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Structure-Preserving Dynamics of Stochastic Epidemic Model with the Saturated Incidence Rate

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Abstract: The structure-preserving features of the nonlinear stochastic models are positivity, dynamical consistency and boundedness. These features have a significant role in different fields of computational biology and many more. Unfortunately, the existing stochastic approaches in literature do not restore aforesaid structure-preserving features, particularly for the stochastic models. Therefore, these gaps should be occupied up in literature, by constructing the structure-preserving features preserving numerical approach. This writing aims to describe the structure-preserving dynamics of the stochastic model. We have analysed the effect of reproduction number in stochastic modelling the same as described in the literature for deterministic modelling. The usual explicit stochastic numerical approaches are time-dependent. We have developed the implicitly driven explicit approach for the stochastic epidemic model. We have proved that the newly developed approach is preserving the structural, dynamical properties as positivity, boundedness and dynamical consistency. Finally, convergence analysis of a newly developed approach and graphically illustration is also presented.

Keywords: Epidemic model, stochastic numerical approaches, convergence analysis.

1 Introduction

Among the significant disasters to human health, one is an infectious disease. Particular methods like pathology, virology, epidemiology and culture are being used by scientists or researchers to study the growth of infectious disease for the protection of human life and to prevent the common-ness of infectious disease. Dietz et al. [Dietz and Heesterbeek

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(2002)] presented a very beneficial method called mathematical modelling method used firstly for the examining of smallpox. Zhao et al. [Zhao, Yuan and Liu (2018)] presented a differential equation model on the study of malaria disease. Commonly, the total population into three separate classes which are listed as S(t): susceptible means healthy but may be open to diseases, I(t): infective means who are sick and may transfer it further and R(t): recovery means who are sick but got the immunity against it. Many scholars have explored the susceptible, infected and recovered (SIR) model. According to the SIR model, a susceptible individual may be infected as well as infected may be recovered and vaccinated intended for the whole lifespan. A short-term retrieval, recovery may produce, which may mislay resistance with time and goes to the susceptible group, it happened in some prevalent ailment like smallpox, tetanus, cholera, influenza and typhoid fever. Bian et al. [Bian, Zhao, Song et al. (2017)] have found a different kind of SIS and susceptible, infected, recoved and susceptible (SIRS) models.

2 Literature survey

Li et al. [Li, Zhang, Liu et al. (2017)] suggested discrete-time stochastic models, which continued by ages of infectives. Considering the entire inhabitants secured by *N*, the correspondent of the threshold R_0^S can be recognized. In the condition $R_0^S > 1$ the model is verified to declare minimum one arbitrary interrupted solution, and this solution is non-linear. Besides, situations responsible for the determination and demolition at ailment are also well known. For the circumstances in which level of the sound is kept minor. Relating to the independency level SIS model, it comes near to close value in the unit cycle. Zhang at el. [Zhang, Meng and Wang (2018)] have studied on the random outcomes on the increase of contagious illness with full frequency rate and the unusual transmission from the virulent is debated in this article. The threshold dynamics discover the situations concerning moderately lesser uproar the consequences displayed that the blowout of the disease can be overpowered by its eradication which has its roots in large noise. Yu et al. [Yu, Yuan and Zhang (2018)] have studied on atmosphere haphazardness can be interrelated with alee effect and this effect is operational when inhabitants figure is less. Specifically, alee effect is used for the creation and inspection of a random solo class model under rule transferring. Initially, the presence of universal positive result of the model is demonstrated. Secondly, the existence of scrutiny is accomplished. To peruse enough situations for the extermination, non-existence in mean and existence in mean and random stability. By devising an appropriated Lyapunov function. It is displayed that the model is optimistically frequent and random. Our outcomes designate that rule transferring can control the demolition of species. Concluding, numerical simulations are performed to show the achieved hypothetical fallouts, which also debates on an actual sample presenting that adding alee effect in model delivers an improved tie to the data. Epidemics of infectious ailments with a complete spread rate will be discussed. The verge of dynamics is investigated comparatively small noise. Under a SIRS model, two types of Lyapunov functional techniques are employed with sorted restoration and incomplete recuperation rates. Complete universal modelling crescent are applied where R_0, the elementary reproduction number is the threshold criterion. For instance, when $R_0 < 1$ a balance without any infection is established asymptomatically world-wide. And when $R_0 > 1$ an endemic equilibrium is established asymptomatically

world-wide. Meng et al. [Meng, Wang and Zhang (2016)] has suggested a stochastic fatal diseases model which a cure through vaccination has introduced and appropriately incorporates. Consequently, deceptiveness and optimistic approach for the establishment of equilibrium regarding diseases free environment have been identified. As the deterministic model based on improbability whereas stochastic model accounts the number of individuals as a discretion. It is not imaginary, but close to the real world.

2.1 Stochastic epidemic modelling why?

A stochastic model is ideal when contemplating a little network. Be that as it may, notwithstanding when thinking about a huge network which deterministic models fundamentally are gone for some additional inquiries can be raised when thinking about stochastic plague models. For instance: what is the likelihood of a noteworthy episode? Furthermore, for models portraying an endemic circumstance: How long is the ailment liable to persevere (with or without intercession)? Later stochastic models have additionally demonstrated to be invaluable when the contact structure in the network contains little complete charts; families and other nearby interpersonal organizations being regular models. Allen et al. [Allen, Allen, Arciniega et al. (2008)] presented both deterministic and stochastic pandemic models have their significant tasks to carry out notwithstanding, the concentration in the present paper is on stochastic plague models. Cresson et al. [Cresson and Pierret (2014)] have found the dynamical properties of stochastic systems. Raza et al. [Raza, Arif, Rafiq et al. (2019a)], [Raza, Arif and Rafiq (2019b)] have given the rules to construct the implicitly driven explicit system for the given model. The detail of the rest of the sections is as follows: In the first section, explains the details about deterministic epidemic model and the points of equilibrium. In the second and third section, explains the stochastic model and its symmetries. In the fourth section compares the results of stochastic numerical approaches with the deterministic results. In the last section, we have a conclusion and coming directions.

