Numerical Control Measures of Stochastic Malaria Epidemic Model

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Abstract: Nonlinear stochastic modeling has significant role in the all discipline of sciences. The essential control measuring features of modeling are positivity, boundedness and dynamical consistency. Unfortunately, the existing stochastic methods in literature do not restore aforesaid control measuring features, particularly for the stochastic models. Therefore, these gaps should be occupied up in literature, by constructing the control measuring features numerical method. We shall present a numerical control measures for stochastic malaria model in this manuscript. The results of the stochastic model are discussed in contrast of its equivalent deterministic model. If the basic reproduction number is less than one, then the disease will be in control while its value greater than one shows the perseverance of disease in the population. The standard numerical procedures are conditionally convergent. The propose method is competitive and preserve all the control measuring features unconditionally. It has also been concluded that the prevalence of malaria in the human population may be controlled by reducing the contact rate between mosquitoes and humans. The awareness programs run by world health organization in developing countries may overcome the spread of malaria disease.

Keywords: Malaria disease model, stochastic modelling, stochastic methods, convergence.

1 Literature survey

Malaria is a fatal infectious disease and is given a particular place in the previous records of human history. Human of the stone age to ancient Chinese are the affectees. In the 20th

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century, about 300 million people were affected by malaria. It is affecting human living in tropical regions sub-Saharan of Africa, Asia and the Amazon baron. Forty per cent of the human population in these areas is still under threat due to malaria. Ancient writing's and artefacts have proved the long period of malaria effect. Clay tablets with engravings from Mesopotamia are clear evidence of malaria hostility. The historians had declared malaria as "king of diseases" in the redic period. In 270 B.C. the norm of malaria was widespread and brutal. It was declared as tertian (every 3rd day) and quartan (every 4th day) fever along with revelling spleen. The Chinese concerned malaria's headache, chills and fevers as three friends. The Greek poet Homer (750 B.C.) relished the taste of malaria fatalism in their lifetimes and had mentioned it in their works and declared it as Sarus, "The dog star as desolation". The arrival of malaria to the Rome in the 1st century A.D. proved to be turnery point in the history of Europe. Otieno et al. [Otieno, Koske and Mustiso (2016) have streamlined transmission of malaria from African forests the mediterranean, align the nile to fertile crescent of Egypt. Traore et al. [Traore, Sangare and Traore (2017)] have presented different kind of mathematical modeling of malaria epidemic. The visitors of these areas had described the poor, shabby conditions of these people and strongly condemned on the fragility of the population. Population growth in China forced people to become settled in semitropical zones which are malaria promoting areas. Indus valley is hot and dry, so its habitants migrated towards the wet Ganges where they were plagued by malaria and other mosquito and water-borne diseases. European travellers, conquistadores and migrants transmitted plasmodium and vivax (malaria viruses). African slaves were another source of transmission of malaria (falciparum) to the rest of the world. The ships and boats Europeans shifted their slaves to their homelands thus carried malaria with them also. The Europeans settlers and native Americans and their lineage were more susceptible. Olaniyi et al. [Olaniyi and Obabiyi (2013)] have presented deforestation along with met agriculture favoured breeding of female mosquito anopheles which in the chief transported of plasmodium. Malaria affecting the USA until the early 20th Century. Civil war soldiers were also its sufferers in 1862. Then it was transmitted to California and further spreader across the continent. Huge expenses were done to take measures in order to control the spread of malaria. It rained both physical and economic health of the entire region. USA claimed its complete wipe out from societies, but again it was noticed during World War II. In pacific campaigns more soldiers killed by malaria instead of enemy attack. Agvingi et al. [Agyingi, Ngwa and Wiandt (2016)] have given another idea of drug-resistant type of malaria was discovered as a big challenge for both biologists and administrators in the war of Vietnam. Despite all these efforts, malaria remained to inflict upon us for all times, past or present. As an essential pathogen, it was an obstacle to Africa's colonization. It was given the name of killing fever. Whenever the Europeans had tried to build their out pasts on the continent, they have repelled time and again by malaria, vellow fever and other tropical monsters. By the 18th century, it gained the name of the white man's grave. The most worried were biologists who were effortless in all aspects. They were studying human blood cells and found sickled shaped haemoglobin in the blood of malaria patients. This disease is caused by single-celled parasitic microorganisms belonging to plasmodium group. It is an infectious disease introduced by a mosquito bite carrying plasmodium into human blood. Rainy season provides a suitable

environment for malaria spread in tropical and semi-tropical zones of the world. Plasmodium is injected into human blood through mosquito bite and effects most vital body organ the liver. The virus is multiplied, there comes back into the bloodstream and destroys red blood cells. The process further leads to a cascade of reactions and symptoms start appearing. Symptoms start appearing within two weeks, but in some cases the parasite becomes dormant and appear after some time. Its initial symptoms are similar to flu, so a blood test is necessary for confirmation. After confirmation through blood test proper care and good hygiene around the patient of malaria is to be maintained, especially protection of patient from mosquitoes. If someone is travelling into malariaprone areas, special instructions should be taken from doctors and must carry mosquito repellents and necessary medicine. The standard drugs which are effectively used in malarial situations are chloroquine, malarone and mefloquine. Malaria is wholly cured able disease by following the set parameters by NGOs and other health organizations. The use of insecticide-treated nets (ITNs), indoor residual sprays (IRS) and most importantly to uproot entirely the nurseries of mosquito larva. Chemoprevention for the most vulnerable population is particularly pregnant women and infants. According to analysis, almost nighty-nine countries and territories are where malaria spread is standard out of the eighty have been safe sided now through tiring efforts, and nineteen countries are in pre-elimination and elimination phase. To get control over the spread of malaria disease, it was necessary to get knowledge of mosquito populations and how to control them. For this sake scientists, through a series of different experiments, gathered and analyzed statistics. Mathematicians made use of these statistics to develop the deterministic and stochastic SEIR models which make it convenient to know the dynamics and transmission of malaria involving variables in human and mosquito populations. Mathematicians believed that the dynamics of disease is governed by a threshold parameter R_0 . If $R_0 > I$, then disease will persist in a population and eventually it will be in endemic equilibrium. If $R_0 < l$, the disease will disappear from population and another steady state called disease-free equilibrium (DFE) becomes stable. Mathematical modeling helps giving a complete insight into such epidemic diseases. The construction of model along with other statistics and possible simulations help in analyzing the sensitivity of malaria transmission and to get control over its spread. With the aid of these efforts, scientists have become resourceful in devising tools and mechanisms, which have helped us implement the outcomes to get a check on malaria transmission properly. These models are studied from different angles. These models involve the use of nonlinear initial value problems along with differential equations which may or may not be according to our expectations. Arif et al. [Arif, Raza, Shatanawi et al. (2019)] have found the existing techniques in literature can bring about deceptive chaos and deceitful fluctuations for certain passions of the discretization limitations. These facts make them less dependent while solving such models and when the question of human health across the world is considered more reliable tact is like the making of stochastic epidemic models are to be favoured by experts. The environmental fluctuations are strongly dependent on the transmission of diseases in population. Therefore, the occurrence of an epidemic in a population is a random process. Guo et al. [Guo, Cai, Zhang et al. (2018)] have found more realistic strategy to understand environmental stochasticity is stochastic epidemic models. Stochastic epidemic models are generally governed by stochastic

differential equations (SDEs). These equations are highly non-linear and do not have analytic solutions. Allen et al. [Allen, Allen, Arciniega et al. (2008)] have used the different numerical methods to handle such problems. Pierret [Pierret (2015)] have presented essential control measures of the initial system. Raza et al. [Raza, Arif and Rafiq (2019)] have given the idea for construction, implementation and analysis of a stochastic nonstandard finite difference (SNSFD) method. The remaining paper is organized as follows. In Section 2, we discuss the deterministic malaria model and its steady states. In Section 3, we describe the construction of stochastic differential equations. In Section 4, we present different numerical methods and compared the results with deterministic parts. In this section, we also discuss the convergence of proposed method. In Section 6, we shall give conclusions and coming guidelines.

