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A reliable numerical analysis for stochastic dengue epidemic model with incubation period of virus

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Abstract

This article represents a numerical analysis for a stochastic dengue epidemic model with incubation period of virus. We discuss the comparison of solutions between the stochastic dengue model and a deterministic dengue model. In this paper, we have shown that the stochastic dengue epidemic model is more realistic as compared to the deterministic dengue epidemic model. The effect of threshold number R_1 holds in the stochastic dengue epidemic model. If $R_1 < 1$, then situation helps us to control the disease while $R_1 > 1$ shows the persistence of disease in population.

Unfortunately, the numerical methods like Euler–Maruyama, stochastic Euler, and stochastic Runge–Kutta do not work for large time step sizes. The proposed framework of stochastic nonstandard finite difference scheme (SNSFD) is independent of step size and preserves all the dynamical properties like positivity, boundedness, and dynamical consistency.

Keywords: Dengue virus; Stochastic differential equations; Euler–Maruyama method; Stochastic Euler method; Stochastic Runge–Kutta method; Stochastic SNSFD method

1 Introduction

Dengue is the most common infection caused by virus transmitted by mosquitos. Dengue is incurable disease. The transmission of dengue virus exists in a cycle. An infected human is bitten by a mosquito which bites another human and the cycle continues. Dengue is found mostly during and after the rainy season in the tropical and sub-tropical areas of the world [1].

In the middle of the twentieth century the disease was restricted geographically. But during the Second World War the eggs of *Aedes aegypti* were transported by cargo, which played a crucial role in the spreading of the virus over a larger area. Before this, the disease was considered to be relatively small. During the epidemics in Philippines and Thailand, the disease was documented for the first time properly. It is found in more than 100 countries of Asia, America, and Africa. According to a survey of WHO, annually 50 to 100 million infections occur in the world [2]. Dengue fever spreads through a mosquito called *Aedes aegypti*, and it is a viral infection. The tropical and subtropical countries are the major host of this viral infection. Pakistan is also one of them. Dengue virus has four types [3]. A person who has recovered from one type of dengue virus would gain immu-

nity from the same type for the rest of his life, but he is not protected from the other three types of the virus. World Health Organization disclosed that 50 million dengue infections are present in the world and 2.5 billion are at the risk of getting infection from this virus.

Dengue fever has also ability to convert into dengue hemorrhagic fever (DHF) or dengue shock syndrome (DSS). In DHF the patient suffers from bleeding, low level of blood platelets, and also from plasma leakage; whereas in DSS the blood pressure of the patient drops dangerously. No medicine or vaccine is still discovered to treat dengue fever. The only way to control the disease is to control the source of spreading disease which is *Aedes aegypti* mosquito. At the beginning this mosquito breeds in the water storage containers, discarded water containers, used automobile tires, and other items which could store rainwater. The only way to control the reproduction of mosquito is proper disposal of rainwater and proper water storage practices [4].

Aedes mosquitoes are said to be a predominant cause of transmission of dengue virus. It has been observed that these mosquitoes commonly reside in the regions with latitudes between 35° north and 35° south and an elevation below 1000 meters (3300 ft). *Aedes* mosquitoes bite their prey approximately two times, i.e., early in the morning and in the evening. However, there is a possibility that they can bite during other times of the day, and hence infection may spread. World Health Organization (WHO) [2] reports relate that nearly 390 million victims of dengue fever do exist over the world, whereas 96 million call for medical treatment. Every year about 500,000 cases of hemorrhagic fever and the most severe type of dengue disease requires hospitalization. Around 40% of the world's population resides in dengue endemic areas. According to the WHO estimates, approximately 22,000 persons die every year including a high number of children.

Dengue fever is an old disease. It was found in Chinese region in 610 AD and again in 922 AD. The dengue virus multiplied in the eighteenth and nineteenth centuries in many big cities of the world. Dengue plague extended after WW2 (World War Two). It was first isolated in Japan in 1943. In 1944, the virus was isolated at many parts of the world including Indian city Calcutta. Its severe form occurred for the first time in Manila in 1953 to 1954. Dengue expanded in Asia geographically from southeast Asian countries west to India, Sri Lanka, Maldives, and Pakistan. This horrible disease attacked USA in 1980. Similarly, African area was unknown to this disease before 1980. In 1997 dengue virus and *Aedes aegypti* had a worldwide distribution in tropical and subtropical regions.

Malaysia was also reported to be its victim from 2000 to 2012. A list of 237 identified relevant cases and 28 data sources were found satisfying for the inclusion criteria. A survey was reported between 2000 and 2011 in Thailand to describe the dengue disease dynamics. 610 relevant searches were identified, 40 of which fulfilled the inclusion criteria. The number of dengue cases increased during 2001, 2002, 2008, and 2010. These cases were more fatal in young children. In a survey reported between 2000 and 2010 in Brazil, 51 out of 714 identified relevant searches fulfilled the insertion criteria. In Brazil the epidemiological design of dengue fever disease is complex due to influence of multiple socio-environmental factors. This work studies a model with incubation periods of dengue virus in human and vector populations. A susceptible person bitten by an infectious vector cannot transfer dengue virus instantly. Such an individual is called an infected human during this period. Intrinsic incubation period of dengue virus in human is about five days [5].