3 Deterministic epidemic model

In this part, we consider the deterministic epidemic presented in the literature. For any time t, the specification of variables as follows: $S(t)$: signifies by a susceptible group of humans, $I(t)$: signifies by an infectious group of humans, $R(t)$: signifies by a recovered group of humans. The flow of the epidemic model as follows:

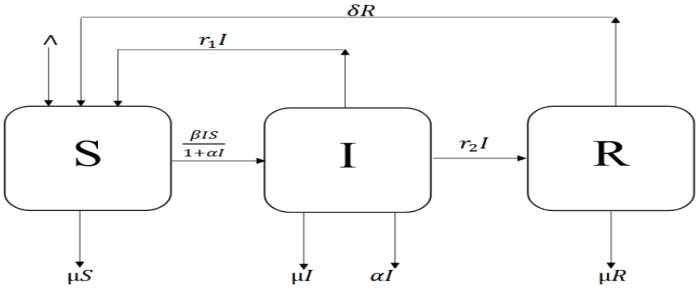


Figure 1: Diagram of epidemic model

where, Λ (denotes the recruitment of humans), β(denotes the ratio at which humans are infected), γ_1 (denotes the ratio of infectious who are susceptible after treatment), γ_2 (denotes the ratio at which humans are moved in the recovered compartment), δ (denotes the ratio of recovered humans who are again susceptible), μ (denotes the natural mortality ratio) and α (denotes the ratio of mortality due to sickness). The governing differential equations of the model as follows:

$$\frac{dS}{dt} = \Lambda - \mu S - \frac{\beta SI}{1+\alpha I} + \gamma_1 I + \delta R. \tag{1}$$

$$\frac{dI}{dt} = \frac{\beta SI}{1+\alpha I} - (\mu_1 + \gamma_1 + \gamma_2 + \alpha). \tag{2}$$

$$\frac{dR}{dt} = \gamma_2 I - (\mu + \delta)R. \tag{3}$$

where, $S \geq 0, I \geq 0, R \geq 0$.

$$S + I + R \leq \frac{\Lambda}{\mu}. \tag{4}$$

3.1 Equilibria of model

The equilibria of epidemic model as:

Disease free equilibrium is $D_1 = (\frac{\Lambda}{\mu}, 0, 0)$.

Endemic equilibrium is $E_1 = (S, I, R)$.

where, $S = \frac{(\mu_1+\gamma_1+\gamma_2+\alpha)}{\beta}$, $I = \frac{\beta\Lambda(\mu+\delta)-(\mu+\delta)\mu(\mu_1+\gamma_1+\gamma_2+\alpha)}{\beta[\mu\mu_1+\mu_1\delta+\gamma_2\mu+\alpha\mu+\alpha\delta]}$,

$R = \frac{\gamma_2[\beta\Lambda(\mu+\delta)-(\mu+\delta)\mu(\mu_1+\gamma_1+\gamma_2+\alpha)]}{\beta(\mu+\delta)[\mu\mu_1+\mu_1\delta+\gamma_2\mu+\alpha\mu+\alpha\delta]}$.

3.2 Force of infection

The reproduction number of the deterministic epidemic model is as follows:

$$R_o^d = \frac{\beta\Lambda}{\mu(\mu_1+\gamma_1+\gamma_2+\alpha)}. \tag{5}$$

This model has two states as the force of infection $R_o^d < 1$, will represent the disease-free population and the force of infection $R_o^d > 1$ will represent disease present in the human population.

4 Stochastic epidemic model

Arif et al. [Arif, Raza, Rafiq et al. (2019a)], [Arif, Raza, Rafiq et al. (2019b)] have given idea to introduce the stochastic environmental factors in Eqs. (1) to (3) as follows:

$$\beta dt = \beta dt + \sigma dB. \tag{6}$$

$$\gamma_2 dt = \gamma_2 dt + \sigma_1 dB. \tag{7}$$

$$dS = \left(\Lambda - \frac{\beta SI}{1+\alpha I} + \gamma_1 I + \delta R - \mu S\right) dt - \sigma \frac{SI}{1+\alpha I} dB. \tag{8}$$

$$dI = \left[\frac{\beta SI}{1+\alpha I} - (\mu_1 + \gamma_1 + \gamma_2 + \alpha)I\right] dt + \sigma \frac{SI}{1+\alpha I} dB - \sigma_1 I dB. \tag{9}$$

$$dR = [\gamma_2 I - (\mu + \delta)R]dt + \sigma_1 I dB. \tag{10}$$

where B is represented as Brownian movement and σ, σ_1 are the random environmental effects in the contact rates of humans. But $\frac{dS}{dt}+\frac{dI}{dt}+\frac{dR}{dt} \leq \Lambda - \mu(S+I+R)$. Let $N_1 = S+I+R$, then ${N_1}' \leq \Lambda - \mu N_1$. The initial value problem $\emptyset' = \Lambda - \mu\emptyset$ with $\emptyset(0) = N(0)$ has a solution $\emptyset(t) = \frac{\Lambda}{\mu} - ce^{-\mu t}$, and $\lim_{n\to\infty} \emptyset(t) = \frac{\Lambda}{\mu}$. Therefore, $N_1(t) \leq \emptyset(t)$, which shows $\lim_{t\to\infty} sup\, N_1(t) \leq \frac{\Lambda}{\mu}$. Thus the feasible region of the model is $\Omega = \left\{S,I,R): S+I+R \leq \frac{\Lambda}{\mu}\right\}$. The region Ω contains all the answers of Eqs. (8) to (10) which are bounded. Ultimately Ω is positive invariant.