2 Deterministic malaria model

In this part, we have considered the deterministic malaria model. For any time t, the specification of variables are S(t) characterizes the susceptible humans, E(t) characterizes the exposed humans, I(t) characterizes the infected humans and R(t) characterizes the removed humans. The transmission of malaria model as shown in Fig. 1.



Figure 1: Movement map of malaria disease in population

The model transmission rates are labelled as Λ (denote the recruitment rate of susceptible humans), β (denote the infectious rate of humans), α_1 (denote the rate of exposed humans who are infectious), α_2 (denote removal rate of humans), μ (denotes the natural death rate of humans) and δ (denotes the induce death rate of humans). The ordinary differential equations of model as follows:

$$\frac{dS(t)}{dt} = \Lambda - \beta S(t)I(t) - \mu S(t).$$
(1)

$$\frac{dE(t)}{dt} = \beta S(t)I(t) - (\alpha_1 + \mu)E(t).$$
(2)

$$\frac{dI(t)}{dt} = \alpha_1 E(t) - (\alpha_2 + \mu + \delta)I(t).$$
(3)

$$\frac{dR(t)}{dt} = \alpha_2 I(t) - \mu R(t). \tag{4}$$

where, the region for Eqs. (1) to (4) is $\Omega = \{(S, E, I, R): S + E + I + R \le \frac{\Lambda}{\mu}, S \ge 0, E \ge 0, I \ge 0, R \ge 0\}$. Here, the region Ω is called positive invariant. So, the solution of the Eqs. (1) to (4) lies in this region Ω .

2.1 Equilibria of malaria model

There are two equilibria of the Eqs. (1) to (4) as follows:

DFE is
$$D = \left(\frac{\Lambda}{\mu}, 0, 0, 0\right)$$
.
EE is $E = (S_1, E_1, I_1, R_1)$.
 $S_1 = \frac{(\alpha_1 + \mu)(\alpha_2 + \mu + \delta)}{\alpha_1 \beta}, E_1 = \frac{\Lambda \alpha_1 - \mu(\alpha_1 + \mu)(\alpha_2 + \mu + \delta)}{\alpha_1 \beta(\alpha_1 + \mu)}, I_1 = \frac{\Lambda \alpha_1 - \mu(\alpha_1 + \mu)(\alpha_2 + \mu + \delta)}{\beta(\alpha_1 + \mu)(\alpha_2 + \mu + \delta)},$
 $R_1 = \frac{\alpha_2}{\mu} \left(\frac{\Lambda \alpha_1 - \mu(\alpha_1 + \mu)(\alpha_2 + \mu + \delta)}{\beta(\alpha_1 + \mu)(\alpha_2 + \mu + \delta)}\right), R_0 = \frac{\beta \Lambda \alpha_1}{\mu(\alpha_1 + \mu)(\alpha_2 + \mu + \delta)}.$

where R_0 is called basic reproduction number and this number has an active role in disease dynamics. If $R_0 < 1$ means this approach helps to control the disease in the human population. If $R_0 > 1$ means the disease is endemic in the human population.

3 Stochastic malaria model

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Let $M = [S, E, I, R]^T$. On the way to arrange the stochastic differential equations (SDEs) from the Eqs. (1) to (4). We estimate the expectations $E^*[M_i]$ and $E^*[M_iM_i^T]$.

Transition	Probabilities
$M_1 = [1,0,0,0]^T$	$P_1 = \wedge \Delta t.$
$M_2 = [-1, 1, 0, 0]^T$	$P_2 = \beta S(t) I(t) \Delta t.$
$M_3 = [-1,0,0,0]^T$	$P_3 = \mu S(t) \Delta t.$
$M_4 = [0, -1, 1, 0]^T$	$P_4 = \alpha_1 E(t) \Delta t.$
$M_5 = [0, -1, 0, 0]^T$	$P_5 = \mu E(t) \Delta t.$
$M_6 = [0,0,-1,1]^T$	$P_6 = \alpha_2 I(t) \Delta t.$
$M_7 = [0,0,-1,0]^T$	$P_7 = (\mu + \delta)I(t)\Delta t.$
$M_8 = [0,0,0,-1]^T$	$P_8 = \mu R(t) \Delta t.$

 Table 1: Possibilities in malaria model

$$E [M_{i}] = \sum_{i=1}^{n} P_{i} M_{i}.$$
Expectation = $E^{*}[M_{i}] = \begin{bmatrix} \wedge -\beta S(t)I(t) - \mu S(t) \\ \beta S(t)I(t) - (\alpha_{1} + \mu)E(t) \\ \alpha_{1}E(t) - (\alpha_{2} + \mu + \delta)I(t) \\ \alpha_{2}I(t) - \mu R(t) \end{bmatrix} \Delta t.$

$$Var = \sum_{i=1}^{8} P_{i} M_{i} M_{i}^{T}$$

$$= \begin{bmatrix} \wedge +\beta S(t)I(t) + \mu S(t) & -\beta S(t)I(t) \\ -\beta S(t)I(t) & \beta S(t)I(t) + \alpha_{1}E(t) + \mu E(t) & -\alpha_{1}E(t) & \mathbf{0} \\ \mathbf{0} & -\alpha_{1}E(t) & \alpha_{1}E(t) + \alpha_{2}I(t) + (\mu + \delta)I(t) & -\alpha_{2}I(t) \\ \mathbf{0} & \mathbf{0} & -\alpha_{2}I(t) & \alpha_{2}I(t) + \mu R(t) \end{bmatrix} \Delta t$$

If we define drift = $\mathcal{X}(M(t), t) = \frac{E^*[M_i]}{\Delta t}$ and diffusion= $\mathcal{Y}(M(t), t) = \sqrt{\frac{E^*[M_iM_i^T]}{\Delta t}}$, then $dM(t) = \mathcal{X}(M(t), t)dt + \mathcal{Y}(M(t), t)dB(t).$ (5) with $M(0) = M_0 = [0.4, 0.3, 0.2, 0.1]^T$, $0 \le t \le T$ and B(t) is the Brownian motion.