When an infectious person is bitten by the susceptible vector, then prior to its infectious state it will be an infected vector. On the other hand, the extrinsic incubation period of dengue virus in vector population is about ten days [5]. An uninfected person with no immunity to resist the dengue virus is called a susceptible person. A victim of dengue infection who reacquires their immunity is named a recovered person.

Mathematical modelling has emerged as an effective tool to extract comprehensive insight about epidemic diseases. The formation of the model and the possible simulations allow for analyzing the sensitivity and comparison of conjuncture paradigms. As a result, the prediction of mediator, host, and ecological factor affecting public health is possible, and health policy makers can scientifically suggest and implement health services [2]. Several studies have been conducted on various models of dengue transmission dynamics [6]. It is well known that nonlinear initial value problems (IVPs) do not always possess analytical solutions. The available classical explicit finite-difference schemes, such as Runge–Kutta and Euler methods, can bring about misleading chaos and deceitful oscillations for certain intensities of the discretization parameters [7–11]. Due to these reasons and some other scheme-dependent numerical instabilities such methods prove to be less privileged options.

In general, the determination of stochastic differential equations (SDEs) is either difficult, or these stochastic differential equations (SDEs) do not have the explicit solutions. We use different numerical schemes to integrate these equations [12–15]. A natural question on numerical schemes can be the following despite the convergence analysis: Do the numerical schemes preserve the dynamical properties of the initial system [16]?

In the deterministic modeling we have observed that the usual numerical schemes like Euler and Runge–Kutta do not preserve dynamical properties. Even in the stochastic case the Euler–Maruyama, stochastic Euler, and atochastic Runge–Kutta do not preserve the dynamical properties. This is the question: Can we construct a stochastic numerical scheme which preserves all the dynamical properties?

The main theme of this paper is to introduce the notion of stochastic nonstandard finite difference scheme (SNSFD) based on the rules introduced in the deterministic case by Mickens [17–19].

The strategy of this paper is as follows:

In Sect. 2, we remind classical definitions and some history of stochastic differential equations (SDEs). In Sect. 3, we explain the formulation of stochastic epidemic models. In Sect. 4, we discuss the deterministic dengue model and equilibrium points. In Sect. 5, we explore the stochastic dengue model. In Sect. 6, we introduce different stochastic numerical schemes and compare their result with deterministic solutions. In Sect. 7, we draw the conclusion and give the future directions.

2 Preliminaries

Einstein is considered the pioneer of the idea of stochastic differential equations (SDEs) as he gave a mathematical assembly between microscopic random motion of particles and the macroscopic diffusion equation in 1905 [20]. Nowadays the SDEs attract a lot of attention due to physical developments in real life systems because the ordinary differential equations (ODEs) did not apprehend random forcing and stochastic inputs. A stochas-

tic calculus delivers a mathematical substance for the conduct of stochastic differential equations (SDEs). In general form, we can write the stochastic differential equation that comprises parameters, a continuous time t and variable X_t , as follows:

$$dX_t = u(t, X_t) dt + v(t, X_t) dB(t) \quad (1)$$

and the integral form is

$$X(t) = c + \int_{t_0}^t u(s, X_s) ds + \int_{t_0}^t v(s, X_s) dB_s. \quad (2)$$

The above stochastic differential equation is also called Ito's stochastic differential equation (SDE) where $u(t, X_t)$ and $v(t, X_t)$ are drift and diffusion coefficients, respectively. The random variable c is called the initial value at instant t_0 . A solution X_t of Eq. (1) or (2) is called stochastic process.

2.1 Brownian motion

The Brownian motion can be defined as a continuous time random walk with the following properties [21]:

- (i) $B_0 = 0$.
- (ii) B_t must be continuous, the event happens with probability one. The sample trajectories $t \rightarrow B_t$ are continuous with probability one.
- (iii) For any finite sequence of times $t_1 < t_2 < t_3 < \dots < t_n$, the following paths $B_{t_1} - B_{t_0}$, $B_{t_2} - B_{t_1}$, $B_{t_3} - B_{t_2}$, $\dots$, $B_{t_n} - B_{t_{n-1}}$ are independent.
- (iv) For any times $0 \leq s \leq t$, $B_t - B_s$ is normally distributed with mean zero and variance is $t - s$. In particular, we say that

$$\text{Expectation}[B_t - B_s] = 0 \quad \text{and} \quad \text{Variance}[B_t - B_s] = t - s.$$