4.1 Stochastic threshold dynamics

For Eqs. (8) to (10), the infected group represented by *I(t)* are said to be extinction if $\lim_{t\to\infty} I(t) = 0$ nearly sure.

Let us introduced,

$R_o^S = R_o^d - \frac{\sigma^2\Lambda^2}{2\mu^2\kappa.}$; $\kappa = (\mu + r_1 + r_2 + \alpha)$.

Theorem: If $\sigma^2 < \frac{\beta\mu}{\Lambda}$ and $R_o^S < 1$, then the infected group of the Eqs. (8) to (10) approaches to zero.

Proof:

Consider, (S,I,R) is a solution of the Eqs. (8) to (10) sustaining the condition $\big(S(0),I(0),R(0)\big) \in {R_+}^3$ by Ito's lemma and $f(I) = ln\,(I)$.

$dln(I) = f'(I)dI + \frac{1}{2}f''(I)I^2\left(\frac{S^2I^2\sigma^2}{1+\alpha I^2}\right)dt$

$d\,ln\,I = \left(\frac{\beta S}{1+\alpha I} - \kappa - \frac{\sigma^2S^2}{2(1+\alpha I)^2}\right)dt + \frac{\sigma S}{1+\alpha I}dB.$

$In\,I = \int_0^t \left(\frac{\beta S}{1+\alpha I} - \frac{\sigma^2S^2}{2(1+\alpha I)^2}\right)dt - \kappa t + C_m + In\,I(0).$

where the C_m is called the martingale and $C_m = \int_0^t \frac{\sigma S}{1+\alpha I}dB$, with $C_m(0) = 0$.

If $\sigma^2 > \frac{\beta\mu}{\Lambda}$,

$In\,I \leq \left(\frac{\beta^2}{2\sigma^2} - (\kappa - \alpha + \sigma)\right)t + C_m + InI(0).$

$\frac{InI}{t} \leq -\left(\kappa - \frac{\beta^2}{2\sigma^2}\right) + \frac{C_m}{t} + \frac{InI(0)}{t}.$

if $\lim_{t\to\infty}\frac{C_m}{t} = 0$.

$\lim_{t\to\infty}\frac{InI}{t} \leq -\left(\kappa - \frac{\beta^2}{2\sigma^2}\right) < 0,$

when $\sigma^2 > \frac{\beta^2}{2\kappa,}$, it means

$lim_{\,t\to\infty} I(t) = 0$ nearly surely.

If $\sigma^2 < \frac{\beta\mu}{\Lambda}$

$InI \leq \left(\frac{\beta\Lambda}{\mu} - \frac{\sigma^2\Lambda^2}{2\mu^2} - \kappa\right)t + C_m(t) + InI(0)$.

$\frac{InI}{t} \leq \kappa\left[\frac{\beta\Lambda}{\mu\kappa} - \frac{\sigma^2\Lambda^2}{2\mu^2\kappa} - 1\right] + \frac{C_m}{t} + \frac{InI(0)}{t}$.

$\lim\limits_{t\to+\infty} sup\frac{\ln I}{t} \leq \kappa({R_0}^s - 1)$, then when ${R_0}^s < 1$, we get

$\lim\limits_{t\to\infty} sup\frac{lnI}{t} < 0$

$\lim\limits_{t\to\infty} I(t) = 0$ nearly sure.

$\mathrm{R_o^S} = \mathrm{R_o^d} - \frac{\sigma^2\Lambda^2}{2\mu^2\kappa.} < 1$.

Note that R_o^S is the stochastic threshold number, the human population will be disease-free if $R_o^S < 1$, and disease will present if $\mathrm{R_o^S} > 1$.

5 Numerical results

We demonstrate the numerical solutions of the Eqs. (8) to (10). Song et al. [Song, Miao, Zhang et al. (2018)] have given the values of parameters and presented in Tab. 1 as follows:

Table 1: Parameters values

Parameters	Values (Days)
β	DFE=0.2
	EE=4
α	0.2
γ_1	0.7
γ_2	0.5
μ	0.5
σ_1	0.02
σ	0.5
Λ	0.5

5.1 Stochastic Euler approach

The given approach could be constructed for the Eqs. (8) to (10) as follows:

$$S^{n+1} = S^n + h\left(\Lambda - \frac{\beta S^n I^n}{1+\alpha I^n} + \gamma_1 I^n + \delta R^n - \mu S^n - \sigma\frac{S^n I^n}{1+\alpha I^n}\Delta B_n\right). \quad (11)$$

$$I^{n+1} = I^n + h\left(\frac{\beta S^n I^n}{1+\alpha I^n} - (\mu_1 + \gamma_1 + \gamma_2 + \alpha)I^n + \sigma\frac{S^n I^n}{1+\alpha I^n}\Delta B_n - \sigma_1 I^n \Delta B_n\right). \quad (12)$$

$$R^{n+1} = R^n + h(\gamma_2 I^n - (\mu + \delta)R^n + \sigma_1 I^n \Delta B_n). \quad (13)$$

where *'h'* shows the time step size and ΔB_n means geometric Brownian motion normally distributed in the feasible region Ω i.e., $\Delta B_n \sim N(0,1)$. The simulation of the Eqs. (11) to (13), by manipulating the MATLAB and parameters suggested in Tab. 1.