3.1 Euler maruyama method

Rahman et al. [Rahman, Osman and Adu (2017)] have given the parameters values for the numerical result of Eq. (5) reported in Tab. 2.

	Values		
Parameters	DFE	EE	
μ	0.5	0.5	
α_1	0.3	0.3	
α2	0.35	0.35	
Λ	0.5	0.5	
β	1.001	3.001	
δ	0.010	0.010	
σ_1	0.09	0.09	
σ_2	0.08	0.08	
σ_3	0.07	0.07	
σ_4	0.05	0.05	

Table 2: Values	of parameters
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The Euler Maruyama method of Eq. (5) as follows:

$M_{n+1} = M_n + \mathcal{X}(M_n, t)\Delta t + \mathcal{Y}(M_n, t)\Delta B_n.$

But, the time step size is denoted by Δt and $\Delta B_n \sim N[\mathcal{X}(M, t), \mathcal{Y}(M, t)]$. The disease-free equilibria (DFE) is D = (1, 0, 0, 0) if the basic reproduction number $R_0 = 0.4365 < 1$. The endemic equilibria (EE) is E = (0.7642, 0.1474, 0.05141, 0.03599) if $R_0 = 1.3086 > 1$. Allen et al. [Allen and Burgin (2000)] have also found that the mean of stochastic outputs is approximately equal to the deterministic results as presented in Fig. 2.





Figure 2: (a) Converges behavior of compartment at h=0.1 (b) Diverges behavior of compartment at h=1 (c) First run for exposed humans at h=0.001 (d) Second run for exposed humans at h=1

4 Non-parametric perturbation of model

Rafiq et al. [Rafiq, Raza, Iqbal et al. (2019)] have presented the idea to introduced the non-parametric perturbation term. See Eqs. (1) to (4) as follows:

$$dS(t) = \left(\Lambda - \beta S(t)I(t) - \mu S(t)\right)dt + \sigma_1 dB_1(t)S(t).$$
⁽⁷⁾

$$dE(t) = \left(\beta S(t)I(t) - (\alpha_1 + \mu)E(t)\right)dt + \sigma_2 dB_2(t)E(t).$$
(8)

$$dI(t) = (\alpha_1 E(t) - (\alpha_2 + \mu + \delta)I(t))dt + \sigma_3 dB_3(t)I(t).$$
(9)

$$dR(t) = \left(\alpha_2 I(t) - \mu R(t)\right) dt + \sigma_4 dB_4(t) R(t).$$
⁽¹⁰⁾

where $\sigma_1, \sigma_2, \sigma_3$ and σ_4 are stochastic perturbations of each state variable and $B_m(t)$, (m = 1, 2, 3, 4) is the autonomous Brownian motions. Due to the non-differentiability term of Brownian motions, these equations do not have the exact solutions.

4.1 Stochastic Euler method

The Eqs. (7) to (10) in this method as follows: $S^{n+1}(t) = S^{n}(t) + h[\Lambda -\beta S^{n}(t)I^{n}(t) - \mu S^{n}(t) + \sigma_{1}\Delta B_{1}S^{n}(t)]. \qquad (11)$ $E^{n+1}(t) = E^{n}(t) + h[\beta S^{n}(t)I^{n}(t) - (\alpha_{1} + \mu)E^{n}(t) + \sigma_{2}\Delta B_{2}E^{n}(t)]. \qquad (12)$ $I^{n+1}(t) = I^{n}(t) + h[\alpha_{1}E^{n}(t) - (\alpha_{2} + \mu + \delta)I^{n}(t) + \sigma_{3}\Delta B_{3}I^{n}(t)]. \qquad (13)$

$$R^{n+1}(t) = R^{n}(t) + h[\alpha_{2}I^{n}(t) - \mu R^{n}(t) + \sigma_{4}\Delta B_{4}R^{n}(t)]$$
(14)

We simulate the results and the constants values presented in Tab. 2, by using the MATLAB program and $\Delta B_n \sim N(0,1)$, n = 1, 2, 3, 4.



Figure 3: (a) Converges behavior of compartment at h=0.1 (b) Diverges behavior of compartment when h=2 (c) Exposed humans at EE Point for h=0.1 (d) Diverges behavior of exposed humans when h=1

4.2 Stochastic Runge Kutta method

The Eqs. (7) to (10) in this method as follows: First stage $K_{1} = h[\Lambda - \beta S^{n}(t)I^{n}(t) - \mu S^{n}(t) + \sigma_{1}\Delta B_{1}S^{n}(t)].$ $M_{1} = h[\beta S^{n}(t)I^{n}(t) - (\alpha_{1} + \mu)E^{n}(t) + \sigma_{2}\Delta B_{2}E^{n}(t)].$ $N_{1} = h[\alpha_{1}E^{n}(t) - (\alpha_{2} + \mu + \delta)I^{n}(t) + \sigma_{3}\Delta B_{3}I^{n}(t)].$ $L_{1} = h[\alpha_{2}I^{n}(t) - \mu R^{n}(t) + \sigma_{4}\Delta B_{4}R^{n}(t)].$ Second stage $K_{2} = h[\Lambda - \beta \left(S^{n}(t) + \frac{K_{1}}{2}\right) \left(I^{n}(t) + \frac{N_{1}}{2}\right) - \mu \left(S^{n}(t) + \frac{K_{1}}{2}\right) + \sigma_{1}\Delta B_{1}(S^{n}(t) + \frac{K_{1}}{2})].$ $M_{2} = h \left[\beta \left(S^{n}(t) + \frac{K_{1}}{2}\right) \left(I^{n}(t) + \frac{N_{1}}{2}\right) - (\alpha_{1} + \mu) \left(E^{n}(t) + \frac{M_{1}}{2}\right) + \sigma_{2}\Delta B_{2}(E^{n}(t) + \frac{M_{1}}{2})\right].$ $N_{2} = h[\alpha_{1}(E^{n}(t) + \frac{M_{1}}{2}) - (\alpha_{2} + \mu + \delta)(I^{n}(t) + \frac{N_{1}}{2}) + \sigma_{3}\Delta B_{3}(I^{n}(t) + \frac{N_{1}}{2})].$ $L_{2} = h[\alpha_{2}(I^{n}(t) + \frac{N_{1}}{2}) - \mu(R^{n}(t) + \frac{L_{1}}{2}) + \sigma_{4}\Delta B_{4}(R^{n}(t) + \frac{L_{1}}{2})].$ Third stage

