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Essential Features Preserving Dynamics of Stochastic Dengue Model

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ABSTRACT

Nonlinear stochastic modelling plays an important character in the different fields of sciences such as environmental, material, engineering, chemistry, physics, biomedical engineering, and many more. In the current study, we studied the computational dynamics of the stochastic dengue model with the real material of the model. Positivity, boundedness, and dynamical consistency are essential features of stochastic modelling. Our focus is to design the computational method which preserves essential features of the model. The stochastic non-standard finite difference technique is most efficient as compared to other techniques used in literature. Analysis and comparison were explored in favour of convergence. Also, we address the comparison between the stochastic and deterministic models.

KEYWORDS

Dengue model; stochastic ordinary differential equations; numerical methods; convergence of the proposed method

1 Introduction

Dengue disease was first described in 1780 when dengue cases are observed in the Philadelphia epidemic (Rush 1789). In 1906, dengue transmission was established by the Aedes mosquito. In 1953, dengue hemorrhagic was first apprehend in the Philippines and 1955 it was recognized in



Thailand. Dengue virus is one of the most common viruses. Four dengue (DENV-1 to DENV-4) serotypes viruses that can spread to feminine mosquitoes causes dengue fever (DF). A DENV serotype induces permanent immunity to serotypes but only partial temporary immunity to three different serotypes. Annually there are about 390 million dengue infections, of which 96 million are registered on a clinical basis (any severity of the disease). DENV infection is at risk for 128 countries. This is because of a mosquito-borne virus. A mosquito bites a human and repeats this process to another human, and this cycle continues. The rainy season is a suitable season for this virus [1]. Virus serotypes cause the disease (DENV 1 to 4) not all of these were exposed at the same time. DENV 1 and 2 were exposed in 1960 [2]. The DENV-3 strain of dengue was exposed in 1961, while DENV-4 was discovered in 1963. The disease was restricted in the middle of the 20th century. The fifty million dengue infections have been reported globally by the world health organization and 2.5 million people face the illness. Dengue virus can change into (DHF) and (DSS). The patient suffers from bleeding and plasma leakage in DHF. Whereas, in (DSS) the patient suffers from low blood pressure at a dangerous level. The dengue virus is incurable. No medicine is discovered to cure this disease permanently. It is the only way to overcome its spreading sources, namely water storage in car pipes used in containers and other sources that can store rainwater. Aedes virus bites its prey two times a day in the morning and also in the evening. It is an old virus which was at first found in China in 610 AD and then in 922 AD. It was multiplied in the 18th and 19th centuries. It was first isolated in Japan in 1943 and 1944 in Calcutta, an Indian city. Before 1980, Africa was the only region that was unknown from this virus, but in 1997 the virus disturbed the tropical and subtropical areas of the world [3]. For the past couple of years, many authors have contributed to the development of mathematical dengue disease modelling. In order to model dynamics and to determine the efficacy or cost efficiency of behaviour, mathematical simulations were used. Some models were compartmental, usually suggesting that the mosquito bites of humans are universally mixed, i.e., every mosquito can bite every host with the same probability. However, this concept contrasts with actual epidemics that are heterogeneous at multiple levels: Geographic heterogeneity and heterogeneity in individuals. In particular, environmental and ecological change has influenced spatial heterogeneity leading to poorly coupled host mosquito encounters. In particular, the contact to mosquito bites was due to the heterogeneous nature of hosts, such as their proximity to aquatically immature mosquito habitats, their house type, their use of insecticides, human movement, and so on. Heterogeneous heterogeneity was characterized by some mosquitoes to have more muzzle than other mosquitoes, correlated with, among others, sex, age, height, pregnancy, protective behaviour, type of blood and human odour variability. The effects of heterogeneity on transmission dynamics have already been discussed with metapopulation models and agent-based models. This subject, however, is still more important. Nevai et al. have used a negative binomial distribution (NBD) approach in order to investigate the heterogeneity rates of Middle East respiratory coronavirus transmission in South Korea by 2015 and have found considerable potential for over-spreading. The NBD was widely used for representing count data, particularly for data with excessive dispersion. In biology literature, NBD was used to investigate the effect of transmission heterogeneity, including heterogeneity, in various transmission dynamic models [4]. It was beneficial to forecast dengue, malaria, and chikungunya spatial time dynamics as a result of this integrated approach [5]. A multi-strain dengue model involving four equilibrium points was proposed by authors. The effects of reinfection with the same serotype on transmission dynamics of dengue were discussed in that mathematical model [6]. Tay constructed a secondary dengue infection model involving two human populations named as seropositive and seronegative [7,8]. The ratio of these two serotypes was calculated. This ratio could be used as a helping tool in vaccination programs. In 2018 a

human and vector population-based SIR dengue model was proposed by the authors [9,10]. The impacts of vaccination on transmission of dengue were taken into account. Routh–Hurwitz criteria were utilized to explore the stability of the system. Three strategies named mechanical control, chemical control and vaccination were suggested to eradicate the dengue disease. However, only vaccination was found to be a more authentic tool for elimination presented in [11–14]. In recent years, many studies have been conducted for essential features preserving analysis of well-known stochastic epidemic models. Raza et al. [15] investigated the structure preserving analysis of the stochastic meme epidemic model. In which, analyzed the randomness of the rumours in social life. Abodayeh et al. [16] studied the dynamics of the stochastic vector borne plant disease, in which how the plant population may infect insects. Secondly, the numerical analysis of the effect of alcohol with gonorrhoea disease in the human population [17]. Thirdly, the stochastic analysis of foot and mouth disease dynamics is investigated in the animal population [18]. The authors discussed a dengue transmission model derived from nonlinear fractional-order differential equations. Mathematically modelling has come forward as an efficient tool to extract in the right about diseases. Usually, the determination of the stochastic differential equation is difficult and does not have closed-form solutions. We used different numerical techniques for this purpose. A natural question about these: Does any numerical scheme reserve the system’s dynamic properties? Our emphasis is on proposing a stochastic non-standard SNSFD (finite difference technique) for this model, and this paper’s game plan is as follows. In Section 2, we discussed the deterministic model and its equilibrium points. In Section 3, we explore the stochastic dengue model and introduce different techniques and also compare their results which deterministic solutions. In the final section, we come to a conclusion and suggest future guidelines.