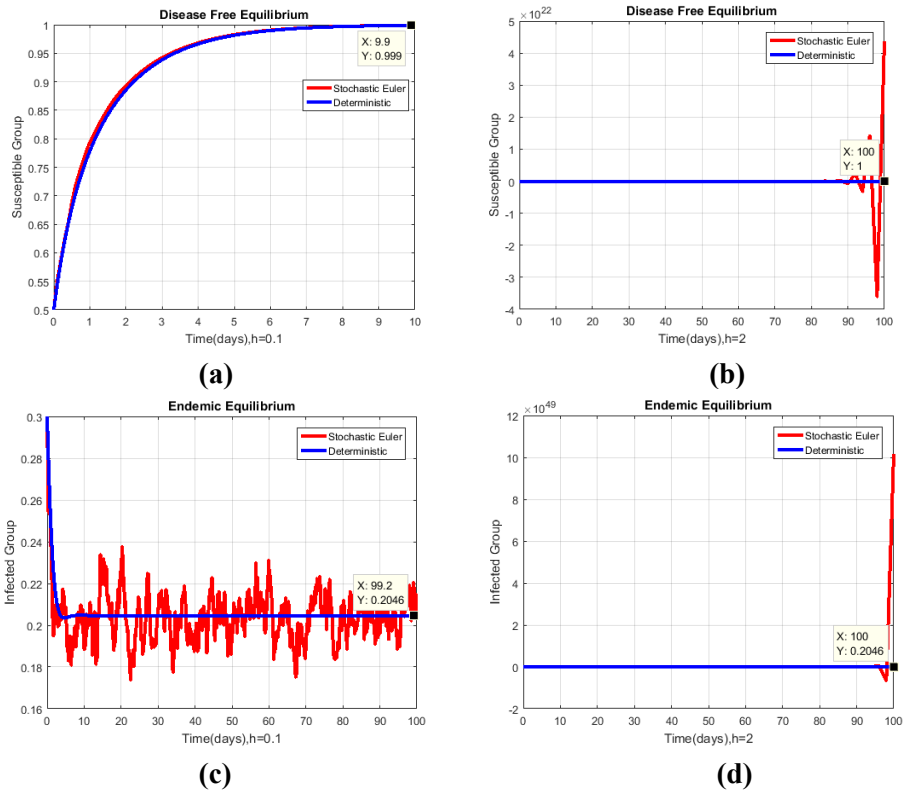


Figure 2: Contrast in the outcomes of stochastic Euler and deterministic (a) Susceptible group at h=0.1 (b) Susceptible group at h=2 (c) Infected group at h=0.1 (d) Infected group at h=2

5.2 Stochastic Runge Kutta approach

The given approach could be constructed for the Eqs. (8) to (10) as follows:

Stage 1

$$E_1 = h\left(\Lambda - \frac{\beta S^n I^n}{1+\alpha I^n} + \gamma_1 I^n + \delta R^n - \mu S^n - \sigma \frac{S^n I^n}{1+\alpha I^n}\Delta B_n\right).$$

$$F_1 = h\left(\frac{\beta S^n I^n}{1+\alpha I^n} - (\mu_1 + \gamma_1 + \gamma_2 + \alpha) I^n + \sigma \frac{S^n I^n}{1+\alpha I^n}\Delta B_n - \sigma_1 I^n \Delta B_n\right).$$

$$G_1 = h(\gamma_2 I^n - (\mu + \delta) R^n + \sigma_1 I^n \Delta B_n).$$

Stage 2

$$E_2 = h\left(\Lambda - \frac{\beta\left(S^n + \frac{E_1}{2}\right)\left(I^n + \frac{F_1}{2}\right)}{1+\alpha\left(I^n + \frac{F_1}{2}\right)} + \gamma_1\left(I^n + \frac{F_1}{2}\right) + \delta\left(R^n + \frac{G_1}{2}\right) - \mu\left(S^n + \frac{E_1}{2}\right) - \sigma \frac{\left(S^n + \frac{E_1}{2}\right)\left(I^n + \frac{F_1}{2}\right)}{1+\alpha\left(I^n + \frac{F_1}{2}\right)}\Delta B_n\right).$$

$$F_2 = h\left(\frac{\beta\left(S^n+\frac{E_1}{2}\right)\left(I^n+\frac{F_1}{2}\right)}{1+\alpha\left(I^n+\frac{F_1}{2}\right)} - (\mu_1+\gamma_1+\gamma_2+\alpha)\left(I^n+\frac{F_1}{2}\right) + \sigma\frac{\left(S^n+\frac{E_1}{2}\right)\left(I^n+\frac{F_1}{2}\right)}{1+\alpha\left(I^n+\frac{F_1}{2}\right)}\Delta B_n - \sigma_1\left(I^n+\frac{F_1}{2}\right)\Delta B_n\right).$$

$$G_2 = h(\gamma_2\left(I^n+\frac{F_1}{2}\right) - (\mu+\delta)\left(R^n+\frac{G_1}{2}\right) + \sigma_1\left(I^n+\frac{F_1}{2}\right)\Delta B_n).$$