$$\begin{split} K_{3} &= h \left[\wedge -\beta \left(S^{n}(t) + \frac{K_{2}}{2} \right) (I^{n}(t) + \frac{N_{2}}{2}) - \mu (S^{n}(t) + \frac{K_{2}}{2}) + \sigma_{1} \Delta B_{1} (S^{n}(t) + \frac{K_{2}}{2}) \right]. \\ M_{3} &= h \left[\beta \left(S^{n}(t) + \frac{K_{2}}{2} \right) \left(I^{n}(t) + \frac{N_{2}}{2} \right) - (\alpha_{1} + \mu) (E^{n}(t) + \frac{M_{2}}{2}) + \sigma_{2} \Delta B_{2} (E^{n}(t) + \frac{M_{2}}{2}) \right]. \\ N_{3} &= h \left[\alpha_{1} (E^{n}(t) + \frac{M_{2}}{2}) - (\alpha_{2} + \mu + \delta) (I^{n}(t) + \frac{N_{2}}{2}) + \sigma_{3} \Delta B_{3} (I^{n}(t) + \frac{N_{2}}{2}) \right]. \\ L_{3} &= h \left[\alpha_{2} (I^{n}(t) + \frac{N_{2}}{2}) - \mu (R^{n}(t) + \frac{L_{2}}{2}) + \sigma_{4} \Delta B_{4} (R^{n}(t) + \frac{L_{2}}{2}) \right]. \end{split}$$
Fourth stage

$$\begin{split} &K_4 = h[\Lambda - \beta(S^n(t) + K_3)(I^n(t) + N_3) - \mu(S^n(t) + K_3) + +\sigma_1 \Delta B_1(S^n(t) + K_3)].\\ &M_4 = h[\beta(S^n(t) + K_3)(I^n(t) + N_3) - (\alpha_1 + \mu)(E^n(t) + M_3) + \sigma_2 \Delta B_2(E^n(t) + M_3)].\\ &N_4 = h[\alpha_1(E^n(t) + M_3) - (\alpha_2 + \mu + \delta)(I^n(t) + N_3) + \sigma_3 \Delta B_3(I^n(t) + N_3)].\\ &L_4 = h[\alpha_2(I^n(t) + N_3) - \mu(R^n(t) + L_3) + \sigma_4 \Delta B_4(R^n(t) + L_3)].\\ &\text{Final stage} \end{split}$$

$$S^{n+1}(t) = S^{n}(t) + \frac{1}{6}[K_{1} + 2K_{2} + 2K_{3} + K_{4}]$$

$$E^{n+1}(t) = E^{n}(t) + \frac{1}{6}[M_{1} + 2M_{2} + 2M_{3} + M_{4}]$$

$$I^{n+1}(t) = I^{n}(t) + \frac{1}{6}[N_{1} + 2N_{2} + 2N_{3} + N_{4}]$$

$$R^{n+1}(t) = R^{n}(t) + \frac{1}{6}[L_{1} + 2L_{2} + 2L_{3} + L_{4}]$$
(15)

We simulate the results and the constants values presented in Tab. 2, by using the MATLAB program and $\Delta B_n \sim N(0,1)$, n = 1, 2, 3, 4.





Figure 4: (a) Converges behavior of compartment when h=0.1 (b) Diverges behavior of compartment when h=2 (c) Exposed humans when h=0.1 (d) Diverges behavior of exposed humans when h=2

4.3 Stochastic NSFD method

Using finite difference approximations for continuous derivatives and non-local approximations of state variables, we rewrite the Eqs. (11) to (14) as follows:

$$\begin{split} S^{n+1}(t) &= S^{n}(t) + h \wedge -h\beta S^{n+1}(t)I^{n}(t) - h\mu S^{n+1}(t) + h\sigma_{1}\Delta B_{1}S^{n}(t).\\ E^{n+1}(t) &= E^{n}(t) + h\beta S^{n}(t)I^{n}(t) - h(\alpha_{1} + \mu)E^{n+1}(t) + h\sigma_{2}\Delta B_{2}E^{n}(t).\\ I^{n+1}(t) &= I^{n}(t) + h\alpha_{1}E^{n}(t) - h(\alpha_{2} + \mu + \delta)I^{n+1}(t) + h\sigma_{3}\Delta B_{3}I^{n}(t).\\ R^{n+1}(t) &= R^{n}(t) + h\alpha_{2}I^{n}(t) - h\mu R^{n+1}(t) + h\sigma_{4}\Delta B_{4}R^{n}(t). \end{split}$$

Then by finding the equations in form of $S^{n+1}(t)$, $E^{n+1}(t)$, $I^{n+1}(t)$ and $R^{n+1}(t)$ as follows:

$$S^{n+1}(t) = \frac{S^{n}(t) + h + h \sigma_1 S(t) \Delta B_1}{(1 + h \beta I^n(t) + h \mu)}.$$
(16)

$$E^{n+1}(t) = \frac{E^{n}(t) + h\beta S^{n}(t)I^{n}(t) + h\sigma_{2}E(t)\Delta B_{2}}{1 + h(\alpha_{1} + \mu)}.$$
(17)

$$I^{n+1}(t) = \frac{I^{n}(t) + h\alpha_{1}E^{n}(t) + h\sigma_{3}I(t)\Delta B_{3}}{1 + h(\alpha_{2} + \mu + \delta)}.$$
(18)

$$R^{n+1}(t) = \frac{R^{n}(t) + h\alpha_2 I^{n}(t) + h\sigma_4 R(t)\Delta B_4}{1 + h\mu}.$$
(19)

We simulate the results and the constants values presented in Tab. 2, by using the MATLAB program and $\Delta B_n \sim N(0, 1)$, n = 1, 2, 3, 4.

4.4 Convergence analysis

For this we shall satisfy the following theorems as follows:

Theorem: For any given initial value $(S^n(0), E^n(0), I^n(0), R^n(0)) \in \mathbb{R}^4_+$, Eqs. (16) to (19) has a unique positive solution $(S^n(t), E^n(t), I^n(t), R^n(t)) \in \mathbb{R}^4_+$ on $n \ge 0$, nearly sure.