2 Preliminaries

A GBM or (otherwise called exponential Brownian motion) is a ceaseless time stochastic procedure wherein the logarithm of the haphazardly fluctuating amount pursues a Brownian movement (additionally known as Wiener procedure) with the float. It is a significant cause of stochastic procedures fulfilling a stochastic differential condition (SDE). Specifically, it is utilized in the scientific fund to show stock costs operating at a profit Scholes model [19]. A Geometric Brownian movement $X(t)$ is the arrangement of an SDE with straight float and dispersion coefficients.

$$dX(t) = \mu X(t) dt + \sigma X(t) dW(t). \quad (1)$$

with initial values $X(0) = x_0$.

3 Deterministic Material

In this section, we considered the dynamics of the deterministic model, as presented in [20]. For any time t , the susceptible humans are detailed as $S_H(t)$, the asymptomatic infected humans are detailed as $A_H(t)$, the symptomatic infected humans are detailed as $I_H(t)$, $T_H(t)$ individual treated humans, $F_H(t)$ represent the humans who fail treatment, $S_V(t)$ shows the amount of disposed of mosquitoes, $E_V(t)$ represent visible mosquitoes and $I_V(t)$ shows the quantity of quick-spreading mosquitoes. The transmission flow is modelled in Fig. 1.

The transmission rates are explained as λ_H (denotes the rate at which the person infected), λ_V (the rate at which mosquitoes infected), μ_V (denotes natural death rate of mosquitoes), μ_H (denotes the natural death rate of persons), λ_V (represented the rate of infection of mosquitoes), Π_H (represented the new recruitment of the humans), Π_V (represented the new recruitment of the

mosquitoes), σ_H (probability of transmission A_H to I_H classes), σ_V (probability of transmission I_V to E_V classes), δ_H (represented the mortality rate of humans), δ_V (represented the mortality rate of the mosquitoes), τ_H (represented the rate of vaccination of the humans), γ_H (represented the rate of unsuccessful vaccination). The nonlinear equations of the model as follows:

$$S'_H = \Pi_H - \lambda_H S_H I_V - \mu_H S_H, \tag{2}$$

$$A'_H = \lambda_H S_H I_V - \sigma_H A_H I_H - \mu_H A_H. \tag{3}$$

$$I'_H = \sigma_H A_H I_H - \tau_H I_H T_H - \mu_H I_H, \tag{4}$$

$$T'_H = \tau_H I_H T_H - \gamma_H F_H T_H - \mu_H T_H, \tag{5}$$

$$F'_H = \gamma_H F_H T_H - (\mu_H + \delta_H) F_H, \tag{6}$$

$$S'_V = \Pi_V - \lambda_V S_V I_H - \mu_V S_V, \tag{7}$$

$$E'_V = \lambda_V S_V I_H - \sigma_V E_V I_V - \mu_V E_V, \tag{8}$$

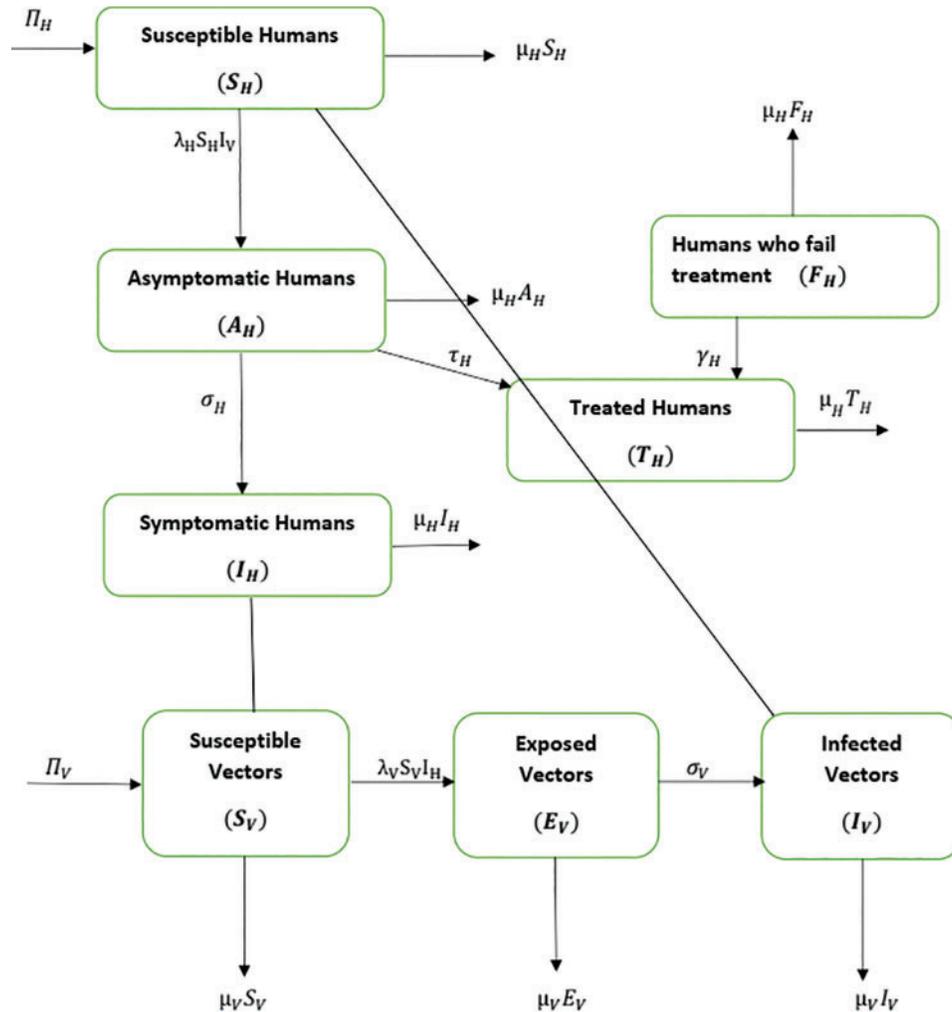


Figure 1: Flow of material from the vector population to the human population

$$I'_V = \sigma_V E_V I_V - \mu_V I_V. \tag{9}$$

where $N_H(t) = S_H(t) + A_H(t) + I_H(t) + T_H(t) + F_H(t).$ (10)

