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A Reliable Stochastic Numerical Analysis for Typhoid Fever Incorporating With Protection Against Infection

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Abstract: In this paper, a reliable stochastic numerical analysis for typhoid fever incorporating with protection against infection has been considered. We have compared the solutions of stochastic and deterministic typhoid fever model. It has been shown that the stochastic typhoid fever model is more realistic as compared to the deterministic typhoid fever model. The effect of threshold number T^* hold in stochastic typhoid fever model. The proposed framework of the stochastic non-standard finite difference scheme (SNSFD) preserves all dynamical properties like positivity, bounded-ness and dynamical consistency defined by Mickens, R. E. The stochastic numerical simulation of the model showed that increase in protection leads to low disease prevalence in a population.

Keywords: Typhoid fever, stochastic differential equations, euler maruyama scheme, stochastic euler scheme, stochastic runge-kutta scheme, stochastic NSFD scheme.

1 Introduction

Typhoid is consequent of a disease with similar symptoms called typhus. The cause of this endemic disease is highly virulent bacterium Salmonella typhi. This bacterium spread through contaminated water and carrier of this bacterium. The symptoms of the typhoid are sustained fever, very poor appetite, vomiting, severe headache and fatigue. Typhoid has an incubation period of 7 to 14 days. The germ lives in the intestine of the patient which is its natural habitat. The multiple mononuclear phagocytic cells are added into the bloodstream [Cai and Li (2010); Nthiiri, Lawi, Akinyi et al. (2016)]. Treatment of typhoid depends upon the blood culture of the patient. When the strain is sensitive amoxicillin, chloramphenicol is given orally. In the asymptomatic carrier, the oral dose of ciprofloxacin or norfloxacin is used to wipe out the problem. It has become difficult to treat by antibiotics throughout the world because of multi-drug resistant strain. In many countries, the goal of wiping out the disease can only be achieved by providing healthy pure water, safe, sanitary conditions, healthy food and above-mentioned medical facilities. Although it is tough to

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achieve this goal, these steps may minimize or blot out the disease. The public can be made conscious after having health educational programs which change the behaviour towards the precautions and treatment of the disease. Millions of people across the world are being affected by typhoid every year. According to many surveys, every year about 20 million cases are reported, and approximately 200,000 are deceased annually. According to a survey in Africa, 50/100,000 people are dying because of typhoid, where 400,000 cases are reported annually [Cvjetanovic, Grab and Uemura (1971); Regan, Kelly, Korobeinikov et al. (2010)]. Currently, oral and injectable vaccines are being used to treat typhoid, but these two are not enough to treat the disease. If the infected person is treated with drug-resistant strain, then it can reduce the duration of illness. Many mathematical models designed have been used to explain and analysis the dynamics of infectious disease. Ordinary differential equations are formulated in the presence of many assumptions and parameters.

The amount of newly infected folks is voiced as a role of the infectious and susceptible folks in a municipal within a given time in this model. The age edifices of the populace are established, which enables more detailed simulation of the upshot of various intercessions and tactics to control the disease in diverse age groups. The study indicates that once the incidence rate of the contagion has collapsed underneath the threshold quantity. It cannot be sustained in a community owed to the loss of the core source of infection long-lasting hailers as they die out unsurprisingly. The Mathematical model for transmission dynamics of typhoid is developed in demand to evaluate the budding, straight and incidental possessions of vaccination. The model is validated against randomized serum prosecutions. It is evaluated on school-based vaccination strategies, and it is discovered that typhoid vaccination is projected to lead a short-term incidental fortification and decrease in typhoid frequencies. However, vaccination alone is questionable lead to the elimination of typhoid. Mutually short and long-term shippers contribute to transmission but not necessarily at the same rate as primary infections [Cui, Tao and Zhu (2008)].

A simple mathematical model is developed by direct and indirect protection by vaccine and the benefits of the generic vaccination program. The population is divided into vaccinated, and the unvaccinated subgroups and its effectiveness redefined. It is found that vaccination reduces the number of susceptible to infection and fewer infected individuals spread the disease among vaccinated and unvaccinated persons. A mathematical model on the influence of control strategies to successfully control the drain of the upshot of shippers on the typhoid fever in Kisii town is developed and analyzed. This model showed the dynamics of typhoid fever by verbalizing and scrutinizing the bearing of hauliers, verdict and health education on typhoid hauliers' control in Kenya [Triampo, Baowan, Tang et al. (2007)]. The model considered that exposed individuals developed the typhoid fever due to endogenous renaissance and exogenous re-infection. The investigation exertion allows the latent and infectious period to have a dispersal other than the exponential. Numerical results show that dipping the typhoid shippers by 9.5% could contribution Kisii county regime in Kenya to accomplish a typhoid free spot by 2030 [Holt, Davis and Leirs (2006)].

Mathematical modelling has emerged as an effective tool to extract comprehensive insight knowledge about epidemic diseases. The formation of the model and the possible simulations allow for scrutinising the sensitivity and comparison of conjuncture patterns. As a result, the prediction of mediator, host and ecological factor affecting public health is

possible, and health policymakers can scientifically suggest and implement health services [Anwar, Goldberg, Fraser et al. (2014)]. Several studies have been conducted on various models of typhoid fever transmission dynamics [Cai and Li (2010); Nthiiri, Lawi, Akinyi et al. (2016)]. 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 Euler Maruyama, stochastic Euler and stochastic Runge-Kutta methods can bring about perplexing chaos and deceiving oscillations for specific concentrations of the discretization parameters [Zafar, Rehan and Mushtaq (2017); Zafar, Rehan, Mushtaq et al. (2017); Zafar, Rehan and Mushtaq (2017); Bayram, Partal and Buyukoz (2018)]. Due to these reasons and some other schemes dependent numerical instabilities such methods proved to be less fortunate options.

In general, the elasticity of stochastic differential equations (SDEs) are difficult, and the solutions of stochastic differential equations do not exist explicitly. We use different numerical schemes to integrate these equations in sagacity of convergence is difficult [Mickens (1994); Mickens (2005); Cresson and Pierret (2014); Pierret (2015)]. A natural question on numerical schemes can be the following despite convergence analysis: Do the numerical schemes preserve the dynamical properties of the initial system [Mickens (2005)].

In the deterministic modelling, we have pragmatic usual numerical schemes Euler and Runge-Kutta do not preserve dynamical properties. However, in stochastic case, the Euler Maruyama scheme, stochastic Euler scheme and stochastic Runge-Kutta scheme do not preserve the dynamical properties. Here the question arises: Can we construct a stochastic numerical scheme which preserves all dynamical properties?