Theorem: The region $\Omega = \left\{ (S^n(t), E^n(t), I^n(t), R^n(t)) \in R^4_+ : S^n(t) \ge 0, E^n(t) \ge 0 \right\}$ $0, I^{n}(t) \ge 0, R^{n}(t) \ge 0, S^{n}(t) + E^{n}(t) + I^{n}(t) + R^{n}(t) \le \frac{h}{n}$ for all $n \ge 0$ is a optimistic invariant set for Eqs. (16) to (19). Proof: The Eqs. (16) to (19) as follows: $S^{n+1}(t) = S^{n}(t) + h \wedge -h\beta S^{n+1}(t)I^{n}(t) - h\mu S^{n+1}(t) + h\sigma_{1}\Delta B_{1}S^{n}(t).$ $E^{n+1}(t) = E^{n}(t) + h\beta S^{n}(t)I^{n}(t) - h(\alpha_{1} + \mu)E^{n+1}(t) + h\sigma_{2}\Delta B_{2}E^{n}(t).$ $I^{n+1}(t) = I^{n}(t) + h\alpha_{1}E^{n}(t) - h(\alpha_{2} + \mu + \delta)I^{n+1}(t) + h\sigma_{3}\Delta B_{3}I^{n}(t).$ $R^{n+1}(t) = R^{n}(t) + h\alpha_{2}I^{n}(t) - h\mu R^{n+1}(t) + h\sigma_{4}\Delta B_{4}R^{n}(t).$ $\frac{S^{n+1}(t)-S^{n}(t)}{h} + \frac{E^{n+1}(t)-E^{n}(t)}{h} + \frac{I^{n+1}(t)-I^{n}(t)}{h} + \frac{R^{n+1}(t)-R^{n}(t)}{h} = \Lambda -\beta S^{n+1}(t)I^{n}(t) - \mu S^{n+1}(t) + \sigma_{1}\Delta B_{1}S^{n}(t) + \beta S^{n}(t)I^{n}(t) - (\alpha_{1}+\mu)E^{n+1}(t) + \sigma_{2}\Delta B_{2}E^{n}(t) + \mu S^{n+1}(t) + \sigma_{2}\Delta B_{2}E^{n}(t) + \sigma_{2}\Delta$ $\alpha_{1}E^{n}(t) - (\alpha_{2} + \mu + \delta)I^{n+1}(t) + \sigma_{3}\Delta B_{3}I^{n}(t) + \alpha_{2}I^{n}(t) - \mu R^{n+1}(t) + \sigma_{4}\Delta B_{4}R^{n}(t)$ $\frac{(S^{n+1}(t)+E^{n+1}(t)+I^{n+1}(t)+R^{n+1}(t))-(S^{n}(t)+E^{n}(t)+I^{n}(t)+R^{n}(t))}{h} = \Lambda -\mu(S^{n}(t)+E^{n}(t)+E^{n}(t)+R^{n}(t))$ $I^{n}(t) + R^{n}(t)) + \sigma_{1}\Delta B_{1}S^{n}(t) + \sigma_{2}\Delta B_{2}E^{n}(t) + \sigma_{3}\Delta B_{3}I^{n}(t) + \sigma_{4}\Delta B_{4}R^{n}(t)$ $(S^{n+1}(t) + E^{n+1}(t) + I^{n+1}(t) + R^{n+1}(t)) - (S^n(t) + E^n(t) + I^n(t) + R^n(t)) = h \wedge$ $-h\mu(S^{n}(t) + E^{n}(t) + I^{n}(t) + R^{n}(t)) + h[\sigma_{1}\Delta B_{1}S^{n}(t) + \sigma_{2}\Delta B_{2}E^{n}(t) +$ $\sigma_3 \Delta B_3 I^n(t) + \sigma_4 \Delta B_4 R^n(t)$]. $(S^{n+1}(t) + E^{n+1}(t) + I^{n+1}(t) + R^{n+1}(t)) \le \frac{h}{\mu} + h[\sigma_1 \Delta B_1 S^n(t) + \sigma_2 \Delta B_2 E^n(t) + \sigma_2 A^n(t) +$ $\sigma_3 \Delta B_3 I^n(t) + \sigma_4 \Delta B_4 R^n(t)$].

nearly sure.

Theorem: The discrete Eqs. (16) to (19) has the same equilibria as continuous Eqs. (7) to (10) for all $n \ge 0$.

Proof: The two states of model as follows:

DFE is
$$D = \left(\frac{\Lambda}{\mu}, 0, 0, 0\right)$$
.
EE is $E = (S_1^n, E_1^n, I_1^n, R_1^n)$.
 $S_1^n = \frac{(\alpha_1 + \mu - \sigma_2 \Delta B_2)(\alpha_2 + \mu + \delta - \sigma_3 \Delta B_3)}{\alpha_1 \beta}, E_1^n = \frac{\Lambda \alpha_1 + (\sigma_3 \Delta B_3 - \mu)(\alpha_1 + \mu - \sigma_2 \Delta B_2)(\alpha_2 + \mu + \delta - \sigma_3 \Delta B_3)}{\alpha_1 \beta (\alpha_1 + \mu - \sigma_2 \Delta B_2)(\alpha_2 + \mu + \delta - \sigma_3 \Delta B_3)},$
 $I_1^n = \frac{\Lambda \alpha_1 + (\sigma_3 \Delta B_2 - \mu)(\alpha_1 + \mu - \sigma_2 \Delta B_2)(\alpha_2 + \mu + \delta - \sigma_3 \Delta B_3)}{\beta (\alpha_1 + \mu - \sigma_2 \Delta B_2)(\alpha_2 + \mu + \delta - \sigma_3 \Delta B_3)},$
 $R_1^n = \frac{\alpha_2}{(\mu - \sigma_4 \Delta B_4)} \left(\frac{\Lambda \alpha_1 + (\sigma_3 \Delta B_2 - \mu)(\alpha_1 + \mu - \sigma_2 \Delta B_2)(\alpha_2 + \mu + \delta - \sigma_3 \Delta B_3)}{\beta (\alpha_1 + \mu - \sigma_2 \Delta B_2)(\alpha_2 + \mu + \delta - \sigma_3 \Delta B_3)}\right).$

Theorem: Eigen values of Eqs. (16) to (19) for both equilibria of model should lie unit radius of circle for all $n \ge 0$.

Proof: We consider the Eqs. (16) to (19) as:

$$F = \frac{S(t)+h\wedge +h\sigma_1 S(t)\Delta B_1}{1+h\beta I(t)+h\mu}, G = \frac{E(t)+h\beta S(t)I(t)+h\sigma_2 E(t)\Delta B_2}{1+h\alpha_1+h\mu}, H = \frac{I(t)+h\alpha_1 E(t)+h\sigma_3 I(t)\Delta B_3}{1+h(\alpha_2+\mu+\beta)}, K = \frac{R+h\alpha_2 I(t)+h\sigma_4 R\Delta B_4}{1+h\mu}.$$

$$\begin{split} \frac{\partial F}{\partial S} &= \frac{1+h\sigma_1\Delta B_1}{1+h\beta I(t)+h\mu}, \frac{\partial F}{\partial E} = 0, \frac{\partial F}{\partial I} = \frac{-(S(t)+h\wedge h+\sigma_1S(t(\Delta B_1)h\beta)}{(1+h\beta I(t)+h\mu)^2}, \frac{\partial F}{\partial R} = 0. \\ \frac{\partial G}{\partial S} &= \frac{h\beta I(t)}{1+h\alpha_1+h\mu}, \frac{\partial G}{\partial E} = \frac{1+h\sigma_2\Delta B_2}{1+h\alpha_1+h\mu}, \frac{\partial G}{\partial I} = \frac{h\beta S(t)}{1+h\alpha_1+h\mu}, \frac{\partial G}{\partial R} = 0. \\ \frac{\partial H}{\partial S} &= 0, \frac{\partial H}{\partial E} = \frac{h\alpha_1}{1+h(\alpha_2+\mu+\beta)}, \frac{\partial H}{\partial I} = \frac{h\sigma_3\Delta B_3}{1+h(\alpha_2+\mu+\beta)}, \frac{\partial H}{\partial R} = 0. \\ \frac{\partial K}{\partial S} &= 0, \frac{\partial K}{\partial E} = 0, \frac{\partial K}{\partial I} = \frac{h\alpha_2}{1+h\mu}, \frac{\partial K}{\partial R} = \frac{1+h\sigma_4\Delta B_4}{1+h\mu}. \\ J &= \begin{bmatrix} \frac{\partial F}{\partial S(t)}, \frac{\partial F}{\partial E(t)}, \frac{\partial F}{\partial I(t)}, \frac{\partial F}{\partial R(t)}, \frac{\partial G}{\partial R(t)}, \frac{\partial F}{\partial I(t)}, \frac{\partial F}{\partial I(t)}, \frac{\partial F}{\partial R(t)}, \frac{\partial F}{\partial I(t)}, \frac{\partial F}{\partial I(t)},$$