$$N_V(t) = S_V(t) + E_V(t) + I_V(t). \tag{11}$$

therefore, $\frac{dN_H}{dt} = 0, \frac{dN_V}{dt} = 0.$

The stabilized form of model (2)–(9) is as follows:

$$S'_H = \Pi_H - \lambda_H S_H I_V - \mu_H S_H, \tag{12}$$

$$A'_H = \lambda_H S_H I_V - \sigma_H A_H I_H - \mu_H A_H, \tag{13}$$

$$I'_H = \sigma_H A_H I_H - \mu_H I_H, \tag{14}$$

$$S'_V = \Pi_V - \lambda_V S_V I_H - \mu_V S_V, \tag{15}$$

$$E'_V = \lambda_V S_V I_H - \sigma_V E_V I_V - \mu_V E_V, \tag{16}$$

$$I'_V = \sigma_V E_V I_V - \mu_V I_V. \tag{17}$$

There are two regions for the system (12)–(17) like humans population and vector population are $\Gamma_1 = \left\{ (S_H, A_H, I_H) \in R_+^3 : S_H + A_H + I_H \leq \frac{\Pi_H}{\mu_H}, S_H \geq 0, A_H \geq 0, I_H \geq 0 \right\}$ and $\Gamma_2 = \left\{ (S_V, E_V, I_V) \in R_+^3 : S_V + E_V + I_V \leq \frac{\Pi_V}{\mu_V}, S_V \geq 0, E_V \geq 0, I_V \geq 0 \right\}$ respectively. These given regions are bounded and closed. The solution of system (12)–(17) lie in the same regions. So, these given regions are also called the non-negative invariant region.

3.1 Equilibria of the Material

The equilibria of system (12)–(17) can be classified into two ways under as Disease-free equilibrium is

$$D_1 = \left(\frac{\Pi_H}{\mu_H}, 0, 0, \frac{\Pi_V}{\mu_V}, 0, 0 \right).$$

Endemic equilibrium is

$$E_1 = (S_H, A_H, I_H, S_V, E_V, I_V)$$

$$S_H = \frac{\Pi_H \sigma_H - \sigma_H I_H - (\mu_H)^2}{\mu_H \sigma_H}, \quad A_H = \frac{\mu_H}{\sigma_H}, \quad I_H = \frac{\lambda_H S_H I_V - \mu_H^2 A_H}{\sigma_H A_H}, \quad E_V = \frac{\mu_V}{\sigma_V},$$

$$S_V = \frac{\Pi_V \sigma_V - \sigma_V I_V - (\mu_V)^2}{\mu_V \sigma_V}, \quad I_V = \frac{\lambda_V S_V I_H - \mu_V^2 A_V}{\sigma_V A_V}$$

$$\text{and } R_2 = \rho \left(F_2 V_2^{-1} \right) = \sqrt{\frac{\lambda_V (\mu_H + \sigma_H) (\mu_V + \sigma_V)}{\Pi_H \mu_V^2 T_1 T_2}}$$

where $T_1 = (\sigma_H + \mu_H), T_2 = (\sigma_V + \mu_V).$ Note that R_2 is the threshold number of the model (12)–(17).

4 Stochastic Material

The construction of a stochastic material of the model from the deterministic model has presented in [21]. For this, we shall substitute in the model (12)–(17) as follows:

Put $\lambda_H dt = \lambda_H dt + \sigma dB$ and $\lambda_V dt = \lambda_V dt + \sigma_1 dB$.

$$dS_H = (\Pi_H - \lambda_H S_H I_V - \mu_H S_H) dt - \sigma S_H I_V dB, \quad (18)$$

$$dA_H = (\lambda_H S_H I_V - \sigma_H A_H I_H - \mu_H A_H) dt + \sigma S_H I_V dB, \quad (19)$$

$$dI_H = (\sigma_H A_H I_H - \mu_H I_H) dt, \quad (20)$$

$$dS_V = (\Pi_V - \lambda_V S_V I_H - \mu_V S_V) dt - \sigma_1 S_V I_H dB, \quad (21)$$

$$dE_V = (\lambda_V S_V I_H - \sigma_V E_V I_V - \mu_V E_V) dt + \sigma_1 S_V I_H dB, \quad (22)$$

$$dI_V = (\sigma_V E_V I_V - \mu_V I_V) dt. \quad (23)$$

where “B” is the geometric Brownian motion.

4.1 Stochastic Euler Method

This scheme is constructed for the model (18)–(23) as follows:

$$S_H^{n+1} = S_H^n + h (\Pi_H - \lambda_H S_H^n I_V^n - \mu_H S_H^n - \sigma S_H^n I_V^n \Delta B_n), \quad (24)$$

$$A_H^{n+1} = A_H^n + h (\lambda_H S_H^n I_V^n - \sigma_H A_H^n I_H^n - \mu_H A_H^n + \sigma S_H^n I_V^n \Delta B_n), \quad (25)$$

$$I_H^{n+1} = I_H^n + h (\sigma_H A_H^n I_H^n - \mu_H I_H^n), \quad (26)$$

$$S_V^{n+1} = S_V^n + h (\Pi_V - \lambda_V S_V^n I_H^n - \mu_V S_V^n - \sigma_1 S_V^n I_H^n \Delta B_n), \quad (27)$$

$$E_V^{n+1} = E_V^n + h (\lambda_V S_V^n I_H^n - \sigma_V E_V^n I_V^n - \mu_V E_V^n + \sigma_1 S_V^n I_H^n \Delta B_n), \quad (28)$$

$$I_V^{n+1} = I_V^n + h (\sigma_V E_V^n I_V^n - \mu_V I_V^n). \quad (29)$$

where ‘h’ is the time parameter. The solution of the deterministic model for the DFE, i.e., $D_1 = \left(\frac{\Pi_H}{\mu_H}, 0, 0, \frac{\Pi_V}{\mu_V}, 0, 0 \right)$ and the reproduction number $R_2 = 0.8944 < 1$ means help us these procedures to switch the dengue virus. The EE, i.e., $E_1 = (0.07339, 0.625, 0.3016, 0.08108, 0.5556, 0.3640)$ and the reproduction number $R_2 = 6.3875 > 1$ means dengue is endemic. We are simulating the solution of this technique by using parameters values prearranged in [20] and see Tab. 1.