The stochastic processes are a fundamental example of Brownian motion. A stochastic epidemic model is articulated in tenure of stochastic modeling process. A stochastic modeling process is an assemblage of random variables $\{Y_t(s)/t \in T, s \in S\}$, where T is the guide set and S is a joint sample space. The guide set often characterizes time such as $T = \{0, 1, 2, \dots\}$ or $T = [0, \infty)$. Time may be discrete or continuous. The homework of stochastic modeling procedures is founded on probability theory. The stochastic epidemic modeling processes can be described in three ways: DTMC (discrete time Markov chain) epidemic models, CTMC (continuous time Markov chain) epidemic models, and SDEs (stochastic differential equations) epidemic models. In discrete time Markov chain (DTMC) epidemic models, we assumed that time and the state variables are discrete. In continuous time Markov chain (CTMC) epidemic models, the time is continuous and the state variables are discrete. In stochastic differential equations (SDEs) epidemic models, we presumed that both the time and the state variables are continuous. Now we discuss stochastic differential equations (SDEs) of epidemic models. They were first introduced and developed by Itô (1942). Stochastic processes evolved almost in all sciences such as economics, mathematics, physics, chemistry, and biology. The solutions of stochastic differential equations (SDEs) are not given explicitly due to their non-differentiable character

of realization of the Brownian motion. So, the stochastic numerical approximation is used to study the properties of stochastic epidemic models [22].

3 Formulation of stochastic epidemic models

Epidemics are generally fashioned by nonlinear systems observed through fragmentary noisy data. This modeling can be divided into two main classes as deterministic epidemic models and stochastic epidemic models. It is interesting to note that after the deterministic system the ordinary differential equations have been articulated for the population dynamics. We can derive several different classes of stochastic epidemic models that consider the irregular nature of the population processes in a persistent manner. The idea here is to apprehend unknown impacts such as changing behaviors, public interventions, seasonal effects, and social cycles. There are various ways to transmit the deterministic epidemic models to stochastic epidemic models. This modeling can be done by discrete time Markov chain (DTMC), continuous time Markov chain (CTMC), and Ito's stochastic differential equations (SDEs). Therefore, the Ito stochastic differential equations give strong arguments for moving away from deterministic epidemic models to stochastic epidemic models. There are various ways to devise stochastic differential equations such as parametric perturbation and non-parametric perturbation methods. In the parametric perturbation method, a parameter from model is chosen and transformed into random variables. In the non-parametric perturbation method, Brownian processes are added to each differential equation (or extra stochasticity parameters are introduced). This type of modeling is more useful as compared to the parametric perturbation method. Another way of the non-parametric perturbation method was introduced by Allen [23–28] in which any extra stochasticity parameters are not introduced in the model. Here we formulate the ways of the non-parametric perturbation method in epidemic models and use different numerical methods to prompt them and check the efficiency of numerical methods on stochastic epidemic models. We observe the relationship between the solutions of deterministic epidemic models and stochastic epidemic models.

4 Deterministic dengue model

In this segment we introduce the deterministic dengue model with incubation period of virus [1]. At any arbitrary time t , the variables are stated as follows: $\overline{S}_h(t)$ represents susceptible humans, $\overline{X}_h(t)$ represents infected humans, $\overline{I}_h(t)$ represents infectious humans, and $\overline{R}_h(t)$ represents recovered humans, whereas $\overline{S}_v(t)$ represents susceptible mosquitos, $\overline{X}_v(t)$ represents infected mosquitos, and $\overline{I}_v(t)$ represents infectious mosquitos. The communication dynamics of dengue model is shown in Fig. 1.

The model parameters are described as follows: N_T (denotes the total human residents size), N_v (denotes the total vector residents size), μ_h (denotes the death rate of human residents), β_h (denotes the infectious proportion from vector to human residents), α_h (denotes the proportion from infected human residents to infectious human residents), r (denotes the immune rate of human residents), β_v (denotes the proportion of dengue virus from human residents to vector residents), μ_v (denotes the death rate of vector residents), α_v (denotes the proportion of infected vector residents to infectious vector residents), and c (denotes the constant recruitment proportion of vector residents).

The governing equations of the dengue model are given below:

$$\left. \begin{aligned} \frac{d\bar{S}_h}{dt} &= \lambda N_T - \beta_h \bar{S}_h \bar{I}_v - \mu_h \bar{S}_h, \\ \frac{d\bar{X}_h}{dt} &= \beta_h \bar{S}_h \bar{I}_v - (\alpha_h + \mu_h) \bar{X}_h, \\ \frac{d\bar{I}_h}{dt} &= \alpha_h \bar{X}_h - (r + \mu_h) \bar{I}_h, \\ \frac{d\bar{R}_h}{dt} &= r \bar{I}_h - \mu_h \bar{R}_h, \\ \frac{d\bar{S}_v}{dt} &= c - \beta_v \bar{I}_h \bar{S}_v - \mu_v \bar{S}_v, \\ \frac{d\bar{X}_v}{dt} &= \beta_v \bar{I}_h \bar{S}_v - (\alpha_v + \mu_v) \bar{X}_v, \\ \frac{d\bar{I}_v}{dt} &= \alpha_v \bar{X}_v - \mu_v \bar{I}_v, \end{aligned} \right\} \quad (3)$$

where the constant size of total human and vector residents respectively is as follows:

$$N_T = \bar{S}_h + \bar{X}_h + \bar{I}_h + \bar{R}_h, \quad (4)$$

$$N_v = \bar{S}_v + \bar{X}_v + \bar{I}_v. \quad (5)$$

Therefore,

$$\frac{dN_T}{dt} = 0 \Rightarrow \lambda = \mu_h \quad \text{and} \quad \frac{dN_v}{dt} = 0 \Rightarrow N_v = \frac{c}{\mu_v}.$$

