps in preserving realistic properties, ensuring that solutions remain biologically feasible across longer time periods, which strengthens the model's practical applicability for public health planning.

Conclusions

Mathematical modelling of epidemiological disorders is a significant tool for studying disease dynamics. We have seen significant solutions to overcoming the disasters of vaccination loss worldwide. Model equilibrium points and reproductive number discovered. As reproductive number increase the disease spread in the community while it decreases then disease reduce. Effect of parameters play a vital role on reproductive number that we can easily identify by sensitivity analysis. With the help of a nonstandard finite difference method, an accurate and dependable numerical solution of a vaccination epidemic model is offered in this study effort. The suggested method keeps all the fundamental aspects of the vaccination epidemic model, demonstrating its efficacy. The well-defined NSFD technique compared with Euler and RK-4 methods.

The simulations revealed that both classical approaches were unsuccessful in providing faultless results, even at very small step sizes. Furthermore, the model's equilibrium points are determined, and it is discovered that the system has two steady states, one of which is disease free, and the other is endemic equilibrium. The stability at DFE and EE points is being studied. The role of R_0 in determining the basic reproduction number is being investigated when $R_0 < 1$, the equilibrium point is locally asymptotically stable; however, when $R_0 > 1$, it is unstable. In this paper, we compared forward Euler, RK-4 and NSFD method. NSFD is more sufficient and reliable as compared to forward Euler and RK-4 because NSFD shows convergence at very small step size. NSFD is dynamically consistent scheme and also by graphical results we proved that NSFD method hold the properties of SEIR mathematical model. Hence this model is reliable and also will provide better results with NSFD technique. This approach can contribute to further understanding of rubella dynamics, enhancing the model's applicability in public health planning. In the future, this model can be extended by incorporating fractional order derivatives by integrating stochastic elements to account for variability and uncertainty in disease transmission, further enhancing its applicability for real-world public health scenarios.

Data Availability

All data available inside the document.

Conflicts of Interest

The authors declare that there is no conflict of interest regarding the publication of this paper.

Acknowledgement

We would like to acknowledge the support received from the Universiti Sains Malaysia main campus also, we thanks to reviewers for their valuable feedback.