 $\lim_{\substack{1+h\mu\\ 1+h\mu}} \frac{1}{1+h\mu} \int Linearization of equilibria D = (S, E, I, R) = \left(\frac{h}{\mu}, 0, 0, 0\right) \text{ and } R_o < 1.$

$$\begin{split} & J\left(\frac{\Lambda}{\mu},0,0,0\right) \\ & = \begin{bmatrix} \frac{1+h\sigma_1\Delta B_1}{1+h\mu} & 0 & -\frac{\left(\frac{\Lambda}{\mu}+h\wedge+h\sigma_1\Delta B_1\right)h\beta}{(1+h\mu)^2} & 0 \\ 0 & \frac{1+h\sigma_2\Delta B_2}{1+h\alpha_1+h\mu} & \frac{h\beta\frac{\Lambda}{\mu}}{1+h\alpha_1+h\mu} & 0 \\ 0 & \frac{h\alpha_1}{1+h(\alpha_2+\mu+\beta)} & \frac{h\sigma_3\Delta B_3}{1+h(\alpha_2+\mu+\beta)} & 0 \\ 0 & 0 & \frac{h\alpha_2}{1+h\mu} & \frac{1+h\sigma_4\Delta B_4}{1+h\mu} \end{bmatrix}. \end{split}$$

These are the eigen values as follows:

$$\begin{split} \lambda_1 &= \frac{1+h\sigma_1\Delta B_1}{1+h\mu} < 1, \ \lambda_2 &= \frac{1+h\sigma_4\Delta B_4}{1+h\mu} < 1 \ \text{if} \ R_o < 1. \\ &= \left[\frac{\frac{1+h\sigma_2\Delta B_2}{1+h\alpha_1+h\mu}}{\frac{h\alpha_1}{1+h\alpha_1+h\mu}} \frac{\frac{h\frac{\alpha_1\beta\wedge}{\mu}}{1+h\alpha_1+h\mu}}{\frac{h\alpha_3\Delta B_3}{1+h(\alpha_2+\mu+\beta)}} \right]. \end{split}$$

A is represented as trace of J.

B is represented as modulus of J.

$$A = \frac{1+h\sigma_2\Delta B_2}{1+h\alpha_1+h\mu} + \frac{h\sigma_3\Delta B_3}{1+h(\alpha_2+\mu+\beta)}.$$

$$B = \frac{(1+h\sigma_2\Delta B_2)(h\sigma_3\Delta B_3)}{(1+h\alpha_1+h\mu)(1+h(\alpha_2+\mu+\beta))} - \frac{h^2\frac{\alpha_1\beta\wedge}{\mu}}{(1+h\alpha_1+h\mu)(1+h(\alpha_2+\mu+\beta))}.$$

Lemma: Brauer et al. [Brauer and Chavez (2001)] have presented result, for given $R_o < 1$ and equation $\lambda^2 - T_1\lambda + T_2 = 0$, $|\lambda_i| < 1, i = 1, 2$, which satisfy the below mentioned conditions then equilibria is stable.

(i)
$$1 + T_1 + T_2 > 0$$

(ii) $1 - T_1 + T_2 > 0$
(iii) $T_2 < 1$

Proof:

$$\begin{array}{ll} (i). & 1+T_{1}+T_{2}>0 \\ \because 1>0 \ , T_{1}>0 \ , To \ prove \ T_{2}>0. \\ \end{array} \\ \Rightarrow \frac{(1+h\sigma_{2}\Delta B_{2})(h\sigma_{3}\Delta B_{3})}{(1+h\alpha_{1}+h\mu)(1+h(\alpha_{2}+\mu+\beta))} - \frac{h^{2}\alpha_{1}\frac{\alpha_{3}\beta\wedge}{\mu}}{(1+h\alpha_{1}+h\mu)(1+h(\alpha_{2}+\mu+\beta))} > 0. \\ \Rightarrow (1+h\sigma_{2}\Delta B_{2})(h\sigma_{3}\Delta B_{3}) - h^{2}\frac{\alpha_{1}\beta\wedge}{\mu}>0. \\ \Rightarrow h\sigma_{3}\Delta B_{3} + h^{2}\sigma_{2}\sigma_{3}\Delta B_{2}\Delta B_{3} > h^{2}\frac{\alpha_{1}\beta\wedge}{\mu}. \\ \Rightarrow h^{2}\left(\sigma_{2}\sigma_{3}\Delta B_{2}\Delta B_{3} - \frac{\alpha_{1}\beta\wedge}{\mu}\right) + h\sigma_{3}\Delta B_{3}>0. \\ \Rightarrow \ h^{2}\left(\sigma_{2}\sigma_{3}\Delta B_{2}\Delta B_{3} - \frac{\alpha_{1}\beta\wedge}{\mu}\right) + h\sigma_{3}\Delta B_{3}>0. \\ \Rightarrow \ h^{2}\left(\tau_{2}\sigma_{3}\Delta B_{2}\Delta B_{3} - \frac{\alpha_{1}\beta\wedge}{\mu}\right) + h\sigma_{3}\Delta B_{3}>0. \\ \Rightarrow \ h^{2}\left(+\frac{\sigma_{3}\Delta B_{3}}{(\sigma_{2}\sigma_{3}\Delta B_{2}\Delta B_{3} - \frac{\alpha_{1}\beta\wedge}{\mu})}h > 0. \\ \Rightarrow \ (h)^{2} + 2(h)\left(\frac{\sigma_{3}\Delta B_{3}}{(\sigma_{2}\sigma_{3}\Delta B_{2}\Delta B_{3} - \frac{\alpha_{1}\beta\wedge}{\mu})}h + 1\right)^{2} > \left(\frac{\sigma_{3}\Delta B_{3}}{(2\sigma_{2}\sigma_{3}\Delta B_{2}\Delta B_{3} - \frac{\alpha_{1}\beta\wedge}{\mu})}h \right)^{2}. \\ \Rightarrow \left(\frac{\sigma_{3}\Delta B_{3}}{2(\sigma_{2}\sigma_{3}\Delta B_{2}\Delta B_{3} - \frac{\alpha_{1}\beta\wedge}{\mu}}h + 1\right)^{2} > \left(\frac{\sigma_{3}\Delta B_{3}}{(\sigma_{2}\sigma_{3}\Delta B_{2}\Delta B_{3} - \frac{\alpha_{1}\beta\wedge}{\mu})}h - \frac{\sigma_{3}\Delta B_{3}}{2(\sigma_{2}\sigma_{3}\Delta B_{2}\Delta B_{3} - \frac{\alpha_{1}\beta\wedge}{\mu})}h - \frac{\sigma_{3}\Delta B_{3}}{2(\sigma_{2}\sigma_{3}\Delta B_{2}\Delta B_{3} - \frac{\alpha_{1}\beta\wedge}{\mu})}h - \frac{\sigma_{3}\Delta B_{3}}{2(\sigma_{2}\sigma_{3}\Delta B_{2}\Delta B_{3} - \frac{\alpha_{1}\beta\wedge}{\mu})}h \\ \Rightarrow h > \frac{\sigma_{3}\Delta B_{3}}{2(\sigma_{2}\sigma_{3}\Delta B_{2}\Delta B_{3} - \frac{\alpha_{1}\beta\wedge}{\mu})} - \frac{\sigma_{3}\Delta B_{3}}{2(\sigma_{2}\sigma_{3}\Delta B_{2}\Delta B_{3} - \frac{\alpha_{1}\beta\wedge}{\mu})}h \\ \Rightarrow h > 0. \ As, \ time step is always positive. \\ (ii). 1 - T_{1} + T_{2} > 0 \\ \Rightarrow 1 - \frac{1+h\sigma_{2}\Delta B_{2}}{1+h\alpha_{1}+h\alpha_{1}+h\alpha_{2}+\mu+\beta}h + \frac{(1+h\sigma_{2}\Delta B_{2})(h\sigma_{3}\Delta B_{3}) -h^{2}\frac{\alpha_{1}\beta\wedge}{\mu}}{(1+h\alpha_{1}+h\mu)(1+h(\alpha_{2}+\mu+\beta))} > 0. \end{array}$$