Stage 3

$$E_3 = h\left(\Lambda - \frac{\beta\left(S^n+\frac{E_2}{2}\right)\left(I^n+\frac{F_2}{2}\right)}{1+\alpha\left(I^n+\frac{F_2}{2}\right)} + \gamma_1\left(I^n+\frac{F_2}{2}\right) + \delta\left(R^n+\frac{G_2}{2}\right) - \mu\left(S^n+\frac{E_2}{2}\right) - \sigma\frac{\left(S^n+\frac{E_2}{2}\right)\left(I^n+\frac{F_2}{2}\right)}{1+\alpha\left(I^n+\frac{F_2}{2}\right)}\Delta B_n\right).$$

$$F_3 = h\left(\frac{\beta\left(S^n+\frac{E_2}{2}\right)\left(I^n+\frac{F_2}{2}\right)}{1+\alpha\left(I^n+\frac{F_2}{2}\right)} - (\mu_1+\gamma_1+\gamma_2+\alpha)\left(I^n+\frac{F_2}{2}\right) + \sigma\frac{\left(S^n+\frac{E_2}{2}\right)\left(I^n+\frac{F_2}{2}\right)}{1+\alpha\left(I^n+\frac{F_2}{2}\right)}\Delta B_n - \sigma_1\left(I^n+\frac{F_2}{2}\right)\Delta B_n\right).$$

$$G_3 = h(\gamma_2\left(I^n+\frac{F_2}{2}\right) - (\mu+\delta)\left(R^n+\frac{G_2}{2}\right) + \sigma_1\left(I^n+\frac{F_2}{2}\right)\Delta B_n).$$

Stage 4

$$E_4 = h\left(\Lambda - \frac{\beta(S^n+E_3)(I^n+F_3)}{1+\alpha(I^n+F_3)} + \gamma_1(I^n+F_3) + \delta(R^n+G_3) - \mu(S^n+E_3) - \sigma\frac{(S^n+E_3)(I^n+F_3)}{1+\alpha(I^n+F_3)}\Delta B_n\right).$$

$$F_4 = h\left(\frac{\beta(S^n+E_3)(I^n+F_3)}{1+\alpha(I^n+F_3)} - (\mu_1+\gamma_1+\gamma_2+\alpha)(I^n+F_3) + \sigma\frac{(S^n+E_3)(I^n+F_3)}{1+\alpha(I^n+F_3)}\Delta B_n - \sigma_1(I^n+F_3)\Delta B_n\right).$$

$$G_4 = h(\gamma_2(I^n+F_3) - (\mu+\delta)(R^n+G_3) + \sigma_1(I^n+F_3)\Delta B_n).$$

Last stage

$$\left.\begin{aligned} S^{n+1} &= S^n + \frac{1}{6}[E_1+2E_2+2E_3+E_4] \\ I^{n+1} &= I^n + \frac{1}{6}[F_1+2F_2+2F_3+F_4] \\ R^{n+1} &= R^n + \frac{1}{6}[G_1+2G_2+2G_3+G_4] \end{aligned}\right\} \quad (14)$$

where *'h'* shows the time step size and $\Delta B_n \sim N(0,1)$. The simulation of the Eq. (14), by manipulating the MATLAB and parameters suggested in Tab. 1.

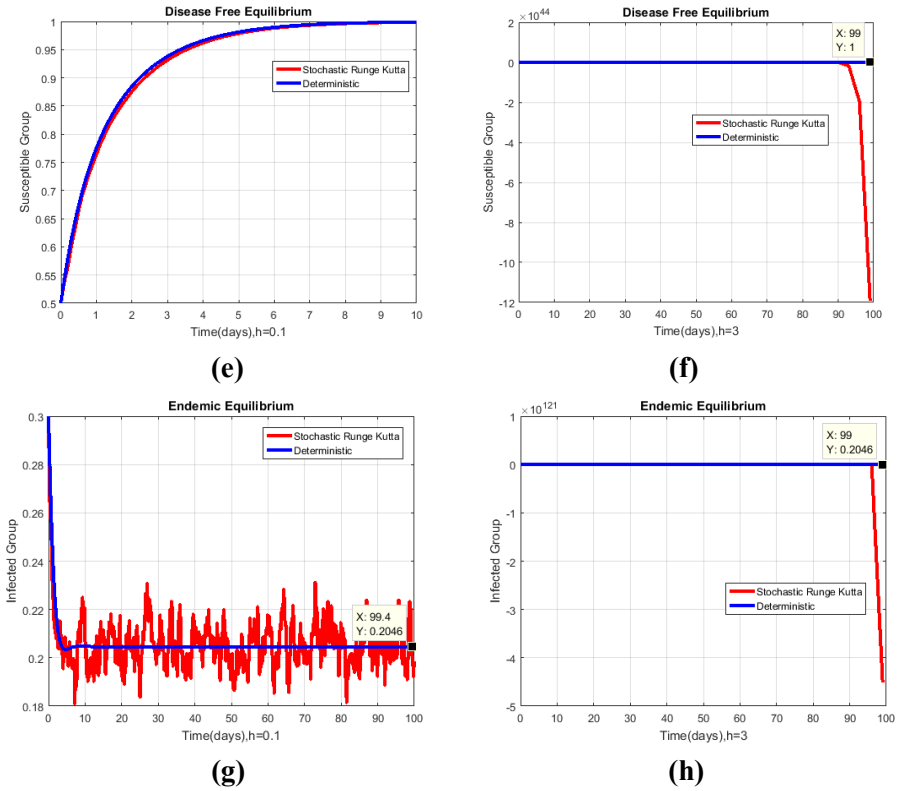


Figure 3: (e) Susceptible group at h=0.1 (f) Susceptible group at h=3 (g) Infected group at h=0.1 (h) Infected group at h=3