References

- [1] Artiono, R., & Prawoto, B. P. (2021). The dynamics of rubella virus with two-dose vaccination strategy. *Commun. Math. Biol. Neurosci.*, 2021.
- [2] Artiono, R., & Prawoto, B. P. (2020). The effects of vaccination to the dynamics of rubella virus with seasonality. *Commun. Math. Biol. Neurosci.*, 2020.
- [3] Lambert, N., Strebel, P., Orenstein, W., Icenogle, J., & Poland, G. A. (2015). Rubella. *The Lancet*, 385(9984), 2297–2307.
- [4] Artiono, R., & Prawoto, B. P. (2021). The dynamics of rubella virus with two-dose vaccination strategy. *Commun. Math. Biol. Neurosci.*, 2021.
- [5] Vynnycky, E., Adams, E. J., Cutts, F. T., Reef, S. E., Navar, A. M., Simons, E., *et al.* (2016). Using seroprevalence and immunisation coverage data to estimate the global burden of congenital rubella syndrome, 1996–2010: A systematic review. *PLoS One*, 11(3), e0149160.
- [6] Jember, T. H. (2014). Challenges of schistosomiasis prevention and control in Ethiopia: Literature review and current status. *J Parasitol Vector Biol*, 6(6), 80–86.
- [7] Vynnycky, E., Adams, E. J., Cutts, F. T., Reef, S. E., Navar, A. M., Simons, E., & Dabbagh, A. J. (2016). Using seroprevalence and immunisation coverage data to estimate the global burden of congenital rubella syndrome, 1996–2010: A systematic review. *PLoS One*, 11(3), e0149160.
- [8] Artiono, R., & Prawoto, B. P. (2021). The dynamics of rubella virus with two-dose vaccination strategy. *Commun. Math. Biol. Neurosci.*, 2021, Article-ID.
- [9] Shafayi, A., & Mohammadi, A. (2021). A review on rubella vaccine: Iran (1975–2010). *Archives of Razi Institute*, 76(2), 167.
- [10] LeBaron, C. W., Forghani, B., Matter, L., Reef, S. E., Beck, C., Bi, D., *et al.* (2009). Persistence of rubella antibodies after 2 doses of measles-mumps-rubella vaccine. *The Journal of Infectious Diseases*, 200(6), 888–899.
- [11] Hamborsky, J., Kroger, A., & Wolfe, C. (Eds.). (2015). *Epidemiology and prevention of vaccine-preventable diseases: The Pink Book: Course Textbook*. Public Health Foundation.
- [12] Gao, Z., Wood, J. G., Burgess, M. A., Menzies, R. I., McIntyre, P. B., & MacIntyre, C. R. (2013). Models of strategies for control of rubella and congenital rubella syndrome: A 40-year experience from Australia. *Vaccine*, 31(4), 691–697.
- [13] Wu, Y., Wood, J., Khandaker, G., Waddington, C., & Snelling, T. (2016). Informing rubella vaccination strategies in East Java, Indonesia through transmission modelling. *Vaccine*, 34(46), 5636–5642.
- [14] Brauer, F. (2005). The Kermack–McKendrick epidemic model revisited. *Mathematical Biosciences*, 198(2), 119–131.
- [15] Zhou, L., Wang, Y., Xiao, Y., & Li, M. Y. (2019). Global dynamics of a discrete age-structured SIR epidemic model with applications to measles vaccination strategies. *Mathematical Biosciences*, 308, 27–37.
- [16] Sun, C., & Hsieh, Y. H. (2010). Global analysis of an SEIR model with varying population size and vaccination. *Applied Mathematical Modelling*, 34(10), 2685–2697.
- [17] Hethcote, H. W. (1976). Qualitative analyses of communicable disease models. *Mathematical Biosciences*, 28(3–4), 335–356.
- [18] Tamhaji, N. H., & Hamdan, N. I. (2023). The dynamics of tuberculosis through BSEIR model with immigration in Malaysia. *Malaysian Journal of Fundamental and Applied Sciences*, 19(6), 1176–1189.
- [19] Erinde-Ibrahim, L. M., Lawal, W. O., Adebimpe, O., & Sontan, G. R. (2021). A susceptible exposed infected recovered susceptible (SEIRS) model for the transmission of tuberculosis. *Tanzania Journal of Science*, 47(3), 917–927.
- [20] Widyaningsih, P., Nugroho, A. A., & Saputro, D. R. S. (2018, September). Susceptible infected recovered model with vaccination, immunity loss, and relapse to study tuberculosis transmission in Indonesia. In *AIP Conference Proceedings* (Vol. 2014, No. 1). AIP Publishing.
- [21] Chantler, J. K., Tingle, A. J., & Petty, R. E. (1985). Persistent rubella virus infection associated with chronic arthritis in children. *New England Journal of Medicine*, 313(18), 1117–1123.
- [22] Patel, M. K., Antoni, S., Danovaro-Holliday, M. C., Desai, S., Gacic-Dobo, M., Nedelec, Y., & Kretsinger, K. (2020). The epidemiology of rubella, 2007–18: An ecological analysis of surveillance data. *The Lancet Global Health*, 8(11), e1399–e1407.
- [23] Alfwzan, W. F., Baleanu, D., Raza, A., Rafiq, M., & Ahmed, N. (2023). Dynamical analysis of a class of SEIR models through delayed strategies. In *AIP Advances* (Vol. 13, Issue 7). AIP Publishing. <https://doi.org/10.1063/5.0159942>
- [24] Naveed, M., Baleanu, D., Raza, A., Rafiq, M., & Hassan Soori, A. (2022). Treatment of polio delayed epidemic model via computer simulations. In *Computers, Materials & Continua* (Vol. 70, Issue 2, pp. 3415–3431). Computers, Materials and Continua (Tech Science Press). <https://doi.org/10.32604/cmc.2022.020112>
- [25] Ahmad, W., Butt, A. I. K., Akhtar, N., Rafiq, M., Gohar, M., Idrees, Z., & Ahmad, N. (2024). Developing computationally efficient optimal control strategies to eradicate rubella disease. In *Physica Scripta* (Vol. 99, Issue 3, p. 035202). IOP Publishing. <https://doi.org/10.1088/1402-4896/ad1fc0>
- [26] Butt, A. I. K., Ahmad, W., Rafiq, M., Ahmad, N., & Imran, M. (2023). Computationally efficient optimal control analysis for the mathematical model of Coronavirus pandemic. In *Expert Systems with Applications* (Vol. 234, p. 121094). Elsevier BV. <https://doi.org/10.1016/j.eswa.2023.121094>
- [27] Raza, A., Rafiq, M., Ahmed, N., Sajid Iqbal, M., Rezapour, S., & Inc, M. (2024). Computer modeling: A gateway to novel advancements in solving real-life problems. In *Biomedical Signal Processing and Control* (Vol. 95, p. 106414). Elsevier BV. <https://doi.org/10.1016/j.bspc.2024.106414>
- [28] Raza, A., Awrejcewicz, J., Rafiq, M., & Mohsin, M. (2021). Breakdown of a nonlinear stochastic Nipah virus epidemic model through efficient numerical methods. *Entropy*, 23(12), 1588.

- <https://doi.org/10.3390/e23121588>
- [29] Alqarni, M. M., Nasir, A., Alyami, M. A., Raza, A., Awrejcewicz, J., Rafiq, M., Ahmed, N., Sumbal Shaikh, T., & Mahmoud, E. E. (2022). A SEIR epidemic model of whooping cough-like infections and its dynamically consistent approximation. In S. A. Cheong (Ed.), *Complexity* (Vol. 2022, pp. 1–13). Hindawi Limited. <https://doi.org/10.1155/2022/3642444>
- [30] Zeb, S., Mohd Yatim, S. A., Rafiq, M., Ahmad, W., Kamran, A., & Karim, Md. F. (2024). Treatment and delay control strategy for a non-linear Rift Valley fever epidemic model. In *AIP Advances* (Vol. 14, Issue 11). AIP Publishing. <https://doi.org/10.1063/5.0228513>
- [31] LeBaron, C. W., Forghani, B., Matter, L., *et al.* (2009). Persistence of rubella antibodies after 2 doses of measles-mumps-rubella vaccine. *J. Infect. Dis.*, 200, 888–899.
- [32] Saleem, S., Rafiq, M., Ahmed, N., *et al.* (2024). Fractional epidemic model of coronavirus disease with vaccination and crowding effects. *Sci Rep*, 14, 8157. <https://doi.org/10.1038/s41598-024-58192-7>