The main theme of this paper is to introduce the idea of stochastic nonstandard finite difference scheme (SNSFD) based on the rules introduced in the deterministic case by [Mickens (1994, 2005)].

The strategy of this paper as follows:

In Section 2, we review classical definitions and some history of stochastic differential equations (SDEs) calculus. In Section 3, we instruct the invention of stochastic epidemic models. In Section 4, we discuss the deterministic typhoid fever model and equilibrium points. In Section 5, we discover the stochastic typhoid fever model. In Section 6, we will introduce the different stochastic numerical schemes and linked their result with deterministic solutions. In Section 7, we will conclude and will give the future directions.

2 Preliminaries

Einstein gave the idea of stochastic differential equations (SDEs) in (1905) and a mathematical gathering between microscopic random motion of particles and the macroscopic diffusion equation [Gard (1988); Karatzas and Shreve (1991); Platen (1991); Mickens (2005); Allen (2007); Britton (2010)]. Today the SDEs are fascinating many attentions due to physical expansions in real life system because the ordinary differential equations (ODEs) did not include random apprehension forcing and stochastic inputs. A stochastic calculus distributes a mathematical constituent for the manner of stochastic differential equations (SDEs). In general form, we can write the stochastic differential equation that comprises parameters. Continuous time t and variable T_t, as follows:

$$dT_t = u(t, T_t)dt + v(t, T_t)dB(t). \quad (1)$$

moreover, the integral form is

$$T(t) = c + \int_{t_o}^{t} u(s, T_s)ds + \int_{t_o}^{t} v(s, T_s)dB_s. \quad (2)$$

The differential equation (1) is also called the Ito stochastic differential equation (SDE) where $u(t, T_t)$ and $v(t, T_t)$ are drift and diffusion coefficients respectively. The casual variable c is called the initial value at the instant t_o. A solution T_t of Eqs. (1) or (2) is called a stochastic process.

2.1 Brownian motion

The Brownian motion can be defined as a continuous time haphazard walk with the following properties [Gard (1988); Oksendal (2003)].

(i) $B_0 = 0$.

(ii) B_t must be continuous, the event happens with probability one. The sample trajectories $t \to B_t$ are continuous with probability one.

(iii) For any finite sequence of times $t_1 < t_2 < t_3 \ldots < t_n$ then the following paths $B_{t_1} - B_{t_o}, B_{t_2} - B_{t_1}, B_{t_3} - B_{t_2} \ldots, B_{t_n} - B_{t_{n-1}}$ are independent.

(iv) For any times $0 \le s \le t, B_t - B_s$ is normally distributed with mean zero and variance is $t - s$. In particularly we say that $\text{expectation of } [B_t - B_s] = 0 \text{ and variance of } [B_t - B_s] = t - s.$

The stochastic process is a fundamental example of Brownian motion. The study of stochastic epidemic model based on stochastic modelling processes, but the stochastic modelling process is a grouping of random variables $\{T_t(S)/t\epsilon T, s\epsilon S\}$, where T the guide is set and S is a joint sample space. The guide set may often personify time such as $T = \{0,1,2, \ldots\}$ or $T = [0, \infty)$. So, the time may have discrete or continuous. The study of stochastic modelling processes is based on probability theory. We will describe the stochastic epidemic modelling processes in three different ways such as DTMC (Discrete Time Markov Chain) epidemic models, CTMC (Continuous Time Markov Chain) epidemic models and SDEs (Stochastic Differential Equations) epidemic models [Shoji and Ozaki (1997); Shoji and Ozaki (1998)]. We will assume the time and the state variables are discrete in discrete time Markov chain (DTMC) epidemic models and the time is continuous, and the state variables are discrete in continuous time Markov chain (CTMC) epidemic models. We will assume both time and variables as continuous in stochastic differential equations (SDEs) epidemic models. Now! We will discuss Ito stochastic differential equations (SDEs) in stochastic epidemic models. It was first introduced and developed by Ito in 1942. In order to illustrate the development of the stochastic process is almost in all sciences such as economics, mathematics, physics, chemistry and biology. Due to their non-differentiable character of realization of the Brownian motion, the solutions of stochastic differential equations (SDEs) are not given explicitly. So! The stochastic numerical approximation is used to study the properties of stochastic epidemic models [Maruyama (1955); Kloeden and Platen (1992); Kloeden, Platen and Schurz (1994); Bayram, Partal and Buyukoz (2018)].

3 Construction of stochastic epidemic models

Epidemics are usually twisted by non-linear systems pragmatic through patchy noisy data. The epidemic models can be divided into two main types such as deterministic epidemic models and stochastic epidemic models. The deterministic epidemic models do not preserve the natural uncertainty of disease dynamics, but the idea of stochastic epidemic models preserves all types of the uncertainty of disease dynamics. There are numerous conducts to diffuse the deterministic epidemic models to stochastic epidemic models [Allen, Allen, Arciniega et al. (2008)]. The stochastic epidemic modelling has been done by discrete time Markov chain (DTMC), continuous time Markov chain (CTMC) and Ito stochastic differential equations (SDEs). Consequently! The idea of Ito stochastic differential equations gives a more opportune way to move from deterministic epidemic models to stochastic epidemic models. The idea of Ito stochastic differential equation can be pronounced by the following methods such as parametric perturbation and non-parametric perturbation methods. In parametric perturbation method, we will choose a parameter from the model and transformed into random variables of the model. In non-parametric perturbation method, we will introduce the Brownian processes in each differential equation (or introduce the extra stochasticity parameters). The non-parametric perturbation method is more useful as compared to parametric perturbation method. Another way of non-parametric perturbation method is introduced by Allen [Karatzas and Shreve (1991); Platen (1991); Allen and Burgin (2000); Holt, Davis and Leirs (2006); Allen (2007); Britton (2010)] in which any extra stochasticity parameters is not introduced in the model. Here we will frame the ways of non-parametric perturbation method in deterministic epidemic models and use different numerical methods to prompt them and check the efficiency of numerical methods on stochastic epidemic models. We will observe the association between the solutions of deterministic epidemic models and stochastic epidemic models.

4 Deterministic typhoid fever model

Figures and tables should be inserted in the text of the manuscript.

In this segment, we consider the deterministic typhoid fever model incorporating with protection against infection [Nthiiri, Lawi, Akinyi et al. (2016)]. Let at any arbitrary time t, the variables are stated as $T_1(t)$ exemplifies protected humans' fraction, $T_2(t)$ exemplifies susceptible humans' fraction, $T_3(t)$ exemplifies infected humans' fraction and $T_4(t)$ exemplifies treated humans' fraction. The communication dynamics of typhoid fever model as shown in Fig. 1.