5.3 Stochastic NSFD approach

SNSFD could be constructed for the Eqs. (8) to (10) as follows:

$$S^{n+1} = \frac{S^n + h\Lambda + h\gamma_1 I^n + h\delta R^n}{[1 + h\frac{\beta I^n}{1+\alpha I^n} + h\mu + h\sigma I^n \Delta B_n]}. \tag{15}$$

$$I^{n+1} = \frac{I^n + h\frac{\beta S^n I^n}{1+\alpha I^n} + h\sigma\frac{S^n I^n \Delta B_n}{1+\alpha I^n}}{[1 + h(\mu_1 + \gamma_1 + \gamma_2 + \alpha) + h\sigma_1 \Delta B_n]}. \tag{16}$$

$$R^{n+1} = \frac{R^n + h\gamma_2 I^n + h\sigma_1 I^n \Delta B_n}{[1 + h(\mu + \delta)]}. \tag{17}$$

5.4 Convergence analysis

The following theorems could be presented as follows:

Theorem: The Eqs. (15) to (17) has a unique positive solution $(S^n, I^n, R^n) \in R_+^3$ on $n \geq 0$, with any given initial value $(S^n(0), I^n(0), R^n(0)) \in R_+^3$, nearly surely.

Theorem: The section $\Omega = \left\{(S^n, I^n, R^n) \in R_+^3 : S^n \geq 0, I^n \geq 0, R^n \geq 0, S^n + I^n + R^n \leq \frac{\Lambda}{\mu}\right\}$ for all $n \geq 0$ is a translation-invariant for Eqs. (15) to (17).

Proof: Rewriting the Eqs. (15) to (17) below as:

$\frac{S^{n+1}-S^n}{h} = \left(\Lambda - \frac{\beta S^n I^n}{1+\alpha I^n} + \gamma_1 I^n + \delta R^n - \mu S^n - \sigma \frac{S^n I^n}{1+\alpha I^n}\Delta B_n\right)$.

$\frac{I^{n+1}-I^n}{h} = \left(\frac{\beta S^n I^n}{1+\alpha I^n} - (\mu_1 + \gamma_1 + \gamma_2 + \alpha)I^n + \sigma \frac{S^n I^n}{1+\alpha I^n}\Delta B_n - \sigma_1 I^n \Delta B_n\right)$.

$\frac{R^{n+1}-R^n}{h} = h(\gamma_2 I^n - (\mu + \delta)R^n + \sigma_1 I^n \Delta B_n)$.

$\frac{(S^{n+1}+I^{n+1}+R^{n+1})-(S^n+I^n+R^n)}{h} = \Lambda - \mu(S^n + I^n + R^n)$.

$(S^{n+1} + I^{n+1} + R^{n+1}) - (S^n + I^n + R^n) = \Lambda - \mu(S^n + I^n + R^n)$.

$(S^{n+1} + I^{n+1} + R^{n+1}) \leq \frac{\Lambda}{\mu}$.

Theorem: The system Eqs. (15) to (17) has identical equilibria as that of the Eqs. (8) to (10) for all $n \geq 0$.

Proof: For resolving the Eqs. (15) to (17) as follows:

DFE is $D_2 = (\frac{\Lambda}{\mu}, 0, 0)$.

EE is $E_2 = (S^n, I^n, R^n)$.

where,

$S^n = \frac{(\mu_1+\gamma_1+\gamma_2+\alpha)}{\beta}$, $I^n = \frac{\beta\Lambda(\mu+\delta)-(\mu+\delta)\mu(\mu_1+\gamma_1+\gamma_2+\alpha)}{\beta[\mu\mu_1+\mu_1\delta+\gamma_2\mu+\alpha\mu+\alpha\delta]}$,

$R^n = \frac{\gamma_2[\beta\Lambda(\mu+\delta)-(\mu+\delta)\mu(\mu_1+\gamma_1+\gamma_2+\alpha)]}{\beta(\mu+\delta)[\mu\mu_1+\mu_1\delta+\gamma_2\mu+\alpha\mu+\alpha\delta]}$.

Theorem: The eigenvalues of the Eqs. (15) to (17) should lie in unit circle.

Proof: Imagine F, G and H below as:

$F = \frac{S+h\Lambda+h\gamma_1 I+h\delta R}{[1\frac{h\beta I}{1+aI}+h\mu+h\sigma I\Delta B_n]}$

$G = \frac{I+\frac{h\beta SI}{1+aI}+\frac{h\sigma SI\Delta B}{1+aI}}{[1+h(\mu_1+\gamma_1+\gamma_2+\alpha)+h\sigma_1\Delta B_n]}$

$H = \frac{R+h\gamma_2 I+h\sigma_1 I\Delta B_n}{[1+h(\mu+\delta)]}$

$\frac{\partial F}{\partial S} = \frac{1}{[1+\frac{h\beta I}{1+aI}+h\mu+h\sigma I\Delta B_n]}$,

$\frac{\partial F}{\partial I} = \frac{h\gamma_1\left(1+\frac{h\beta I}{1+aI}+h\mu+h\sigma I\Delta B_n\right)-(\frac{h\beta(1+aI)-a(h\beta I)}{(1+aI)^2}+h\sigma\Delta B_n)(S+h\Lambda+h\gamma_1 I+h\delta R)}{[1+h\beta I+h\mu+h\sigma I\Delta B_n]}$,