The normalized form of dengue model (3) is

$$\left. \begin{aligned} \frac{dS}{dt} &= \mu_h - \beta_h S I_v \left(\frac{c}{\mu_v} \right) - \mu_h S, \\ \frac{dX}{dt} &= \beta_h S I_v \left(\frac{c}{\mu_v} \right) - \alpha_h X - \mu_h X, \\ \frac{dI}{dt} &= \alpha_h X - r I - \mu_h I, \\ \frac{dX_v}{dt} &= \beta_v I N_T (1 - X_v - I_v) - \alpha_v X_v - \mu_v X_v, \\ \frac{dI_v}{dt} &= \alpha_v X_v - \mu_v I_v, \end{aligned} \right\} \quad (6)$$

where $S = \frac{\bar{S}_h}{N_T}$, $X = \frac{\bar{X}_h}{N_T}$, $I = \frac{\bar{I}_h}{N_T}$, $X_v = \frac{\bar{X}_v}{N_v}$, $I_v = \frac{\bar{I}_v}{N_v}$ with condition $S + X + I = 1$,

$$S_v + X_v + I_v = 1.$$

4.1 Steady state of the dengue model

The steady state of dengue model (6) can be categorized into two ways of equilibrium points as follows:

Disease-free equilibrium is $D_1 = (1, 0, 0, 0, 0)$.

Endemic equilibrium is $E_1 = (S^o, X^o, I^o, X_v^o, I_v^o)$.

$$S^o = \frac{(\alpha_v + \mu_v)(MN\mu_h^2\mu_v + \alpha_v\gamma_v\mu_h)}{\alpha_h\gamma_v(\mu_h(\alpha_v + \mu_v) + \alpha_v\gamma_h)},$$

$$X^o = \frac{M\mu_h^2\mu_v(\alpha_v + \mu_v)(R_1 - 1)}{\alpha_h\alpha_v(\mu_h(\alpha_v + \mu_v) + \alpha_v\gamma_h)},$$

$$I^o = \frac{\mu_h\mu_v(\alpha_v + \mu_v)(R_1 - 1)}{\alpha_h(\mu_h(\alpha_v + \mu_v) + \alpha_v\gamma_h)},$$

$$X_v^o = \frac{\mu_v(MN\mu_h^3\mu_v)(R_1 - 1)}{\alpha_v\gamma_h(\alpha_h\mu_h\gamma_v + MN\mu_h^2\mu_v)},$$

$$I_v^o = \frac{(MN\mu_h^3\mu_v)(R_1 - 1)}{\gamma_h(\alpha_h\mu_h\gamma_v + MN\mu_h^2\mu_v)},$$

where

$$R_1 = \frac{\alpha_h\alpha_v\gamma_v\gamma_h}{\mu_v(r + \mu_h)(\alpha_v + \mu_v)(\alpha_h + \mu_h)}, \quad M = \frac{r + \mu_h}{\mu_h},$$

$$N = \frac{\alpha_h + \mu_h}{\mu_h}, \quad \gamma_h = \frac{\beta_h C}{\mu_v}, \quad \gamma_v = \beta_v N_T.$$

Note that R_1 is the reproductive number of dengue model (6). The reproductive number has a vital role in disease dynamics. If the reproductive number $R_1 < 1$, then this strategy helps us to control the disease, and if $R_1 > 1$, then the alarming situation of disease in population occurs. If $R_1 = 1$, then the disease will start diffusion in residents.

5 Stochastic dengue model

Let $Y = [S, X, I, X_v, I_v]^T$ to form the stochastic differential equations (SDEs) of model (6). We need to compute the expectations $E^*[\Delta Y]$ and $E^*[\Delta Y \Delta Y^T]$. To find these expectations, the possible changes along with their associated transition probabilities are listed in Table 1.

The expectation and variance of dengue model (6) is defined as

$$E^*[\Delta Y] = \sum_{i=1}^n P_i(\Delta Y)_i,$$

$$\text{Expectation} = E^*[\Delta Y] = \begin{bmatrix} \mu_h - \beta_h SI_v(\frac{c}{\mu_v}) - \mu_h S \\ \beta_h SI_v(\frac{c}{\mu_v}) - \alpha_h X - \mu_h X \\ \alpha_h X - rI - \mu_h I \\ \beta_v IN_T(1 - X_v - I_v) - \alpha_v X_v - \mu_v X_v \\ \alpha_v X_v - \mu_v I_v \end{bmatrix} \Delta t,$$