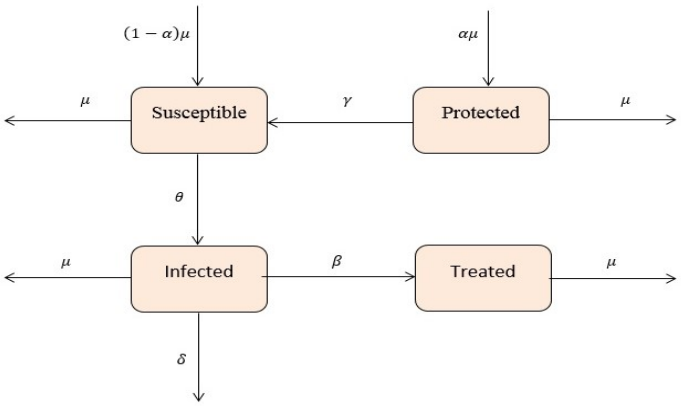


Figure 1: Flow diagram of typhoid fever model

The model parameters are pronounced as β (pronounces rate of treated humans from infected humans fraction), α (pronounces the enrolment rate into the conference of protected humans against typhoid), $(1-\alpha)$ (pronounces the rate of those humans who have chances to get virus), δ (pronounces the transience rate of humans by typhoid fever), θ (pronounces the susceptible humans acquire typhoid fever infection at per capita rate), μ (pronounces the natural rate of death/birth of humans).

The governing equations of the typhoid fever model are given below as

$$\left.\begin{aligned}
\frac{dT_1(t)}{dt} &= \alpha\mu - (\gamma+\mu)T_1(t)\\
\frac{dT_2(t)}{dt} &= (1-\alpha)\mu + \gamma T_1(t) - \theta T_2(t)T_3(t) - \mu T_2(t)\\
\frac{dT_3(t)}{dt} &= \theta T_2(t)T_3(t) - (\delta+\beta+\mu)T_3(t)\\
\frac{dT_4(t)}{dt} &= \beta T_3(t) - \mu T_4(t)
\end{aligned}\right\} \tag{3}$$

where the constant size of total humans under as

$$T_1(t) + T_2(t) + T_3(t) + T_4(t) = 1. \tag{4}$$

4.1 Steady states of the typhoid fever model

The steady state of typhoid fever model (3) can be categorized into two ways of equilibrium points under as

Disease-free equilibrium is $D_1{}^* = (T_1{}^o, T_2{}^o, T_3{}^o, T_4{}^o) = \left(\frac{\alpha\mu}{\gamma+\mu}, \frac{(\gamma+\mu)(1-\alpha)\mu+\gamma\alpha\mu}{\mu(\gamma+\mu)}, 0, 0\right)$.

Endemic equilibrium is $E_1{}^* = (T_1{}^o, T_2{}^o, T_3{}^o, T_4{}^o)$.

$E_1{}^* =$

$$\left(\frac{\alpha\mu}{\gamma+\mu}, \frac{\delta+\beta+\mu}{\theta}, \frac{(\gamma+\mu)(1-\alpha)\mu\theta+\gamma\alpha\mu\theta-\mu(\delta+\beta+\mu)(\gamma+\mu)}{\theta(\delta+\beta+\mu)(\gamma+\mu)}, \frac{\beta[(\gamma+\mu)(1-\alpha)\mu\theta+\gamma\alpha\mu\theta-\mu(\delta+\beta+\mu)(\gamma+\mu)]}{\mu\theta(\delta+\beta+\mu)(\gamma+\mu)}\right).$$

where $T^* = \frac{\theta(\gamma+\mu-\alpha\mu)}{(\delta+\beta+\mu)(\gamma+\mu)}$.

Note that T^* is the reproductive number of the typhoid fever model (3). The reproductive number has a vital role in disease dynamics. If the reproductive number $T^* < 1$ then this strategy helps us to control the disease and if $T^* > 1$ then this will be an alarming situation of disease in the population.

5 Stochastic typhoid fever model

Let $T(t) = [T_1(t), T_2(t), T_3(t), T_4(t), T_5(t)]^T$ to form the stochastic differential equations (SDEs) of typhoid fever model (1). We want to calculate the expectations $E^*[\Delta T]$ and $E^*[\Delta T \Delta T^T]$ to find these expectations the possible changes along with their associated transition probabilities are listed in the following (see Tab. 1).

Table 1: Possible changes in the process for the typhoid fever model (3)

Transition	Probabilities
$(\Delta Z)_1 = [1,0,0,0]^T$	$P_1 = \alpha\mu\, \Delta t.$
$(\Delta Z)_2 = [-1,1,0,0]^T$	$P_2 = \gamma T_1(t)\Delta t.$
$(\Delta Z)_3 = [-1,0,0,0]^T$	$P_3 = \mu T_1(t)\Delta t.$
$(\Delta Z)_4 = [0,1,0,0]^T$	$P_4 = (1-\alpha)\mu\Delta t.$
$(\Delta Z)_5 = [0,-1,1,0]^T$	$P_5 = \theta T_2(t)T_3(t)\Delta t.$
$(\Delta Z)_6 = [0,-1,0,0]^T$	$P_6 = \mu T_2(t)\Delta t.$
$(\Delta Z)_7 = [0,0,-1,0]^T$	$P_7 = (\delta+\mu)T_3(t)\Delta t.$
$(\Delta Z)_8 = [0,0,-1,1]^T$	$P_8 = \beta T_3(t)\Delta t.$
$(\Delta Z)_9 = [0,0,0,-1]^T$	$P_9 = \mu T_4(t)\Delta t.$

The expectation of typhoid fever model (3) is defined as

$E^*[\Delta T] = \sum_{i=1}^{9} P_i\, (\Delta T)_i.$

$$\text{Expectation} = E^*[\Delta T] = \begin{bmatrix} \alpha\mu - (\gamma+\mu)T_1(t) \\ (1-\alpha)\mu + \gamma T_1(t) - \theta T_2(t)T_3(t) - \mu T_2(t) \\ \theta T_2(t)T_3(t) - (\delta+\beta+\mu)T_3(t) \\ \beta T_3(t) - \mu T_4(t) \end{bmatrix} \Delta t.$$