4.2 Stochastic Runge–Kutta Method

The pseudo-code for stochastic Runge Kutta for the system (18)–(23) is as follows:

Begin:

Declare all constants

Set the step size ‘h’

Declare arrays for $S_H, A_H, I_H, S_V, E_V, I_V$. The arrays should be able to store 2000 values.

Put initial values for $S_H, A_H, I_H, S_V, E_V, I_V$ at index 1 of the corresponding arrays.

Index = 2

For t from 0.1 till $t < 200$

Calculate stage 1 equations

Calculate stage 2 equations

Calculate stage 3 equations

```

Calculate stage 4 equations
Calculate final stage equations
Index = Index + 1
t = t + 0.1
End For
Plot required data
end program
    
```

Table 1: Rates of the material of the model

Rates	DFE	EE
μ_H	0.5	0.5
λ_H	0.1	10.1
μ_V	0.5	0.5
λ_V	0.2	10.2
σ_V	0.9	0.9
σ_H	0.8	0.8
Π_V	0.5	0.5
Π_H	0.5	0.5
σ_1	0.1	0.1
σ	0.5	0.5

We make the simulation of discussed technique by using parameters values prearranged in [20] and see [Tab. 1](#).

Table 2: Covariance coefficient

Sub-populations	Correlation coefficient (ρ)	Relationship
(S_H, I_H)	-0.3595	Inverse
(I_H, A_H)	-0.7083	Inverse
(S_H, A_H)	-0.3930	Inverse
(S_V, I_V)	-0.2906	Inverse
(I_V, E_V)	-0.8331	Inverse
(S_V, E_V)	-0.2824	Inverse

4.3 Stochastic NSFD Method

This scheme is constructed for the model (18)–(23) as follows:

$$S_H^{n+1} = (S_H^n + h\Pi_H)/(1 + h(\lambda_H I_V^n + \mu_H + \sigma I_V^n \Delta B_n)), \tag{30}$$

$$A_H^{n+1} = (A_H^n + h\lambda_H S_H^n I_V^n + h\sigma S_H^n I_V^n \Delta B_n)/(1 + h\sigma_H I_H^n + h\mu_H), \tag{31}$$

$$I_H^{n+1} = (I_H^n + h\sigma_H A_H^n I_H^n)/(1 + h\mu_H), \tag{32}$$

$$S_V^{n+1} = (S_V^n + h\Pi_V)/(1 + h(\lambda_V I_H^n + \mu_V + \sigma_1 I_H^n \Delta B_n)), \tag{33}$$

$$E_V^{n+1} = (E_V^n + h\lambda_V S_V^n I_H^n + h\sigma_1 S_V^n I_H^n \Delta B_n) / (1 + h(\sigma_V I_V^n + \mu_V)), \tag{34}$$

$$I_V^{n+1} = (I_V^n + h\sigma_V E_V^n I_V^n) / (1 + h\mu_V). \tag{35}$$

We make the simulation of the above-discussed technique by using parameters values prearranged in [20] and see Tab. 1.

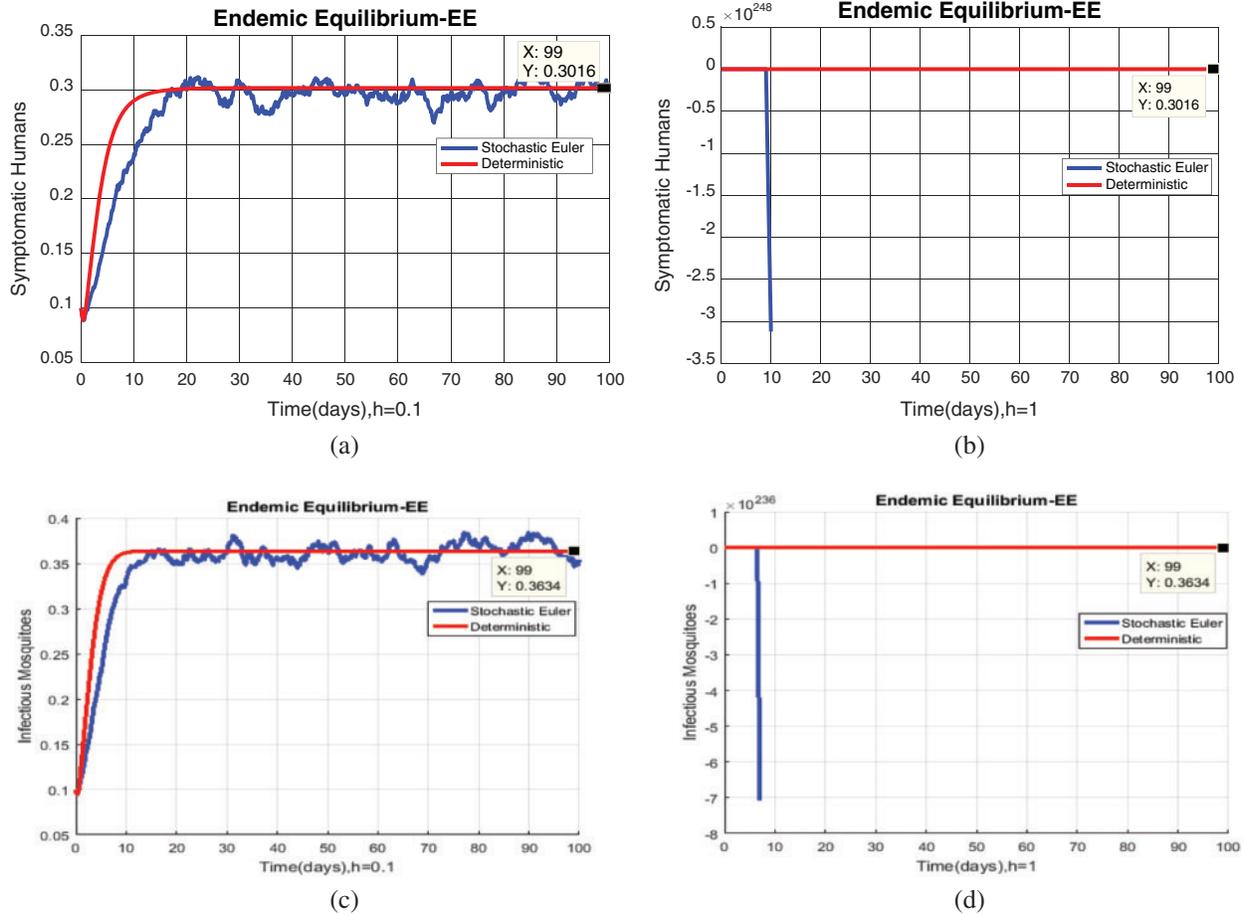


Figure 2: (a) Symptomatic humans at $h = 0.1$ (b) symptomatic humans at $h = 1$ (c) infected mosquitoes' section at $h = 0.1$ (d) infected mosquitoes' section at $h = 1$