Table 1 Transition probabilities

Transition	Probabilities
$(\Delta Y)_1 = [1, 0, 0, 0, 0]^T$	$P_1 = \mu_h \Delta t$
$(\Delta Y)_2 = [-1, 1, 0, 0, 0]^T$	$P_2 = \beta_h SI_v \left(\frac{c}{\mu_v} \right) \Delta t$
$(\Delta Y)_3 = [-1, 0, 0, 0, 0]^T$	$P_3 = \mu_h S \Delta t$
$(\Delta Y)_4 = [0, -1, 1, 0, 0]^T$	$P_4 = \alpha_h X \Delta t$
$(\Delta Y)_5 = [0, -1, 0, 0, 0]^T$	$P_5 = \mu_h X \Delta t$
$(\Delta Y)_6 = [0, 0, -1, 0, 0]^T$	$P_6 = (r + \mu_h) \Delta t$
$(\Delta Y)_7 = [0, 0, 0, 1, 0]^T$	$P_7 = \beta_v IN_T \Delta t$
$(\Delta Y)_8 = [0, 0, 0, -1, 0]^T$	$P_8 = \beta_v IN_T X_v \Delta t$
$(\Delta Y)_9 = [0, 0, 0, -1, 0]^T$	$P_9 = \beta_v IN_T I_v \Delta t$
$(\Delta Y)_{10} = [0, 0, 0, -1, 1]^T$	$P_{10} = \alpha_v X_v \Delta t$
$(\Delta Y)_{11} = [0, 0, 0, -1, 0]^T$	$P_{11} = \mu_v X_v \Delta t$
$(\Delta Y)_{12} = [0, 0, 0, 0, -1]^T$	$P_{12} = \mu_v I_v \Delta t$

$$\text{Var} = E^*[\Delta Y \Delta Y^T] = \sum_{i=1}^n P_i [(\Delta Y)_i][(\Delta Y)_i]^T,$$

$$E^*[\Delta Y \Delta Y^T] = \begin{bmatrix} \mu_h + \beta_h SI_v \left(\frac{c}{\mu_v} \right) + \mu_h S & -\beta_h SI_v \left(\frac{c}{\mu_v} \right) & 0 & 0 & 0 & 0 \\ -\beta_h SI_v \left(\frac{c}{\mu_v} \right) & \beta_h SI_v \left(\frac{c}{\mu_v} \right) + \alpha_h X + \mu_h X & -\alpha_h X & 0 & 0 & 0 \\ 0 & -\alpha_h X & rI + \mu_h I & 0 & 0 & 0 \\ 0 & 0 & 0 & \beta_v IN_T (1 + X_v + I_v) + \alpha_v X_v + \mu_v X_v & 0 & 0 \\ 0 & 0 & 0 & 0 & \mu_v X_v & 0 \\ 0 & 0 & 0 & 0 & 0 & \mu_v I_v \end{bmatrix} \Delta t.$$

The stochastic differential equation satisfies the diffusion processes. So,

$$\frac{dY(t)}{dt} = f(Y(t), t) + L(Y(t), t) \frac{dB(t)}{dt}.$$

If we define

$$\text{drift} = f(Y(t), t) = \frac{E^*[\Delta Y]}{\Delta t}$$

and

$$\text{diffusion} = L(Y(t), t) = \sqrt{\frac{E^*[\Delta Y \Delta Y^T]}{\Delta t}},$$

then the stochastic differential equation of dengue model (6) is

$$dY(t) = f(Y(t), t) dt + L(Y(t), t) dB(t) \quad (7)$$

with initial conditions $Y(0) = Y_o = [0.1, 0.0001, 0.0001, 0.001, 0.001]^T$, $0 \leq t \leq T$, and $B(t)$ is the Brownian motion.

5.1 Euler–Maruyama method

Here we use the Euler–Maruyama method [29] to find the numerical solution of SDEs (7) of the dengue model by using the parameter values given in [1] (see Table 2).

We can write the Euler–Maruyama method of SDEs (7) as

$$Y_{n+1} = Y_n + f(Y_n, t) \Delta t + L(Y_n, t) dB(t),$$

Table 2 Parameter values [1]

Parameters	Values (Days)	
	DFE	EE
μ_h	0.0000391	0.0000391
β_h	0.00005	0.00005
α_h	1/5	1/5
r	1/14	1/14
NT	5000	5000
β_v	0.00008	0.00008
μ_v	1/14	1/14
α_v	1/10	1/10
c	3	300
σ_1	0.0004	0.0004
σ_2	0.0003	0.0003
σ_3	0.0002	0.0002
σ_4	0.0001	0.0001
σ_5	0.0001	0.0001

where ' Δt ' is time step size. The solution of SDEs lies in confidence interval for both disease-free equilibrium and endemic equilibrium as shown in Figs. 2 and 3. The solution of deterministic dengue model for the disease-free equilibrium $D_1 = (1, 0, 0, 0, 0)$ and the reproductive number $R_1 = 0.0960 < 1$ means these measures help to control the disease in residents. The endemic equilibrium $E_1 = (0.1042, 0.0001751, 0.00049, 0.001144, 0.001601)$ and the reproductive number $R_1 = 9.5969 > 1$ means disease is endemic in residents. We have observed here that the solutions of SDEs are most probabilistic as compared to the solutions of deterministic ODEs and also the solutions of deterministic ODEs are the mean of solutions of SDEs.