The variance of typhoid fever model is defined as Var= $E^*[\Delta T \Delta T^T] = \sum_{i=1}^{9} P_i\, [(\Delta T)_i][(\Delta T)_i]^T.$

$$E^*[\Delta T \Delta T^T] = \begin{bmatrix} W_{11} & W_{12} & W_{13} & W_{14} \\ W_{21} & W_{22} & W_{23} & W_{24} \\ W_{31} & W_{32} & W_{33} & W_{34} \\ W_{41} & W_{42} & W_{43} & W_{44} \end{bmatrix} \Delta t.$$

where,

$W_{11} = \alpha\mu + (\gamma+\mu)T_1(t)\,, W_{12} = -\gamma T_1(t)\,,\ W_{13} = 0 W_{14} = 0\,, W_{21} = -\gamma T_1(t)\,, W_{22} = (1-\alpha)\mu + \gamma T_1(t) + \theta T_2(t)T_3(t) + \mu T_2(t)\,, W_{23} = -\theta T_2(t)T_3(t)\ W_{24} = 0, W_{31} = 0\,, W_{32} = -\theta T_2(t)T_3(t), W_{33} = \theta T_2(t)T_3(t) + (\delta+\beta+\mu)T_3(t)\,, W_{34} = -\beta T_3(t) W_{41} = 0, W_{42} = 0,\ W_{43} = -\beta T_3(t)$ and $W_{44} = \beta T_3(t) + \mu T_4(t).$

The stochastic differential equation satisfies the diffusion processes, So
$\frac{dT(t)}{dt} = G(T(t), t) + H(T(t), t)\frac{dB(t)}{dt}$.

If we define $\text{drift} = G(T(t), t) = \frac{E^*[\Delta T]}{\Delta t}$ and $\text{diffusion} = H(T(t), t) = \sqrt{\frac{E^*[\Delta T \Delta T^T]}{\Delta t}}$, then the stochastic differential equation of typhoid fever model (3) is

$$dT(t) = G(T(t), t)dt + H(T(t), t)dB(t). \quad (5)$$

with initial conditions $T(0) = T_o = [0.2, 0.4, 0.3, 0.1]^T$, $0 \le t \le T$ and $B(t)$ is the Brownian motion.

5.1 Euler maruyama scheme

Here we use Euler Maruyama scheme [Maruyama (1955)] to find the numerical solution of SDEs (5) by using the parameters values given in literature [Nthiiri, Lawi, Akinyi et al. (2016)] (see Tab. 2).

Table 2: Values of Parameter

Parameters	Values (Days)	
	DFE	**EE**
μ	0.0044	0.0044
θ	0.1	10
δ	0.005	0.005
α	0.8	0.8
β	0.9	0.9
γ	0.1	0.1
σ_1	0.09	0.09
σ_2	0.08	0.08
σ_3	0.07	0.07
σ_4	0.06	0.06

We can write the Euler Maruyama scheme of SDEs (5) is

$$T_{n+1} = T_n + f(T_n, t)\Delta t + L(T_n, t)dB(t).$$

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 numerical experiments. The solution of deterministic typhoid fever model for the disease-free equilibrium $D_1{}^* = (0.0332, 0.9663, 0, 0)$ and the reproductive number helps us to control this infection in human's population. The endemic equilibrium $E_1{}^* = (0.0332, 0.09094, 0.004235, 0.8666)$ and the reproductive number shows that disease is endemic in human's population. The graphical behaviour of Euler Maruyama scheme for both disease-free equilibrium and endemic equilibrium at different sub populations as shown in figures.

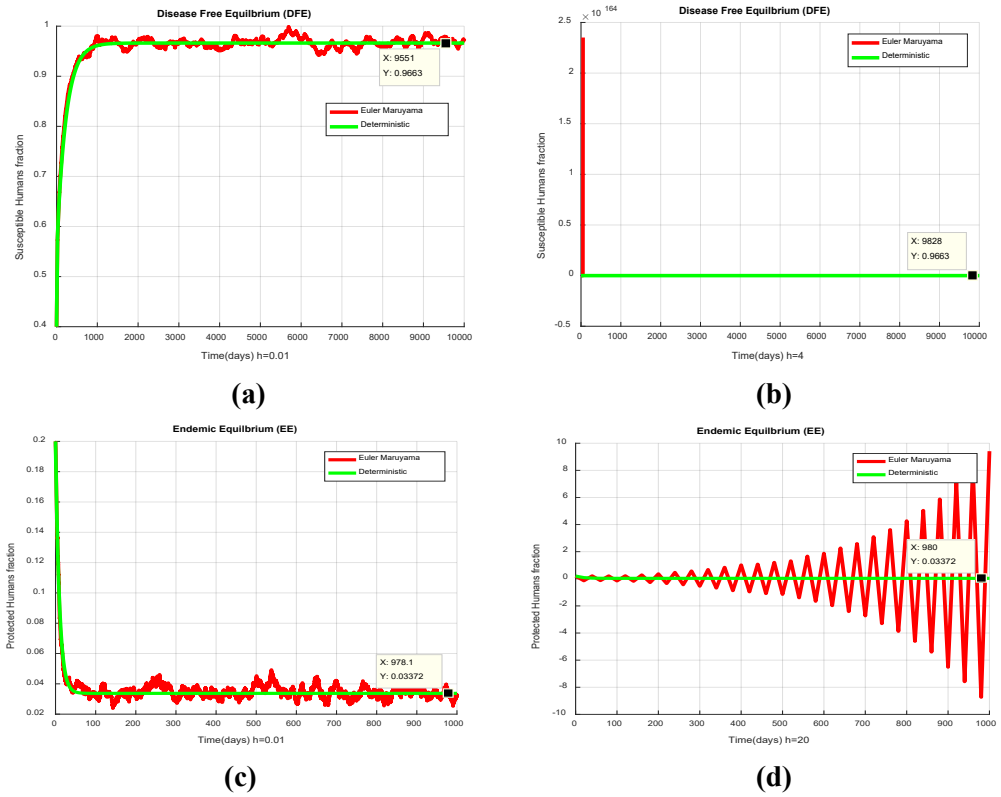


Figure 2: Comparison in solutions of euler maruyama and deterministic **(a)** Susceptible humans fraction at DFE Point for h=0.01 **(b)** Susceptible humans fraction at DFE Point for h=4 **(c)** Protected humans fraction at EE Point for h=0.01 **(d)** Protected humans fraction at EE Point for h=20

5.2 Non-parametric perturbation of stochastic typhoid fever model

Another way to construct the stochastic differential equations (SDEs) from the deterministic ordinary differential equations (ODEs) is to introduce the non-parametric perturbation in each differential equation of typhoid fever model (3) as

$$\left.\begin{aligned} dT_1(t) &= \big(\alpha\mu - (\gamma+\mu)T_1(t) + \sigma_1 dB_1(t)T_1(t)\big)dt \\ dT_2(t) &= \big((1-\alpha)\mu + \gamma T_1(t) - \theta T_2(t)T_3(t) - \mu T_2(t) + \sigma_2 dB_2(t)T_2(t)\big)dt \\ dT_3(t) &= \big(\theta T_2(t)T_3(t) - (\delta+\beta+\mu)T_3(t) + \sigma_3 dB_3(t)T_3(t)\big)dt \\ dT_4(t) &= \big(\beta T_3(t) - \mu T_4(t) + \sigma_4 dB_4(t)T_4(t)\big)dt \end{aligned}\right\} \quad (6)$$