4.4 Convergence Analysis

For this, we shall satisfy the following theorems as follows:

Theorem 1: Forgiven initial values $(S_H^0, A_H^0, I_H^0) \in R_+^3$ and $(S_V^0, E_V^0, I_V^0) \in R_+^3$ for the system (30)–(35) has a unique positive solutions $(S_H^n, A_H^n, I_H^n) \in R_+^3$ and $(S_V^n, E_V^n, I_V^n) \in R_+^3$, respectively for all $n \geq 0$.

Proof: Since all rates and state variables of the system are non-negative. So, the proof is straightforward.

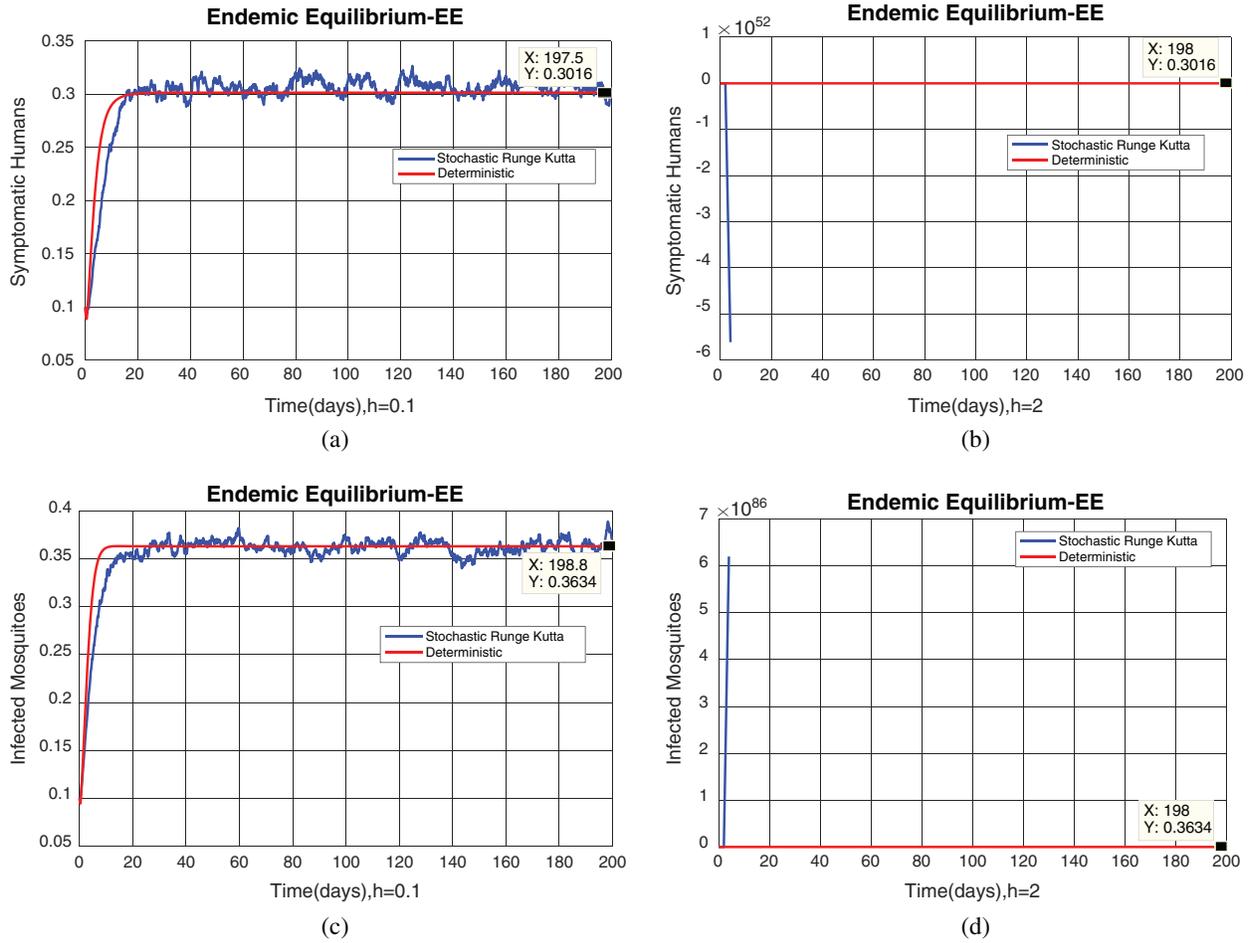


Figure 3: (a) Symptomatic humans section at $h = 0.1$ (b) symptomatic humans section at $h = 2$ (c) infected mosquitoes section at $h = 0.1$ (d) infected mosquitoes section at $h = 2$

Theorem 2: These regions $\Gamma_1 = \left\{ (S_H, A_H, I_H) \in R_+^3 : S_H^n + A_H^n + I_H^n \leq \frac{\Pi_H}{\mu_H}, S_H \geq 0, A_H \geq 0, I_H \geq 0 \right\}$ and $\Gamma_2 = \left\{ (S_V, E_V, I_V) \in R_+^3 : S_V^n + E_V^n + I_V^n \leq \frac{\Pi_V}{\mu_V}, S_V \geq 0, E_V \geq 0, I_V \geq 0 \right\}$ for all $n \geq 0$ are the positive invariant set for the system (31)–(35).