$$\begin{split} &\Rightarrow \left[1+h(\alpha_{1}+\mu)\left[1+h(\alpha_{2}+\mu+\beta)\right]-(1+h\sigma_{2}\Delta B_{2})-h\sigma_{3}\Delta B_{3}+(1+h\sigma_{2}\Delta B_{2})(h\sigma_{3}\Delta B_{3})-h^{2}\frac{\alpha_{1}\beta\wedge}{\mu}>0. \\ &\Rightarrow 1+h(\alpha_{2}+\mu+\beta)+h(\alpha_{1}+\mu)+h^{2}(\alpha_{1}+\mu)(\alpha_{2}+\mu+\beta)-1-h\sigma_{2}\Delta B_{2}-h\sigma_{3}\Delta B_{3}+h\sigma_{3}\Delta B_{3}+h^{2}\sigma_{2}\sigma_{3}\Delta B_{2}\Delta B_{3}-h^{2}\frac{\alpha_{1}\beta\wedge}{\mu}>0. \\ &\Rightarrow h^{2}[(\alpha_{1}+\mu)(\alpha_{2}+\mu+\beta)+\sigma_{2}\sigma_{3}\Delta B_{2}\Delta B_{3}-\frac{\alpha_{1}\beta\wedge}{\mu}]+h[2\mu+\alpha_{1}+\alpha_{2}+\beta-\sigma_{2}\Delta B_{2}]>0. \\ &\Rightarrow h^{2}+\frac{h[2\mu+\alpha_{1}+\alpha_{2}+\beta-\sigma_{2}\Delta B_{2}]}{[(\alpha_{1}+\mu)(\alpha_{2}+\mu+\beta)+\sigma_{2}\sigma_{3}\Delta B_{2}\Delta B_{3}-\frac{\alpha_{1}\beta\wedge}{\mu}]}>0. \\ &\Rightarrow (h)^{2}+2(h)\left(\frac{[2\mu+\alpha_{1}+\alpha_{2}+\beta-\sigma_{2}\Delta B_{2}]}{2[(\alpha_{1}+\mu)(\alpha_{2}+\mu+\beta)+\sigma_{2}\sigma_{3}\Delta B_{2}\Delta B_{3}-\frac{\alpha_{1}\beta\wedge}{\mu}]}\right)^{2}>\left(\frac{[2\mu+\alpha_{1}+\alpha_{2}+\beta-\sigma_{2}\Delta B_{2}]}{2[(\alpha_{1}+\mu)(\alpha_{2}+\mu+\beta)+\sigma_{2}\sigma_{3}\Delta B_{2}\Delta B_{3}-\frac{\alpha_{1}\beta\wedge}{\mu}]}\right)^{2}. \\ &\Rightarrow \left(h+\frac{[2\mu+\alpha_{1}+\alpha_{2}+\beta-\sigma_{2}\Delta B_{2}]}{2[(\alpha_{1}+\mu)(\alpha_{2}+\mu+\beta)+\sigma_{2}\sigma_{3}\Delta B_{2}\Delta B_{3}-\frac{\alpha_{1}\beta\wedge}{\mu}]}\right)^{2}>\left(\frac{[2\mu+\alpha_{1}+\alpha_{2}+\beta-\sigma_{2}\Delta B_{2}]}{2[(\alpha_{1}+\mu)(\alpha_{2}+\mu+\beta)+\sigma_{2}\sigma_{3}\Delta B_{2}\Delta B_{3}-\frac{\alpha_{1}\beta\wedge}{\mu}]}\right)^{2}. \\ &\Rightarrow h+\frac{[2\mu+\alpha_{1}+\alpha_{2}+\beta-\sigma_{2}\Delta B_{2}]}{2[(\alpha_{1}+\mu)(\alpha_{2}+\mu+\beta)+\sigma_{2}\sigma_{3}\Delta B_{2}\Delta B_{3}-\frac{\alpha_{1}\beta\wedge}{\mu}]}>\frac{[2\mu+\alpha_{1}+\alpha_{2}+\beta-\sigma_{2}\Delta B_{2}]}{2[(\alpha_{1}+\mu)(\alpha_{2}+\mu+\beta)+\sigma_{2}\sigma_{3}\Delta B_{2}\Delta B_{3}-\frac{\alpha_{1}\beta\wedge}{\mu}]}^{2}. \end{split}$$

 \Rightarrow *h* > 0. As, time step is always positive.