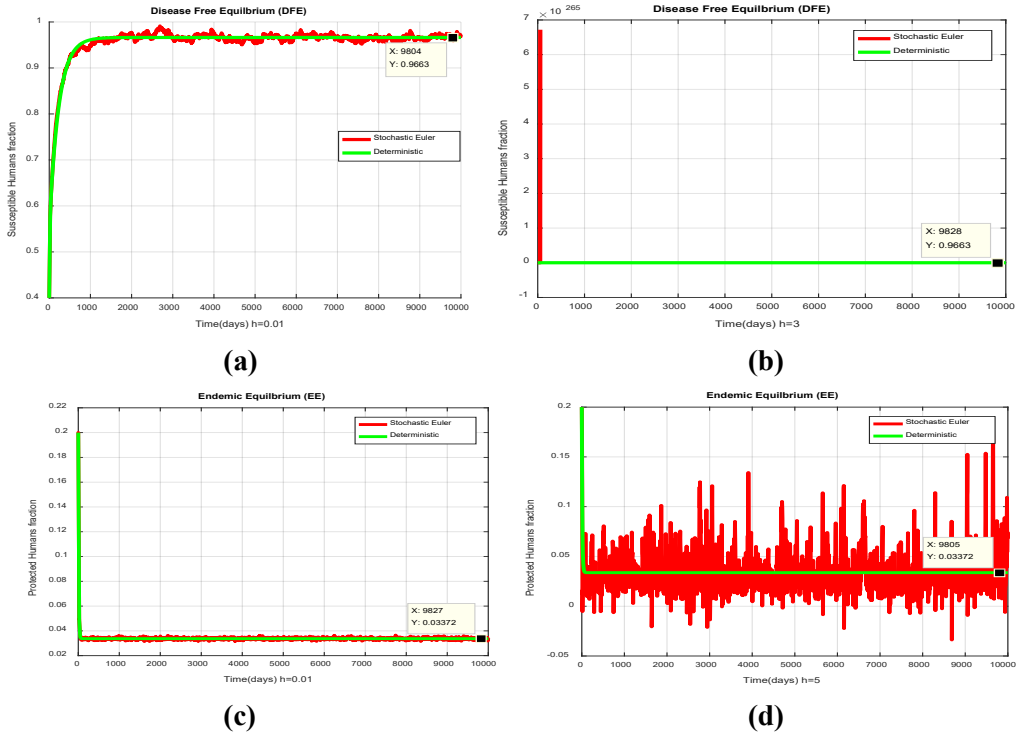
with initial conditions $T(0) = [T_1(0), T_2(0), T_3(0), T_4(0)]^T = [0.2, 0.4, 0.3, 0.1]^T$, where $\sigma_1, \sigma_2, \sigma_3$ and σ_4 is stochasticity of each compartment of the typhoid fever model and $B_j(t), (j = 1,2,3,4)$ are the independent Brownian motions. The non-parametric perturbation of stochastic typhoid fever model does not have the explicit solution due to a non-differentiability term of Brownian motion. So, we introduced some new stochastic numerical methods to find the solution of stochastic typhoid fever model (6).

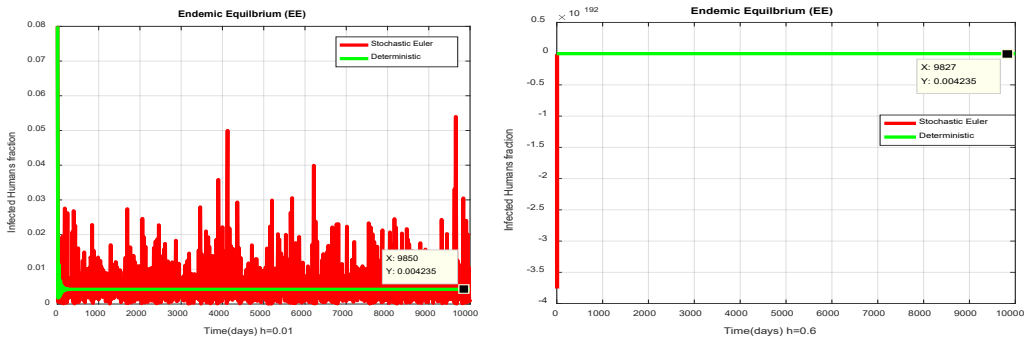
5.2.1 Stochastic euler scheme

The stochastic Euler scheme can be derived from the above non-parametric perturbation of stochastic typhoid fever model (6) as

$$\left.\begin{aligned} T_1^{n+1}(t) &= T_1^n(t) + h[\alpha\mu - (\gamma+\mu)T_1^n(t) + \sigma_1 dB_1(t)T_1^n(t)] \\ T_2^{n+1}(t) &= T_2^n(t) + h[(1-\alpha)\mu + \gamma T_1^n(t) - \theta T_2^n(t)T_3^n(t) - \mu T_2^n(t) + \sigma_2 dB_2(t)T_2^n(t)] \\ T_3^{n+1}(t) &= T_3^n(t) + h[\theta T_2^n(t)T_3^n(t) - (\delta+\beta+\mu)T_3^n(t) + \sigma_3 dB_3(t)T_3^n(t)] \\ T_4^{n+1}(t) &= T_4^n(t) + h[\beta T_3^n(t) - \mu T_4^n(t) + \sigma_4 dB_4(t)T_4^n(t)] \end{aligned}\right\} \quad (7)$$

where "h" is any time step size. We pretend the solution of stochastic Euler scheme by using the Matlab program and parameters values given in Nthiiri et al. [Nthiiri, Lawi, Akinyi et al. (2016)] (see Tab. 2).