Proof: Now, we rewrite the system (30)–(32) as follows:

$$\frac{S_H^{n+1} - S_H^n}{h} = \Pi_H - \lambda_H S_H^n I_V^n - \mu_H S_H^n - \sigma S_H^n I_V^n \Delta B_n.$$

$$\frac{A_H^{n+1} - A_H^n}{h} = \lambda_H S_H^n I_V^n - \sigma_H A_H^n I_H^n - \mu_H A_H^n + \sigma S_H^n I_V^n \Delta B_n.$$

$$\frac{I_H^{n+1} - I_H^n}{h} = \sigma_H A_H^n I_H^n - \mu_H I_H^n.$$

So, $S_H^{n+1} + A_H^{n+1} + I_H^{n+1} \leq \frac{\Pi_H}{\mu_H}$.

Also, we rewrite the system (33)–(35) as follows:

$$\frac{S_V^{n+1} - S_V^n}{h} = \Pi_V - \lambda_V S_V^n I_H^n - \mu_V S_V^n - \sigma S_V^n I_H^n \Delta B_n.$$

$$\frac{E_V^{n+1} - E_V^n}{h} = \lambda_V S_V^n I_H^n - \sigma_V E_V^n I_V^n - \mu_V E_V^n + \sigma S_V^n I_H^n \Delta B_n.$$

$$\frac{I_V^{n+1} - I_V^n}{h} = \sigma_V E_V^n I_V^n - \mu_V I_V^n.$$

More precisely,

$$Ev(n + 1) + S_V^{n+1} + I_V^{n+1} \leq \frac{\Pi_V}{\mu_V}.$$

Theorem 3: For given $n \geq 0$, the eigenvalue of the system (30)–(35) lies in the semi-circle.

Proof: Considering the functions F, G, H, I, J and K from the system (30)–(35) as follows:

$$F = \frac{S_H + h\Pi_H}{1 + h\lambda_H I_V + h\mu_H + h\sigma I_V \Delta B_n}, \quad G = \frac{A_H + h\lambda_H S_H I_V + h\sigma S_H I_V \Delta B_n}{1 + h(\sigma_H I_H + \mu_H)}, \quad H = \frac{I_H + h\sigma_H A_H I_H}{1 + h\mu_H},$$

$$I = \frac{S_V + h\Pi_V}{1 + h(\lambda_V I_H + \mu_V + \sigma_1 I_H \Delta B_n)}, \quad J = \frac{E_V + h\lambda_V S_V I_H + h\sigma_1 S_V I_H \Delta B_n}{1 + h(\sigma_V I_V + \mu_V)} \quad \text{and}$$

$$K = \frac{I_V + h\sigma_V E_V I_V}{1 + h\mu_V}$$

The Jacobi matrix J as

$$J = \begin{bmatrix} \frac{\partial F}{\partial S_H} & \frac{\partial F}{\partial A_H} & \frac{\partial F}{\partial I_H} & \frac{\partial F}{\partial S_V} & \frac{\partial F}{\partial E_V} & \frac{\partial F}{\partial I_V} \\ \frac{\partial G}{\partial S_H} & \frac{\partial G}{\partial A_H} & \frac{\partial G}{\partial I_H} & \frac{\partial G}{\partial S_V} & \frac{\partial G}{\partial E_V} & \frac{\partial G}{\partial I_V} \\ \frac{\partial H}{\partial S_H} & \frac{\partial H}{\partial A_H} & \frac{\partial H}{\partial I_H} & \frac{\partial H}{\partial S_V} & \frac{\partial H}{\partial E_V} & \frac{\partial H}{\partial I_V} \\ \frac{\partial I}{\partial S_H} & \frac{\partial I}{\partial A_H} & \frac{\partial I}{\partial I_H} & \frac{\partial I}{\partial S_V} & \frac{\partial I}{\partial E_V} & \frac{\partial I}{\partial I_V} \\ \frac{\partial J}{\partial S_H} & \frac{\partial J}{\partial A_H} & \frac{\partial J}{\partial I_H} & \frac{\partial J}{\partial S_V} & \frac{\partial J}{\partial E_V} & \frac{\partial J}{\partial I_V} \\ \frac{\partial K}{\partial S_H} & \frac{\partial K}{\partial A_H} & \frac{\partial K}{\partial I_H} & \frac{\partial K}{\partial S_V} & \frac{\partial K}{\partial E_V} & \frac{\partial K}{\partial I_V} \end{bmatrix}$$