6 Non-parametric perturbation stochastic dengue model

Another way to construct the SDEs from the deterministic ODEs is to introduce the non-parametric perturbation in each differential equation of dengue model (6) as follows:

$$\left. \begin{aligned} dS &= (\mu_h - \beta_h SI_v(\frac{c}{\mu_v}) - \mu_h S) dt + \sigma_1 dB_1(t)S, \\ dX &= (\beta_h SI_v(\frac{c}{\mu_v}) - \alpha_h X - \mu_h X) dt + \sigma_2 dB_2(t)X, \\ dI &= (\alpha_h X - rI - \mu_h I) dt + \sigma_3 dB_3(t)I, \\ dX_v &= (\beta_v IN_T(1 - X_v - I_v) - \alpha_v X_v - \mu_v X_v) dt + \sigma_4 dB_4(t)X_v, \\ dI_v &= (\alpha_v X_v - \mu_v I_v) dt + \sigma_5 dB_5(t)I_v, \end{aligned} \right\} \quad (8)$$

where $\sigma_1, \sigma_2, \sigma_3, \sigma_4$, and σ_5 are stochasticity of each compartment of the dengue model and $B_j(t)$, ($j = 1, 2, 3, 4, 5$) are the independent Brownian motions. The non-parametric perturbation stochastic dengue model does not have the explicit solutions due to non-differentiability term of Brownian motion, so we introduce some new stochastic numerical methods to find the solution of model (8).

6.1 Stochastic Euler method

The stochastic Euler method can be derived from the above non-parametric perturbation stochastic dengue model (8) as follows:

$$\left. \begin{aligned} S^{n+1} &= S^n + h[\mu_h - \beta_h S^n I_v^n(\frac{c}{\mu_v}) - \mu_h S^n + \sigma_1 dB_1(t)S^n], \\ X^{n+1} &= X^n + h[\beta_h S^n I_v^n(\frac{c}{\mu_v}) - \alpha_h X^n - \mu_h X^n + \sigma_2 dB_2(t)X^n], \\ I^{n+1} &= I^n + h[\alpha_h X^n - rI^n - \mu_h I^n + \sigma_3 dB_3(t)I^n], \\ X_v^{n+1} &= X_v^n + h[\beta_v I^n N_T(1 - X_v^n - I_v^n) - \alpha_v X_v^n - \mu_v X_v^n + \sigma_4 dB_4(t)X_v^n], \\ I_v^{n+1} &= I_v^n + h[\alpha_v X_v^n - \mu_v I_v^n + \sigma_5 dB_5(t)I_v^n], \end{aligned} \right\} \quad (9)$$

where h is any time step size, and we simulate the solution of stochastic Euler method by using MATLAB program and parameter values given in [1] (see Table 2).

6.2 Stochastic Runge–Kutta method

The stochastic Runge–Kutta method can be derived from the above non-parametric perturbation stochastic dengue model (8) as follows:

$$\begin{aligned}
 A_1 &= h[\mu_h - \beta_h S^n I_v^n (\frac{c}{\mu_v}) - \mu_h S^n + \sigma_1 dB_1(t) S^n], \\
 B_1 &= h[\beta_h S^n I_v^n (\frac{c}{\mu_v}) - \alpha_h X^n - \mu_h X^n + \sigma_2 dB_2(t) X^n], \\
 C_1 &= h[\alpha_h X^n - r I^n - \mu_h I^n + \sigma_3 dB_3(t) I^n], \\
 D_1 &= h[\beta_v I^n N_T (1 - X_v^n - I_v^n) - \alpha_v X_v^n - \mu_v X_v^n + \sigma_4 dB_4(t) X_v^n], \\
 E_1 &= h[\alpha_v X_v^n - \mu_v I_v^n + \sigma_5 dB_5(t) I_v^n], \\
 A_2 &= h[\mu_h - \beta_h (S^n + \frac{A_1}{2})(I_v^n + \frac{E_1}{2})(\frac{c}{\mu_v}) - \mu_h (S^n + \frac{A_1}{2}) + \sigma_1 dB_1(t) (S^n + \frac{A_1}{2})], \\
 B_2 &= h[\beta_h (S^n + \frac{A_1}{2})(I_v^n + \frac{E_1}{2})(\frac{c}{\mu_v}) - \alpha_h (X^n + \frac{B_1}{2}) \\
 &\quad - \mu_h (X^n + \frac{B_1}{2}) + \sigma_2 dB_2(t) (X^n + \frac{B_1}{2})], \\
 C_2 &= h[\alpha_h (X^n + \frac{B_1}{2}) - r (I^n + \frac{C_1}{2}) - \mu_h (I^n + \frac{C_1}{2}) + \sigma_3 dB_3(t) (I^n + \frac{C_1}{2})], \\
 D_2 &= h[\beta_v (I^n + \frac{C_1}{2}) N_T (1 - (X_v^n + \frac{D_1}{2}) - (I_v^n + \frac{E_1}{2})) - \alpha_v (X_v^n + \frac{D_1}{2}) \\
 &\quad - \mu_v (X_v^n + \frac{D_1}{2}) + \sigma_4 dB_4(t) (X_v^n + \frac{D_1}{2})], \\
 E_2 &= h[\alpha_v (X_v^n + \frac{D_1}{2}) - \mu_v (I_v^n + \frac{E_1}{2}) + \sigma_5 dB_5(t) (I_v^n + \frac{E_1}{2})], \\
 A_3 &= h[\mu_h - \beta_h (S^n + \frac{A_2}{2})(I_v^n + \frac{E_2}{2})(\frac{c}{\mu_v}) - \mu_h (S^n + \frac{A_2}{2}) + \sigma_1 dB_1(t) (S^n + \frac{A_2}{2})], \\
 B_3 &= h[\beta_h (S^n + \frac{A_2}{2})(I_v^n + \frac{E_2}{2})(\frac{c}{\mu_v}) - \alpha_h (X^n + \frac{B_2}{2}) \\
 &\quad - \mu_h (X^n + \frac{B_2}{2}) + \sigma_2 dB_2(t) (X^n + \frac{B_2}{2})], \\
 C_3 &= h[\alpha_h (X^n + \frac{B_2}{2}) - r (I^n + \frac{C_2}{2}) - \mu_h (I^n + \frac{C_2}{2}) + \sigma_3 dB_3(t) (I^n + \frac{C_2}{2})], \\
 D_3 &= h[\beta_v (I^n + \frac{C_2}{2}) N_T (1 - (X_v^n + \frac{D_2}{2}) - (I_v^n + \frac{E_2}{2})) - \alpha_v (X_v^n + \frac{D_2}{2}) \\
 &\quad - \mu_v (X_v^n + \frac{D_2}{2}) + \sigma_4 dB_4(t) (X_v^n + \frac{D_2}{2})], \\
 E_3 &= h[\alpha_v (X_v^n + \frac{D_2}{2}) - \mu_v (I_v^n + \frac{E_2}{2}) + \sigma_5 dB_5(t) (I_v^n + \frac{E_2}{2})], \\
 A_4 &= h[\mu_h - \beta_h (S^n + A_3)(I_v^n + E_3)(\frac{c}{\mu_v}) - \mu_h (S^n + A_3) + \sigma_1 dB_1(t) (S^n + A_3)], \\
 B_4 &= h[\beta_h (S^n + A_3)(I_v^n + E_3)(\frac{c}{\mu_v}) - \alpha_h (X^n + B_3) \\
 &\quad - \mu_h (X^n + B_3) + \sigma_2 dB_2(t) (X^n + B_3)], \\
 C_4 &= h[\alpha_h (X^n + B_3) - r (I^n + C_3) - \mu_h (I^n + C_3) + \sigma_3 dB_3(t) (I^n + C_3)], \\
 D_4 &= h[\beta_v (I^n + C_3) N_T (1 - (X_v^n + D_3) - (I_v^n + E_3)) - \alpha_v (X_v^n + D_3) \\
 &\quad - \mu_v (X_v^n + D_3) + \sigma_4 dB_4(t) (X_v^n + D_3)], \\
 E_4 &= h[\alpha_v (X_v^n + D_3) - \mu_v (I_v^n + E_3) + \sigma_5 dB_5(t) (I_v^n + E_3)], \\
 S^{n+1} &= S^n + \frac{1}{6} [A_1 + 2A_2 + 2A_3 + A_4], \\
 X^{n+1} &= X^n + \frac{1}{6} [B_1 + 2B_2 + 2B_3 + B_4], \\
 I^{n+1} &= I^n + \frac{1}{6} [C_1 + 2C_2 + 2C_3 + C_4], \\
 X_v^{n+1} &= X_v^n + \frac{1}{6} [D_1 + 2D_2 + 2D_3 + D_4], \\
 I_v^{n+1} &= I_v^n + \frac{1}{6} [E_1 + 2E_2 + 2E_3 + E_4],
 \end{aligned} \tag{10}$$

where h is any time step size, and we simulate the solution of stochastic Runge–Kutta method by using MATLAB program and parameter values given in [1] (see Table 2).

6.3 Stochastic NSFD method

The proposed framework of stochastic nonstandard finite difference method (SNSFD) can be derived from the above non-parametric perturbation stochastic dengue model (8) as

follows:

$$\left. \begin{aligned} S^{n+1} &= \frac{S^n + \phi(h)[\mu_h + \sigma_1 dB_1(t)S^n]}{(1 + \phi(h)\beta_h I_v^n(\frac{c}{\mu_v}) + \phi(h)\mu_h)}, \\ X^{n+1} &= \frac{X^n + \phi(h)[\beta_h S^n I_v^n(\frac{c}{\mu_v}) + \sigma_2 dB_2(t)X^n]}{(1 + \phi(h)\alpha_h + \phi(h)\mu_h)}, \\ I^{n+1} &= \frac{I^n + \phi(h)[\alpha_h X^n + \sigma_3 dB_3(t)I^n]}{(1 + \phi(h)r + \phi(h)\mu_h)}, \\ X_v^{n+1} &= \frac{X_v^n + \phi(h)[\beta_v I^n N_T(1 + I_v^n) + \sigma_4 dB_4(t)X_v^n]}{(1 + \phi(h)\beta_v I^n N_T + \phi(h)\alpha_v + h\mu_v)}, \\ I_v^{n+1} &= \frac{I_v^n + \phi(h)[\alpha_v X_v^n + \sigma_5 dB_5(t)I_v^n]}{(1 + \phi(h)\mu_v)}, \end{aligned} \right\} \quad (11)$$