$$\begin{split} &\Rightarrow \frac{[1+h\sigma_{2}\Delta B_{2}][h\sigma_{3}\Delta B_{3}]}{[1+h(\alpha_{1}+\mu)][1+h(\alpha_{2}+\mu+\beta)]} - \frac{h^{2}\frac{\alpha_{1}\beta\wedge}{\mu}}{[1+h(\alpha_{1}+\mu)][1+h(\alpha_{2}+\mu+\beta)]} < 1. \\ &\Rightarrow [(1+h\sigma_{2}\Delta B_{2})(h\sigma_{3}\Delta B_{3}) - h^{2}\frac{\alpha_{1}\beta\wedge}{\mu}] < [1+h(\alpha_{1}+\mu)][1+h(\alpha_{2}+\mu+\beta)]. \\ &\Rightarrow h\sigma_{3}\Delta B_{3} + h^{2}\sigma_{2}\sigma_{3}\Delta B_{2}\Delta B_{3} - h^{2}\frac{\alpha_{1}\beta\wedge}{\mu} < 1+h(\alpha_{1}+\alpha_{2}+2\mu+\beta) + h^{2}(\alpha_{1}+\mu)(\alpha_{2}+\mu+\beta). \\ &\Rightarrow h^{2}\left[\frac{\alpha_{1}\beta\wedge}{\mu} + (\alpha_{1}+\mu)(\alpha_{2}+\mu+\beta) - \sigma_{2}\sigma_{3}\Delta B_{2}\Delta B_{3}\right] + h[\alpha_{1}+\alpha_{2}+2\mu+\beta - \sigma_{3}\Delta B_{3}] + 1 > 0. \\ &\Rightarrow h^{2} + \frac{h[\alpha_{1}+\alpha_{2}+2\mu+\beta-\sigma_{3}\Delta B_{3}]}{\left[\frac{\alpha_{1}\beta\wedge}{\mu} + (\alpha_{1}+\mu)(\alpha_{2}+\mu+\beta) - \sigma_{2}\sigma_{3}\Delta B_{2}\Delta B_{3}\right]} + \frac{1}{\left[\frac{\alpha_{1}\beta\wedge}{\mu} + (\alpha_{1}+\mu)(\alpha_{2}+\mu+\beta) - \sigma_{2}\sigma_{3}\Delta B_{2}\Delta B_{3}\right]} > 0. \\ &\Rightarrow (h)^{2} + 2(h)\left(\frac{[\alpha_{1}+\alpha_{2}+2\mu+\beta-\sigma_{3}\Delta B_{3}]}{2\left[\frac{\alpha_{1}\beta\wedge}{\mu} + (\alpha_{1}+\mu)(\alpha_{2}+\mu+\beta) - \sigma_{2}\sigma_{3}\Delta B_{2}\Delta B_{3}\right]}\right)^{2} + \frac{1}{\left[\frac{\alpha_{1}\beta\wedge}{\mu} + (\alpha_{1}+\mu)(\alpha_{2}+\mu+\beta) - \sigma_{2}\sigma_{3}\Delta B_{2}\Delta B_{3}\right]} > (\frac{[\alpha_{1}+\alpha_{2}+2\mu+\beta-\sigma_{3}\Delta B_{3}]}{2\left[\frac{\alpha_{1}\beta\wedge}{\mu} + (\alpha_{1}+\mu)(\alpha_{2}+\mu+\beta) - \sigma_{2}\sigma_{3}\Delta B_{2}\Delta B_{3}\right]})^{2}. \end{split}$$

$$\Rightarrow \left(\frac{\left[\alpha_{1}+\alpha_{2}+2\mu+\beta-\sigma_{3}\Delta B_{3}\right]}{2\left[\frac{\alpha_{1}\beta\Lambda}{\mu}+(\alpha_{1}+\mu)(\alpha_{2}+\mu+\beta)-\sigma_{2}\sigma_{3}\Delta B_{2}\Delta B_{3}\right]}+h\right)^{2}+\frac{1}{\left[\frac{\alpha_{1}\beta\Lambda}{\mu}+(\alpha_{1}+\mu)(\alpha_{2}+\mu+\beta)-\sigma_{2}\sigma_{3}\Delta B_{2}\Delta B_{3}\right]} > \\ \left(\frac{\left[\alpha_{1}+\alpha_{2}+2\mu+\beta-\sigma_{3}\Delta B_{3}\right]}{2\left[\frac{\alpha_{1}\beta\Lambda}{\mu}+(\alpha_{1}+\mu)(\alpha_{2}+\mu+\beta)-\sigma_{2}\sigma_{3}\Delta B_{2}\Delta B_{3}\right]}\right)^{2}.$$

Which is always true if $R_o < 1$. So, the proposed technique is linearizable *D*. Linearization of equilibria $E = (S_1, E_1, I_1, R_1)$ and $R_o > 1$.



Figure 5: Spectral radius of Jacobean matrix for endemic equilibria (EE)

The largest eigen value of J(E) is less than one, eventually remaining two eigen values are also less than one. So, the proposed technique is locally asymptotical stable (LAS) around E.





Figure 6: (a) Converges behavior of compartment when h=0.1 (b) Converges behavior of compartment when h=100 (c) Exposed humans when h=0.1 (d) First run for exposed humans when h=100

4.5 Contrast section

The contrast of existing numerical techniques has presented below as:



Figure 7: (a) First run for exposed humans with Euler Mayuyama (b) Exposed humans' behavior with stochastic Euler (c) Second run for exposed humans with stochastic Runge Kutta (d) Exposed humans' behavior with stochastic Runge Kutta

4.6 Covariance of model

The covariance of model for each compartment has presented. In Tab. 3, the relationship quantity and its consequences are described.

Population Compartments	Relationship Coefficient (ρ)	Relationship
(S, E)	-0.8410	Inverse
(S, I)	-0.8922	Inverse
(S,R)	-0.9091	Inverse
(E, I)	0.9676	Direct
(E, R)	0.9832	Direct
(I,R)	0.9466	Direct

Table 3:	Relationship	Quantity
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We have noticed in Tab. 3, inverse relationship has among susceptible compartment and remaining three sections. Exposed population is directly correlated with infected and recovered populations. There is also a direct relationship between infected and recovered population. So, increase the individuals in susceptible section means population have moved to disease free equilibrium (DFE).

5 Results and discussion

In Fig. 2, the Euler Maruyama behaves well for h=0.1 at disease free equilibrium (DFE) but it diverges for h=0.001 at endemic equilibrium (EE). Furthermore, we have observed more runs at same time step size the given scheme shows negativity, unexpeted flucations and eventually diverge. It means we can not study the malaria dynamics in the human poulation over long period of time by Euler Maruyama method. In Fig. 3, stochastic Euler method behaves same as Euler Maruyama method. But in Fig. 4, stochastic Runge Kutta shows unexpected flucation, negativity and even diverge for certain runs. In Fig. 6, the stochastic non standard finite difference (SNSFD) method shows always convergence at any time step size and even for any run. We have claim that the stochastic non standard finite difference (SNSFD) method at different time step sizes and runs. Also, the deterministic solution called the averages of stochastic solutions. So, we have claim our proposed method is structure preserving method.

6 Conclusion and future framework

Comparatively, the numerical treatment for the stochastic malaria model gives a better understanding of disease dynamics. The Euler Maruyama, stochastic Euler and stochastic Runge-Kutta behave well for very small-time step sizes and may diverge for relatively for large step sizes. The newly constructed stochastic non standard finite difference (SNSFD) method works better for any partition of interval and absorbed the dynamical features defined by Mickens [Mickens (2005)] in stochastic sense. We have claim stochastic analysis of model is most effective and real as compared to deterministic analysis of model. No doubt they are connected to each other. In future, we shall extend our work in stochastic fractional order systems [Salahsour, Ahmadian, Senu et al. (2015)]. Moreover, we shall extend this idea in neural network based finite time control and stochastic resonance dynamics as presented in Wang et al. [Wang, Zhang, Zhou et al. (2019); Deivalakshmi, Palanisamy and Gao (2019)].

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