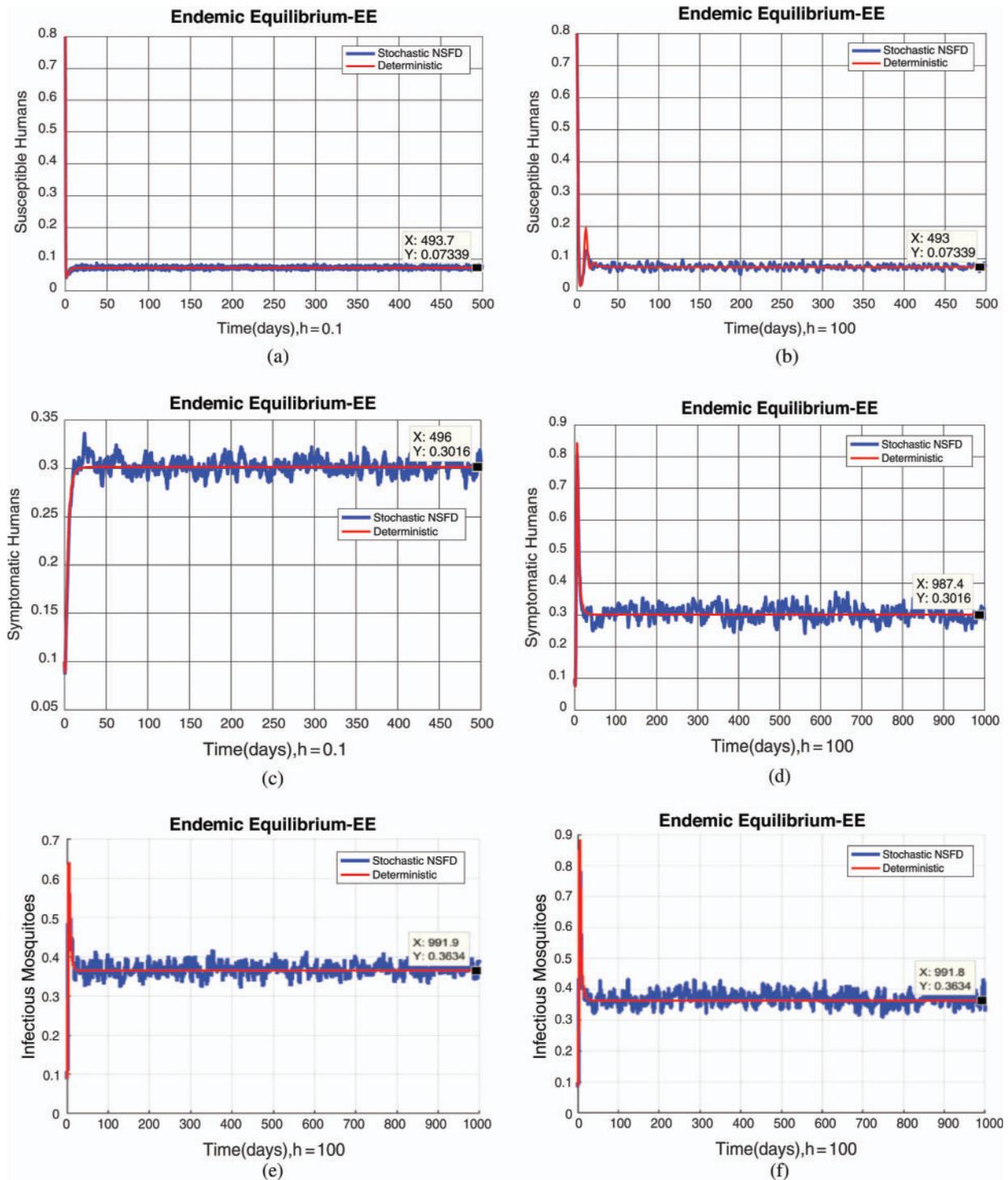


Figure 4: (a) Susceptible humans at $h = 0.1$ (b) susceptible humans at $h = 100$ (c) symptomatic humans at $h = 0.1$ (d) symptomatic humans at $h = 100$ (e) infected mosquitoes at $h = 0.1$ (f) infected mosquitoes at $h = 100$

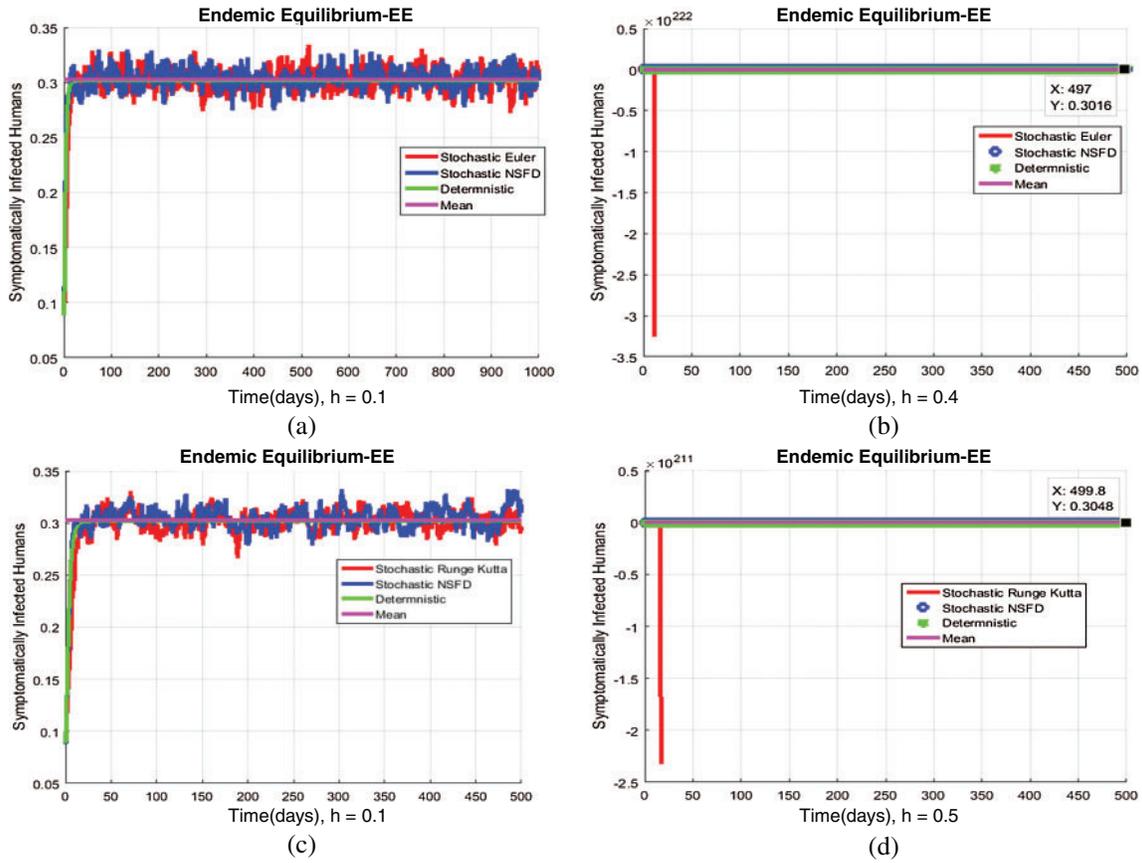


Figure 5: (a) Symptomatic infected individuals' section at $h = 0.1$ (b) symptomatic infected individuals' section at $h = 0.4$ (c) symptomatic infected individuals' section at $h = 0.1$ (d) symptomatic infected individuals' section at $h = 0.5$

By simplify, we get the following eigenvalues as follows:

$$\lambda_1 = \frac{1}{1 + h\mu_H} < 1, \quad \lambda_2 = \frac{\lambda_H + \sigma \Delta B_n}{\mu_H} < 1, \quad \text{if } R_2 < 1, \quad \lambda_3 = \frac{\lambda_H}{\mu_H} < 1, \quad \lambda_4 = \frac{1}{1 + h\mu_V} < 1,$$

$$\lambda_5 = \frac{(\lambda_V + \sigma_1 \Delta B_n)}{1 + h\mu_V} < 1 \quad \text{if } R_2 < 1, \lambda_6 = \frac{\lambda_V}{\mu_V} < 1.$$

Thus, the system is stable at the rates of the material of the model.