$\frac{\partial F}{\partial R} = \frac{h\delta}{[1+\frac{h\beta I}{1+aI}+h\mu+h\sigma I\Delta B_n]}$

$\frac{\partial G}{\partial S} = \frac{\frac{h\beta SI}{1+aI}+\frac{h\sigma SI\Delta B_n}{1+aI}}{[1+h(\mu_1+\gamma_1+\gamma_2+\alpha)+h\sigma_1\Delta B_n]}$, $\frac{\partial G}{\partial I} = \frac{\frac{h\beta S(1+aI)-a(h\beta SI)}{(1+aI)^2}+\frac{[h\sigma S(1+aI)-a(h\sigma SI)]\Delta B}{(1+aI)^2}}{[1+h(\mu_1+\gamma_1+\gamma_2+\alpha)+h\sigma_1\Delta B_n]}$, $\frac{\partial G}{\partial R} = 0$.

$\frac{\partial H}{\partial S} = 0$, $\frac{\partial H}{\partial I} = \frac{h\gamma_2+h\sigma_1\Delta B_n}{[1+h(\mu+\delta)]}$, $\frac{\partial H}{\partial R} = \frac{1}{[1+h(\mu+\delta)]}$.

The Jacobean matrix "J" is defined as

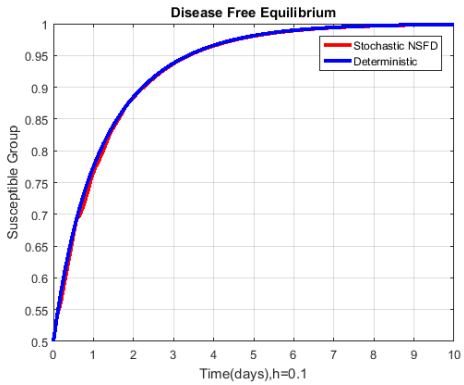
$$J = \begin{bmatrix} \frac{\partial F}{\partial S} & \frac{\partial F}{\partial I} & \frac{\partial F}{\partial R} \\ \frac{\partial G}{\partial S} & \frac{\partial G}{\partial I} & \frac{\partial G}{\partial R} \\ \frac{\partial H}{\partial S} & \frac{\partial H}{\partial I} & \frac{\partial H}{\partial R} \end{bmatrix}$$

Put $D_2 = \left(\frac{\Lambda}{\mu}, 0, 0\right)$ and $R_o^S < 1$, we have

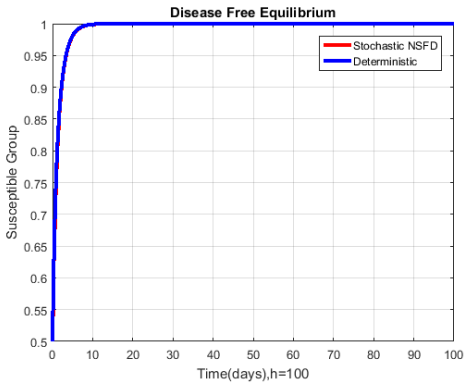
$$J = \begin{bmatrix} \frac{1}{1+h\mu} & \frac{h\gamma_1(1+h\mu) - (h\beta + h\sigma\Delta B_n)(k+h\Lambda)}{[1+h\mu]^2} & \frac{h\delta}{[1+h\mu]} \\ 0 & \frac{h\beta\frac{\Lambda}{\mu} + h\sigma\frac{\Lambda}{\mu}\Delta B_n}{[1+h(\mu_1+\gamma_1+\gamma_2+\alpha)+h\sigma_1\Delta B_n]} & 0 \\ 0 & \frac{h\gamma_2 + h\sigma_1\Delta B_n}{[1+h(\mu+\delta)]} & \frac{1}{[1+h(\mu+\delta)]} \end{bmatrix}$$

The eigenvalues are $\lambda_1 = \frac{1}{1+h\mu} < 1$, $\lambda_2 = \frac{h\beta\Lambda + h\sigma\Lambda\Delta B_n}{\mu[1+h(\mu_1+\gamma_1+\gamma_2+\alpha)+h\sigma_1\Delta B_n]} < 1$, if $R_o^S < 1$, $\lambda_3 = \frac{1}{[1+h(\mu+\delta)]}$.

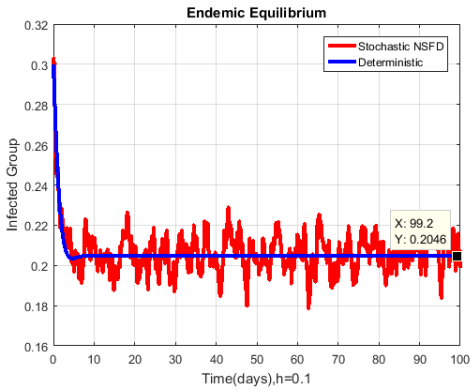
The simulation of the Eqs. (15) to (17), by manipulating the MATLAB and parameters suggested in Tab. 1.