(e) **(f)**

Figure 3: Comparison in solutions of stochastic euler and deterministic **(a)** Susceptible humans fraction at DFE Point for h=0.01 **(b)** Susceptible humans fraction at DFE Point for h=3 **(c)** Protected humans fraction at EE Point for h=0.01 **(d)** Protected humans fraction at EE Point for h=5 **(e)** Infected humans fraction at EE Point for h=0.01 **(f)** Infected humans fraction at EE Point for h=0.6

5.2.2 Stochastic runge-kutta scheme

The stochastic Runge-Kutta scheme can be derived from the above non-parametric perturbation of stochastic typhoid fever model (6) as

First Stage

$$A_1 = h[\alpha\mu - (\gamma+\mu)T_1{}^n(t) + \sigma_1 dB_1(t)T_1{}^n(t)].$$

$$B_1 = h[(1-\alpha)\mu + \gamma T_1{}^n(t) - \theta T_2{}^n(t)T_3{}^n(t) - \mu T_2{}^n(t) + \sigma_2 dB_2(t)T_2{}^n(t)].$$

$$C_1 = h[\theta T_2{}^n(t)T_3{}^n(t) - (\delta+\beta+\mu)T_3{}^n(t) + \sigma_3 dB_3(t)T_3{}^n(t)].$$

$$D_1 = h[\beta T_3{}^n(t) - \mu T_4{}^n(t) + \sigma_4 dB_4(t)T_4{}^n(t)].$$

Second Stage

$$A_2 = h\left[\alpha\mu - (\gamma+\mu)(T_1{}^n(t) + \frac{A_1}{2}) + \sigma_1 dB_1(t)(T_1{}^n(t) + \frac{A_1}{2})\right].$$

$$B_2 = h\left[(1-\alpha)\mu + \gamma\left(T_1{}^n(t) + \frac{A_1}{2}\right) - \theta\left(T_2{}^n(t) + \frac{B_1}{2}\right)\left(T_3{}^n(t) + \frac{C_1}{2}\right) - \mu\left(T_2{}^n(t) + \frac{B_1}{2}\right) + \sigma_2 dB_2(t)(T_2{}^n(t) + \frac{B_1}{2})\right].$$

$$C_2 = h\left[\theta\left(T_2{}^n(t) + \frac{B_1}{2}\right)\left(T_3{}^n(t) + \frac{C_1}{2}\right) - (\delta+\beta+\mu)\left(T_3{}^n(t) + \frac{C_1}{2}\right) + \sigma_3 dB_3(t)\left(T_3{}^n(t) + \frac{C_1}{2}\right)\right].$$

$$D_2 = h\left[\beta\left(T_3{}^n(t) + \frac{C_1}{2}\right) - \mu\left(T_4{}^n(t) + \frac{D_1}{2}\right) + \sigma_4 dB_4(t)\left(T_4{}^n(t) + \frac{D_1}{2}\right)\right].$$

Third Stage

$$A_3 = h\left[\alpha\mu - (\gamma+\mu)(T_1{}^n(t) + \frac{A_2}{2}) + \sigma_1 dB_1(t)(T_1{}^n(t) + \frac{A_2}{2})\right].$$

$$B_3 = h\left[(1-\alpha)\mu + \gamma\left(T_1{}^n(t) + \frac{A_2}{2}\right) - \theta\left(T_2{}^n(t) + \frac{B_2}{2}\right)\left(T_3{}^n(t) + \frac{C_2}{2}\right) - \mu\left(T_2{}^n(t) + \frac{B_2}{2}\right) + \sigma_2 dB_2(t)(T_2{}^n(t) + \frac{B_2}{2})\right].\ C_3 = h\left[\theta\left(T_2{}^n(t) + \frac{B_2}{2}\right)\left(T_3{}^n(t) + \frac{C_2}{2}\right) - (\delta+\beta+\mu)\left(T_3{}^n(t) + \frac{C_2}{2}\right) + \sigma_3 dB_3(t)\left(T_3{}^n(t) + \frac{C_2}{2}\right)\right].$$

$$D_3 = h\left[\beta\left(T_3{}^n(t) + \frac{C_2}{2}\right) - \mu\left(T_4{}^n(t) + \frac{D_2}{2}\right) + \sigma_4 dB_4(t)\left(T_4{}^n(t) + \frac{D_2}{2}\right)\right].$$

Fourth Stage

$$A_4 = h[\alpha\mu - (\gamma+\mu)(T_1{}^n(t) + A_3) + \sigma_1 dB_1(t)(T_1{}^n(t) + A_3)].$$

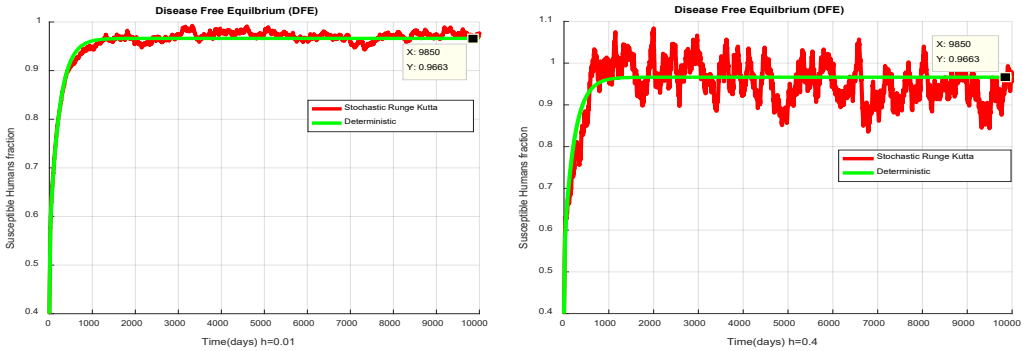
$$B_4 = h[(1-\alpha)\mu + \gamma(T_1{}^n(t) + A_3) - \theta(T_2{}^n(t) + B_3)(T_3{}^n(t) + C_3) - \mu(T_2{}^n(t) + B_3) + \sigma_2 dB_2(t)(T_2{}^n(t) + B_3)].\ C_4 = h[\theta(T_2{}^n(t) + B_3)(T_3{}^n(t) + C_3) - (\delta+\beta+\mu)(T_3{}^n(t) + C_3) + \sigma_3 dB_3(t)(T_3{}^n(t) + C_3)].$$

$$D_4 = h[\beta(T_3{}^n(t) + C_3) - \mu(T_4{}^n(t) + D_3) + \sigma_4 dB_4(t)(T_4{}^n(t) + D_3)].$$

Final Stage

$$\left.\begin{aligned} T_1{}^{n+1}(t) &= T_1{}^n(t) + \left(\frac{1}{6}\right)[A_1 + 2A_2 + 2A_3 + A_4] \\ T_2{}^{n+1}(t) &= T_2{}^n(t) + \left(\frac{1}{6}\right)[B_1 + 2B_2 + 2B_3 + B_4] \\ T_3{}^{n+1}(t) &= T_3{}^n(t) + \left(\frac{1}{6}\right)[C_1 + 2C_2 + 2C_3 + C_4] \\ T_4{}^{n+1}(t) &= T_4{}^n(t) + \left(\frac{1}{6}\right)[D_1 + 2D_2 + 2D_3 + D_4] \end{aligned}\right\} \quad (8)$$

where "h" is any time step size. We pretend the solution of stochastic Runge Kutta scheme by using Mat-lab program and parameters values given in Nthiiri et al. [Nthiiri, Lawi, Akinyi et al. (2016)] (see Tab. 2).