4.5 Comparison Section

In this part, we are going to discuss the comparison of existing stochastic techniques and proposed technique as follows:

4.6 Covariance of the Model

The correlation coefficient has obtained among the compartments of the model, and results are described in [Tab. 2](#).

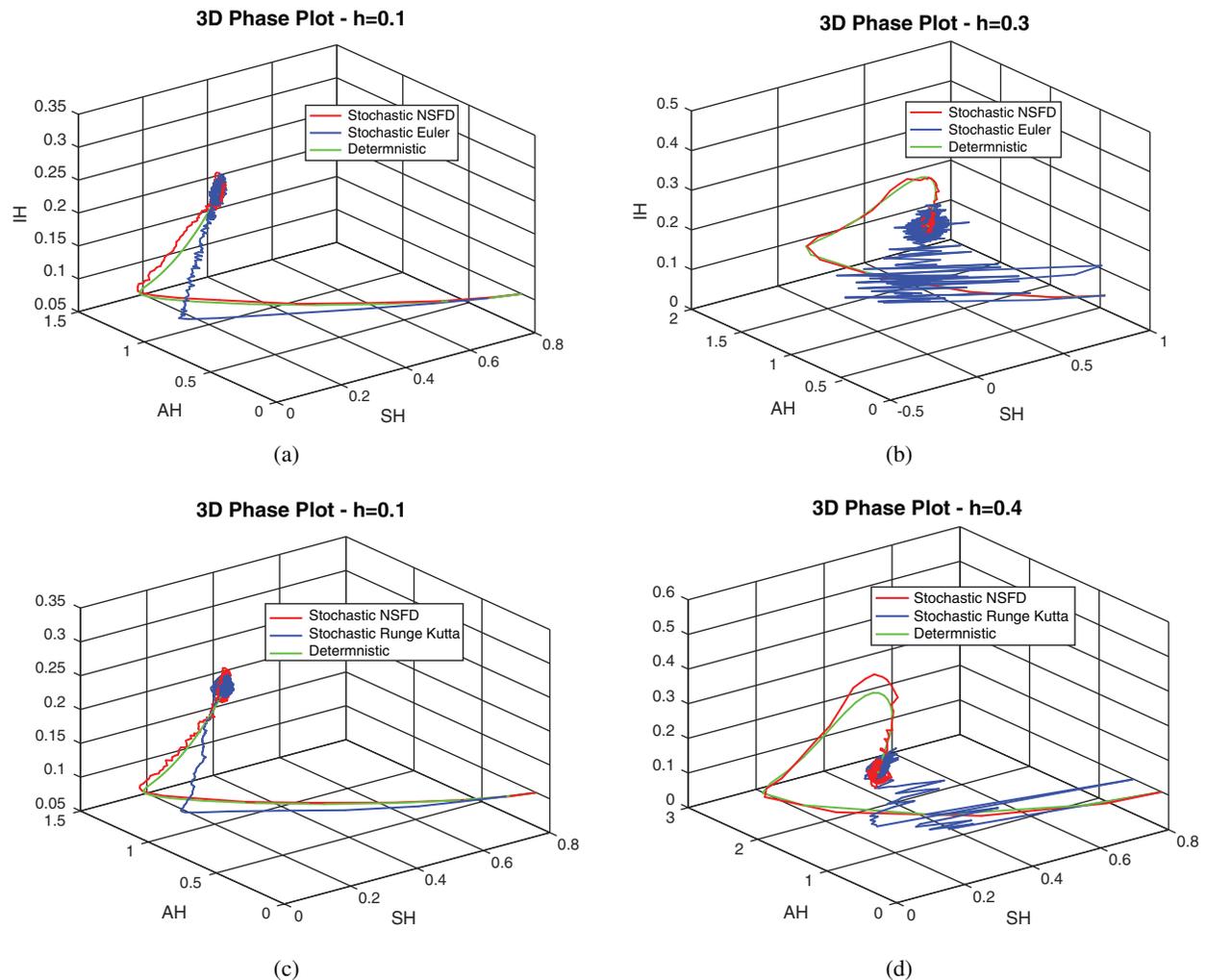


Figure 6: 3D phase plots using stochastic NSFD scheme in comparison with stochastic Euler and stochastic Runge–Kutta methods

Tab. 2 exhibits that susceptible humans have an inverse relation with other components of the model. It is concluded that the decreases in the other components of the model prove the disease-free state for both populations of the model.

5 Results and Discussion

Fig. 2, exhibits the solution of the stochastic Euler method for different discretization values of the parameters, and lose the essential features for $h = 1$, as desired. In Fig. 3, we can observe that the stochastic Runge–Kutta method meets equilibria for step size $h = 0.1$ and lose the essential features. In Fig. 4, we can observe that the stochastic NSFD technique meets for both equilibria for any value of the parameters. In a stochastic context, we have concluded that this method preserves all the essential features presented in [22]. In Fig. 5, we have presented a comparative analysis of both types of modelling. In Fig. 6, the 3D phase plots have presented with the comparison of the computational methods.

6 Conclusion and Future Directions

In this study, we must claim the most effective and real stochastic analysis in comparison with the deterministic analysis of the model. Also, the construction of the stochastic model has presented with the rates of the material of the model. Unfortunately, numerical research is presented in all disciplines of science because of the non-differentiability of Brown's motion. The standard stochastic methods employed here were the usual stochastic Euler method and the fourth-order stochastic Runge–Kutta method. Numerical properties of those methods are well known like positivity, boundedness and dynamical consistency. Various simulations were produced using different step sizes, and comparisons were shown graphically. The results showed that the methodology proposed in this work is capable of preserving the essential features of the relevant solutions of the mathematical model, as expected. On the other hand, the graphical results showed that the standard methods were not able to guarantee the preservation of some of those essential features. In particular, we proved computationally that those standard stochastic methods are incapable of preserving the essential features of the model, as desired. In that sense, the stochastic NSFD method designed in this work is a more reliable technique. Moreover, we shall extend our work in all discipline as bio-economics, biophysics, biochemistry, and many more sub-branches of material sciences. Also, the given essential features preserving analysis could be extended in the fractional-order models [23,24].

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