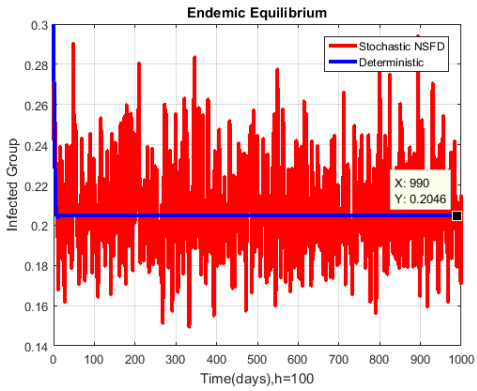
(i)



(j)



(k)

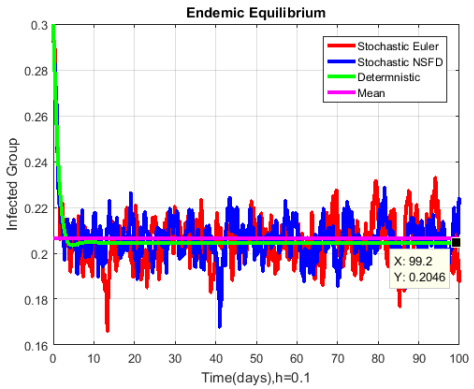


(l)

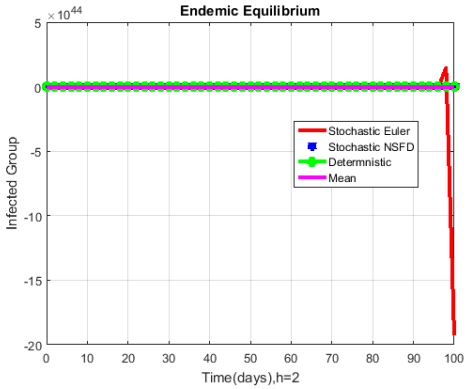
Figure 4: (i) Susceptible group at h=0.1 (j) Susceptible group at h=100 (k) Infected group at h=0.1 (l) Infected group at h=100

5.5 Dissimilarity section

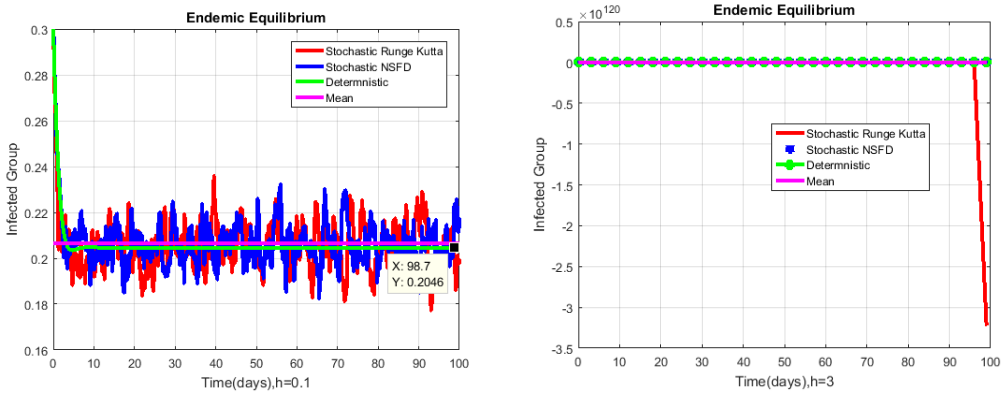
In the following segment, we are going to present the contrast of nominated stochastic nonstandard finite difference (SNSFD) approach and operational stochastic explicit approaches as below:



(m)



(n)



(p) **(q)**

Figure 5: (m) Infected group at h=0.1 (n) Infected group at h=2 (p) Infected group at h=0.1 (q) Infected group at h=3.

5.6 Subpopulations covariance

Here we are to study various sub-populations covariance in the model. The association coefficients are determined, and aftermaths are produced in Tab. 2 below.

Table 2: Correlation coefficient

Sub-Populations	Correlation Coefficient (ρ)	Relationship
(S, I)	-0.9351	Inverse
(I, R)	0.7830	Direct
(R, S)	-0.9390	Inverse

The results in Tab. 2 depicts that the susceptible group and other two sub-communities have a reciprocal relation. It illustrates that there is a rise in the susceptible group with the decline of other sub-populations and ultimately disease-free equilibrium (DFE) will be established by the system.

6 Results and arguments

In Fig. 2, we have drawn the behaviour of a subpopulation of the epidemic model by using stochastic Euler approach. The given approach converges to the untruthful equilibria of the model. We can observe that the approach above is conditionally convergent. In Fig. 3, we have drawn the behaviour subpopulation of the epidemic model by using stochastic Runge Kutta. The given approach converges to the untruthful equilibria of the model. We can observe that the approach above is also conditionally convergent. These stochastic explicit approaches dependent upon the time step parameter and may lose the dynamical properties. In Fig. 4, we have drawn the behaviour of a subpopulation of the epidemic model by using stochastic nonstandard finite difference approach (SNSFD). The given approach converges to the true equilibria of the model. This method is unconditional convergent and works for any time step size. In Fig. 5, we

have shown the effectiveness of stochastic nonstandard finite difference approach (SNSFD) approach with existing stochastic explicit approaches for different time-step sizes. Also, the averages of stochastic approaches are itself deterministic system.

7 Conclusion and future guidelines

The discrete stochastic model and continues model has the same solutions on certain conditions. The first time, the newly developed approach name as stochastic nonstandard finite difference approach (SNSFD) is constructed for the stochastic epidemic model. By using this approach, we can study the dynamics of the disease in the human population over a long period. This scheme is unconditionally convergent approach as compared to stochastic explicit approaches. This approach preserves all dynamical properties of stochastic models like as consistency, stability, positivity and boundedness [Mickens (1994, 2005)] in the stochastic framework. In future, we shall extend this stochastic analysis to all epidemic models of humans, animals and plants.

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