(a) **(b)**

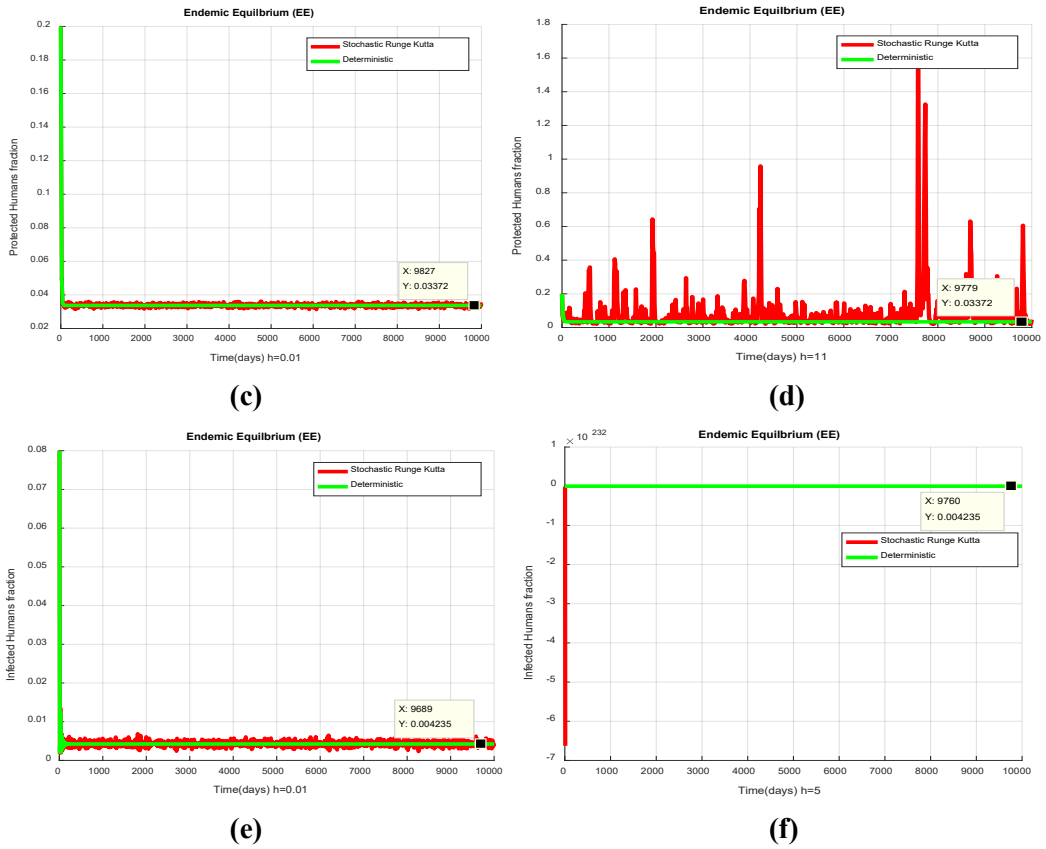


Figure 4: Comparison in solutions of stochastic runge kutta and deterministic **(a)** Susceptible humans fraction at DFE Point for h=0.01 **(b)** Susceptible humans fraction at DFE Point for h=0.4 **(c)** Protected humans fraction at EE Point for h=0.01 **(d)** Protected humans fraction at EE Point for h=11 **(e)** Infected humans fraction at EE Point for h=0.01 **(f)** Infected humans fraction at EE Point for h=5

5.2.3 Stochastic NSFD scheme

The proposed frame work of stochastic nonstandard finite difference scheme (SNSFD) can be derived from the above non-parametric perturbation of stochastic typhoid fever model (6) as

$$\left.\begin{aligned}
T_1^{n+1}(t) &= \frac{T_1^{n}(t)+\phi(h)\left[\alpha\mu+\sigma_1 dB_1(t)T_1^{n}(t)\right]}{\left(1+\phi(h)(\gamma+\mu)\right)}\\
T_2^{n+1}(t) &= \frac{T_2^{n}(t)+\phi(h)\left[(1-\alpha)\mu+\gamma T_1^{n}(t)+\sigma_2 dB_2(t)T_2^{n}(t)\right]}{\left(1+\phi(h)\theta T_3^{n}(t)+\mu\phi(h)\right)}\\
T_3^{n+1}(t) &= \frac{T_3^{n}(t)+\phi(h)\left[\theta T_2^{n}(t)T_3^{n}(t)+\sigma_3 dB_3(t)T_3^{n}(t)\right]}{\left(1+\phi(h)(\delta+\beta+\mu)\right)}\\
T_4^{n+1}(t) &= \frac{T_4^{n}(t)+\phi(h)\left[\beta T_3^{n}(t)+\sigma_4 dB_4(t)T_4^{n}(t)\right]}{\left(1+\mu\phi(h)\right)}
\end{aligned}\right\} \quad (9)$$

where $\phi(h) = 1 - \exp(-h)$ and "h" is any time step size. We pretend the solution of proposed frame work of stochastic nonstandard finite difference (SNSFD) scheme by using the Matlab program and parameters values given in Nthiiri et al. [Nthiiri, Lawi, Akinyi et al. (2016)] (see Tab. 2).