where $\phi(h) = 1 - e^{-h}$ and “ h ” is any time step size. We simulate the solution of the proposed framework of stochastic nonstandard finite difference (SNSFD) method by using MATLAB program and parameter values given in [1] (see Table 2).

7 Results and discussion

In Fig. 2, we observe that the Euler–Maruyama method converges to steady states of the dengue disease model, while deterministic solution is the mean of Euler–Maruyama solution for discretization parameter $h = 1$. When we increase the time step size, we see in Fig. 3 that the Euler–Maryuama method fails to maintain positivity and shows divergence for disease-free equilibrium. Consequently, the Euler–Maryuama method does not work for certain time step sizes. In Figs. 4 and 6, we observe that the stochastic Euler method converges to equilibrium point, while deterministic solution is the mean of stochastic Euler solution for $h = 1$. When we increase the time step size, we see in Figs. 5 and 7 that the stochastic Euler method fails to maintain positivity and consistency for both disease-free and endemic equilibria. So the stochastic Euler method is not a reliable technique to find the solutions of stochastic dengue disease model. In Figs. 8 and 10, we observe that the stochastic Runge–Kutta method converges to disease-free equilibrium and endemic equi-

lbrium, while deterministic solution is the mean of stochastic Runge–Kutta solution for step size $h = 1$. When we increase the time step size, we see in Figs. 9 and 11, the stochastic Runge–Kutta method fails to maintain stability and positivity for both disease-free and endemic equilibrium points. Thus the aforasaid stochastic schemes do not preserve all the dynamical properties [15, 21, 23]. In Figs. 12 to 15, we observe that the stochastic NSFD

method converges for both disease-free and endemic equilibrium points, while deterministic solution is the mean of stochastic NSFD solution for any discretization such as $h = 1$ and $h = 100$. So the stochastic NSFD method preserves all the dynamical properties such as positivity, boundedness, and dynamical consistency defined by Mickens in the stochastic context. The proposed stochastic NSFD scheme is independent of step size and remains convergent for any time step size.

Figure 9 In susceptible human proportion stochastic Runge–Kutta method solution is unbound for disease-free equilibrium for $h = 14$

Figure 10 In infectious human proportion stochastic Runge–Kutta method converges to endemic equilibrium while deterministic solution is the mean of stochastic Runge–Kutta method solution for $h = 1$

8 Conclusion and future framework

The stochastic dengue epidemic model is a more suitable strategy as compared to the deterministic epidemic model to understand the dengue disease dynamics with incubation period of virus. The Euler–Maruyama method, stochastic Euler method, stochastic Runge–Kutta method converge to the true equilibrium points for very small time step

Figure 11 In infectious human proportion stochastic Runge–Kutta method shows failure of dynamical properties for endemic equilibrium for $h = 11$

Figure 12 In susceptible human proportion stochastic-NSFD converges to disease-free equilibrium while deterministic solution is the mean of stochastic NSFD solution for $h = 1$

sizes. After increasing the time step size, these methods diverge and lose the dynamical properties such as positivity, bounded-ness, and dynamical consistency. The proposed framework of stochastic nonstandard finite difference method (SNSFD) of dengue model works for any time step size. The proposed (SNSFD) scheme is suitable for all types of nonlinear and complicated epidemic models. The stochastic solutions are very close with the deterministic solutions. The study of stochastic epidemic models plays the most im-

portant role in disease dynamics. We have observed that stochastic epidemic models are more realistic as compared to deterministic epidemic models. As the future work, the proposed (SNSFD) scheme could be implemented to stochastic delay epidemic models and stochastic diffusion epidemic models. The numerical analysis proposed in this work could also be extended to fractional order dynamical systems [30, 31]. Our future plan also in-

cludes the construction of reliable numerical schemes for the solutions of fractional order stochastic epidemic models for various infectious diseases.

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Abbreviations

CTMC, Continuous Time Markov Chain; DTMC, Discrete Time Markov Chain; DHF, Dengue Hemorrhagic Fever; DSS, Dengue Shock Syndrome; DFE, Disease-Free Equilibrium; EE, Endemic Equilibrium; NSFD, Nonstandard Finite Difference; ODEs, Ordinary Differential Equations; SDE, Stochastic Differential Equation; SNSFD, Stochastic Nonstandard Finite Difference; WHO, World Health Organization; WW2, World War 2.

Competing interests

The authors declare that they have no competing interests.

Authors' contributions

The authors have achieved equal contributions. All authors read and approved the manuscript.

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