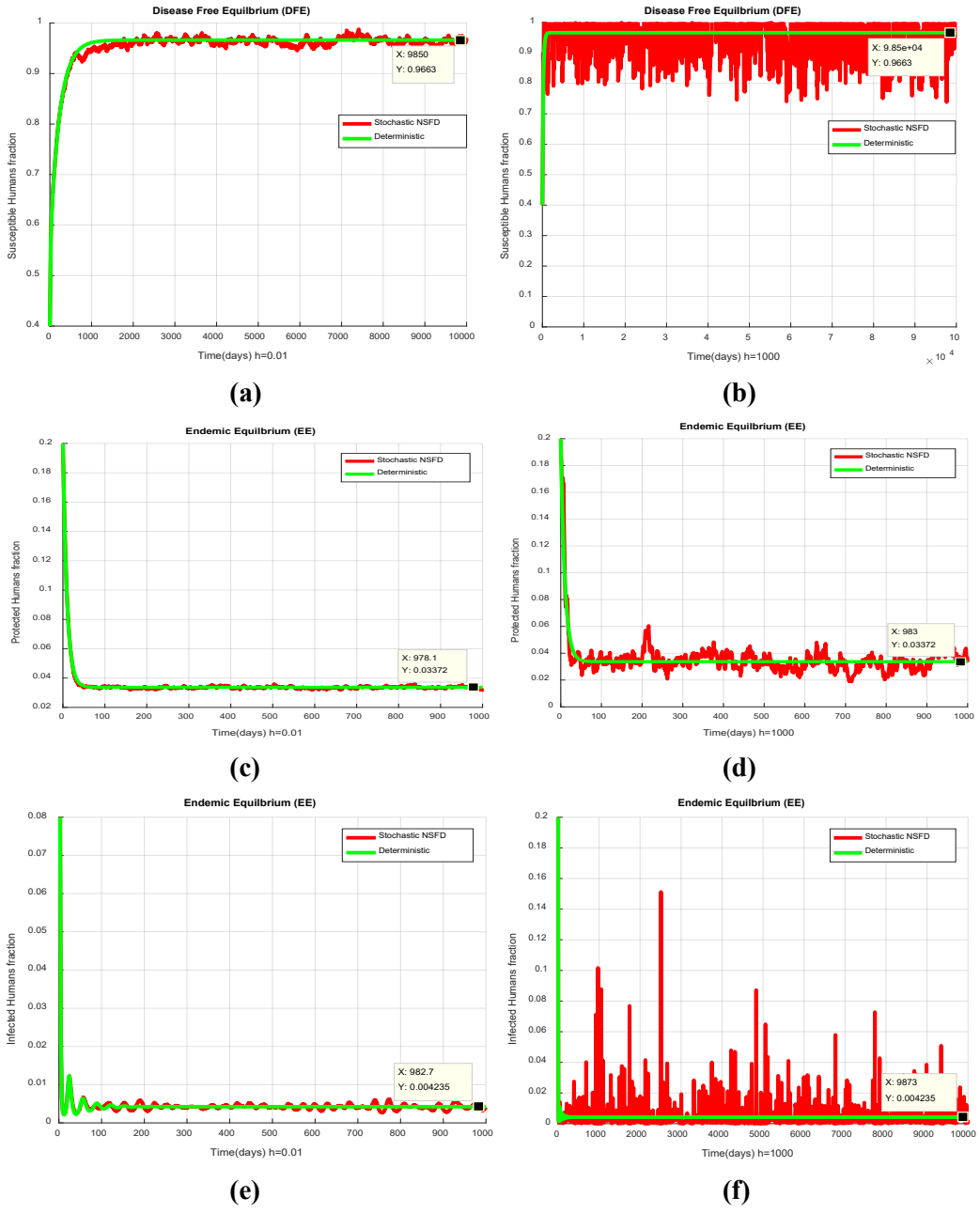


Figure 5: Comparison in solutions of stochastic NSFD and deterministic **(a)** Susceptible humans fraction at DFE Point for h=0.01 **(b)** Susceptible humans fraction at DFE Point for

h=1000 **(c)** Protected humans fraction at EE Point for h=0.01 **(d)** Protected humans fraction at EE Point for h=1000 **(e)** Infected humans fraction at EE Point for h=0.01 **(f)** Infected humans fraction at EE Point for h=1000

6 Results and discussion

In Fig. 2, it is observed that the Euler Maruyama scheme converges the steady states of the typhoid fever model while the deterministic solution is the mean of Euler Maruyama solution for descritezation h=0.01 at different sub population fractions. When the time step size is increased, the Euler Maryuama scheme fails to maintain positivity and boundedness for both disease free equilibrium and endemic equilibrium at different sub population fractions. Consequently, Euler Maryuama scheme does not work for any time step size.

In Fig. 3, it is observed that the stochastic Euler scheme converges the steady states equilibrium while the deterministic solution is the mean of stochastic Euler solution for descritezation h=0.01 at different sub population fractions. When the time step size has been increased, the stochastic Euler scheme fails to maintain positivity and boundedness for both disease free equilibrium and endemic equilibrium at different sub population fractions. So, the stochastic Euler scheme is not a reliable technique to find the solutions of stochastic typhoid model.

Fig. 4 shows that the stochastic Runge-Kutta scheme converges the disease free equilibrium and endemic equilibrium while the deterministic solution is the mean of stochastic Runge-Kutta solution for descritezation h=0.01 at different sub population fractions respectively. When the time step size is increased as shown in Fig. 4, the stochastic Runge-Kutta scheme fails to maintain boundedness and positivity for both disease free equilibrium and endemic equilibrium at different sub population fractions. So, the stochastic Runge-Kutta scheme does not work for any time step size. Thus, the aforasaid stochastic schemes do not preserve all dynamical properities [Mickens (1994, 2005)].

In Fig. 5, it has been shown that the stochastic NSFD scheme converges both disease free equilibrium and endemic equilibrium while the deterministic solution is the mean of stochastic NSFD solution for any descritezation such as h=0.01 and h=1000 at different sub population fractions respectively. So, the stochastic NSFD scheme preserves all dynamical properties such as positivity, boundedness and dynamical consistency defined by R. E. Mickens in a stochastic context. The proposed frame work stochastic NSFD scheme works for any time step size.

7 Conclusion and future frame work

The numerical analysis for the stochastic epidemic model is a more convenient strategy as compare to deterministic epidemic model to understand the typhoid dynamics incorporating with protection against infection. The Euler Maruyama scheme, stochastic Euler scheme and stochastic Runge-Kutta scheme converges the true equilibrium points for very small-time step size, after increasing the time step size these schemes diverge and lose the dynamical properties such as positivity, bounded-ness and dynamical consistency. The proposed frame work of stochastic nonstandard finite difference scheme (SNSFD) of typhoid fever model works for any time step size defined by Mickens [Mickens (1994, 2005)] in the stochastic framework. The above-mentioned frame work (SNSFD) is suitable

for all types of non-linear and complicated stochastic epidemic models. The stochastic solutions are very close to the deterministic ODEs solutions. The study of stochastic epidemic models plays a most important role in disease dynamics. We have observed that stochastic epidemic models are more realistic as compared to deterministic epidemic models. For future work, the proposed (SNSFD) can be implemented to the complicated stochastic delay epidemic models and stochastic diffusion epidemic models. The numerical analysis proposed in this work could also be extended to fractional order dynamical system [Jajarmi and Baleanu (2018); Jajarmi, Baleanu, Bonyah et al. (2018)]. Our future plan is to construct a reliable numerical scheme for the fractional order stochastic epidemic model for various infectious diseases.

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